Fluoride in Drinking-water

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

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.

Acknowledgements

Acronyms and abbreviations used in the text

CAS	Chemical Abstracts Service
DNA	deoxyribonucleic acid
EPA	Environmental Protection Agency (USA)
IARC	International Agency for Research on Cancer
IPCS	International Programme on Chemical Safety
RR	relative risk
USA	United States of America
WHO	World Health Organization

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1. GENERAL DESCRIPTION

1.1 Identity

Fluorine is a common element that does not occur in the elemental state in nature because of its high reactivity. It accounts for about 0.3 g/kg of the Earth's crust and exists in the form of fluorides in a number of minerals, of which fluorspar, cryolite and fluorapatite are the most common. The oxidation state of the fluoride ion is -1.

1.2 Physicochemical properties (IARC, 1982; Slooff et al., 1988; IPCS, 2002)

Hydrogen fluoride (HF, CAS No. 7664-39-3) is a colourless, pungent liquid or gas with a boiling point of 19.5 °C. It is highly soluble in water, in which it forms hydrofluoric acid.

Sodium fluoride (NaF, CAS No. 7681-49-4) is a colourless to white solid that is moderately soluble in water.

Fluorosilicic acid (H_2SiF_6, CAS No. 16961-83-4), which is also known as hexafluorosilicic acid, is a colourless solid that is highly soluble in water.

1.3 Major uses

Inorganic fluorine compounds are used in industry for a wide range of purposes. They are used in aluminium production and as a flux in the steel and glass fibre industries. They can also be released to the environment during the production of phosphate fertilizers (which contain an average of 3.8% fluorine), bricks, tiles and ceramics. Fluorosilicic acid, sodium hexafluorosilicate and sodium fluoride are used in municipal water fluoridation schemes (IARC, 1982; IPCS, 2002).

1.4 Environmental fate

Although sodium fluoride is soluble in water (IARC, 1982), aluminium, calcium and magnesium fluorides are only sparingly so (US EPA, 1985a).

2. ANALYTICAL METHODS

Fluoride is usually determined by means of an ion-selective electrode, which makes it possible to measure the total amount of free and complex-bound fluoride dissolved in water. The method can be used for water containing at least 20 µg/litre (Slooff et al., 1988). For rainwater in which fluoride was present at a concentration of 10 µg/litre, a detection limit of 1 µg/litre was reported (Barnard & Nordstrom, 1982).

A method using a fluoride-selective electrode and an ion analyser to determine fluoride at levels of 0.05–0.4 mg/litre has been described (Liu et al., 1987).

3. ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE

3.1 Air

Natural background concentrations are of the order of 0.5 ng/m^3. If anthropogenic emissions are included, worldwide background concentrations are of the order of 3 ng/m^3. In the Netherlands, concentrations in areas without sources are 30–40 ng/m^3, rising to 70 ng/m^3 in areas with many sources (Slooff et al., 1988). In a survey of fluoride in the air of some communities in the USA and Canada, concentrations were in the range 0.02–2.0 µg/m^3 (IPCS, 1984). In some provinces of China, fluoride concentrations in indoor air ranged from 16 to 46 µg/m^3 owing to the indoor combustion of high-fluoride coal for cooking and for drying and curing food (Cao & Li, 1992).

3.2 Water

Traces of fluorides are present in many waters; higher concentrations are often associated with underground sources. In seawater, a total fluoride concentration of 1.3 mg/litre has been reported (Slooff et al., 1988). In areas rich in fluoride-containing minerals, well water may contain up to about 10 mg of fluoride per litre. The highest natural level reported is 2800 mg/litre. Fluorides may also enter a river as a result of industrial discharges (Slooff et al., 1988). In groundwater, fluoride concentrations vary with the type of rock the water flows through but do not usually exceed 10 mg/litre (US EPA, 1985a). In the Rhine in the Netherlands, levels are below 0.2 mg/litre. In the Meuse, concentrations fluctuate (0.2–1.3 mg/litre) as a result of industrial processes (Slooff et al., 1988).

Fluoride concentrations in the groundwater of some villages in China were greater than 8 mg/litre (Fuhong & Shuqin, 1988; Anonymous, 1990). In Canada, fluoride levels in drinking-water of <0.05–0.2 mg/litre (non-fluoridated) and 0.6–1.1 mg/litre (fluoridated) have been reported in municipal waters; in drinking-water prepared from well water, levels up to 3.3 mg/litre have been reported. In the USA, 0.2% of the population is exposed to more than 2.0 mg/litre (US EPA, 1985a). In the Netherlands, year-round averages for all drinking-water plants are below 0.2 mg/litre (Slooff et al., 1988). In some African countries where the soil is rich in fluoride-containing minerals, levels in drinking-water can be very high (e.g., 8 mg/litre in the United Republic of Tanzania) (US EPA, 1985a).

In a large survey of groundwater boreholes in central Australia, one-half of the supplies contained fluoride at levels exceeding 1.5 mg/litre, with several in the range 3–9 mg/litre (Fitzgerald et al., 2000).

3.3 Food

Virtually all foodstuffs contain at least traces of fluorine. All vegetation contains some fluoride, which is absorbed from soil and water. The highest levels in field-grown vegetables are found in curly kale (up to 40 mg/kg fresh weight) and endive

(0.3–2.8 mg/kg fresh weight) (Slooff et al., 1988). Other foods containing high levels include fish (0.1–30 mg/kg) and tea (US EPA, 1985a; Slooff et al., 1988). High concentrations in tea can be caused by high natural concentrations in tea plants or by the use of additives during growth or fermentation. Levels in dry tea can be 3–300 mg/kg (average 100 mg/kg), so 2–3 cups of tea contain approximately 0.4–0.8 mg (IPCS, 1984; Slooff et al., 1988). In areas where water with a high fluoride content is used to prepare tea, the intake via tea can be several times greater.

3.4 Dental uses

For dental purposes, fluoride preparations may contain low (0.25–1 mg per tablet; 1000–1500 mg of fluorine per kg of toothpaste) or high concentrations (liquids containing 10 000 mg/litre and gels containing 4000–6000 mg/kg are used for local applications) (Slooff et al., 1988).

3.5 Estimated total exposure and relative contribution of drinking-water

Levels of daily exposure to fluoride depend mainly on the geographical area. In the Netherlands, the total daily intake is calculated to be 1.4–6.0 mg of fluoride. Food seems to be the source of 80–85% of fluoride intake; intake from drinking-water is 0.03–0.68 mg/day and from toothpaste 0.2–0.3 mg/day. For children, total intake via food and water is decreased because of lower consumption. Intake of food and water relative to body weight is higher, however, and can be further increased by the swallowing of toothpaste or fluoride tablets (up to 3.5 mg of fluoride per day) (Slooff et al., 1988).

Daily intakes of fluoride vary widely according to the various sources of exposure. Values ranging from 0.46 to 3.6–5.4 mg/day have been reported in several studies (IPCS, 1984), but intakes in areas where high-fluoride coal is used indoors or where there is elevated fluoride in drinking-water can be significantly higher (IPCS, 2002). For example, in some counties in China where coal has a high fluoride content and is burned in houses with poor ventilation, the average daily intake of fluoride ranged from 0.3 to 2.3 mg via air and from 1.8 to 8.9 mg via food (Cao et al., 1992). In areas with relatively high fluoride concentrations in groundwater, drinking-water becomes increasingly important as a source of fluoride. Daily exposure in volcanic areas (e.g., the United Republic of Tanzania) with high fluoride levels in drinking-water may be up to 30 mg for adults, mainly from drinking-water intake.

4. KINETICS AND METABOLISM IN LABORATORY ANIMALS AND HUMANS

After oral uptake, water-soluble fluorides are rapidly and almost completely absorbed in the gastrointestinal tract. Absorbed fluoride is transported via the blood; with prolonged intake of fluoride from drinking-water, concentrations in the blood are the same as those in drinking-water, a relationship that remains valid up to a concentration in drinking-water of 10 mg/litre. Distribution of fluoride is a rapid process. It is incorporated into teeth and bones; there is virtually no storage in soft

tissues. Incorporation into teeth and skeletal tissues is reversible: after cessation of exposure, mobilization from these tissues takes place. Fluoride is excreted via urine, faeces and sweat (IPCS, 1984; US EPA, 1985a; Janssen et al., 1988). Fluoride in inhaled particles is also absorbed, the extent of absorption depending on the size of the particles and the solubility of fluoride compounds present (IPCS, 2002).

5. EFFECTS ON LABORATORY ANIMALS AND IN VITRO TEST SYSTEMS

5.1 Short-term exposure

There have been a number of studies of fluoride in laboratory animals. Effects on the skeleton, such as inhibition of bone mineralization and formation, delayed fracture healing and reductions in bone volume and collagen synthesis, have been observed in a variety of studies in which rats received fluoride orally for periods of 3−5 weeks at doses in excess of 16 mg/litre. Bone fragility was increased at concentrations in excess of 64 mg/litre. In subchronic studies, altered bone remodelling, hepatic megalocytosis, nephrosis, mineralization of the myocardium and necrosis or degeneration of the seminiferous tubules in the testis were observed in mice administered fluoride in drinking-water (>4.5 mg/kg of body weight per day) over a period of 6 months (IPCS, 2002).

5.2 Mutagenicity and related end-points

Many mutagenicity studies have been carried out with fluorides (usually sodium fluoride). Tests in bacteria and insects were negative, as were *in vivo* studies by the oral route (IARC, 1987; Janssen et al., 1988; NTP, 1990; IPCS, 2002). In mammalian cells *in vitro*, fluoride causes genetic damage through chromosomal aberrations at cytotoxic concentrations only (≥10 mg/litre), the mechanism for which is suggested to be an indirect effect on the synthesis of proteins involved in DNA synthesis (IPCS, 2002). This genetic effect is probably of limited relevance for practical human exposures (Janssen et al., 1988).

5.3 Carcinogenicity

In a comprehensive carcinogenicity bioassay in which groups of male and female F344/N rats and B6C3F$_1$ mice were administered drinking-water containing up to 79 mg of fluoride per litre as sodium fluoride for a period of 2 years, there was no statistically significant increase in the incidence of any tumour in any single exposed group. There was a statistically significant trend of an increased incidence of osteosarcomas in male rats with increasing exposure to fluoride. However, the incidence was within the range of historical controls (NTP, 1990; IPCS, 2002).

Another 2-year carcinogenicity bioassay involving Sprague-Dawley rats exposed to up to 11.3 mg/kg of body weight per day in the diet also found no statistically significant increase in the incidence of osteosarcoma or other tumours (Maurer et al., 1990; IPCS, 2002). An additional study, which reported an increased incidence of

osteomas in mice receiving up to 11.3 mg/kg of body weight per day, is difficult to interpret because the animals were infected with type C retrovirus (IPCS, 2002).

6. EFFECTS ON HUMANS

Fluoride may be an essential element for animals and humans. For humans, however, the essentiality has not been demonstrated unequivocally, and no data indicating the minimum nutritional requirement are available. To produce signs of acute fluoride intoxication, minimum oral doses of at least 1 mg of fluoride per kg of body weight were required (Janssen et al., 1988).

Many epidemiological studies of possible adverse effects of the long-term ingestion of fluoride via drinking-water have been carried out. These studies clearly establish that fluoride primarily produces effects on skeletal tissues (bones and teeth). Low concentrations provide protection against dental caries, especially in children. The pre- and post-eruptive protective effects of fluoride (involving the incorporation of fluoride into the matrix of the tooth during its formation, the development of shallower tooth grooves, which are consequently less prone to decay, and surface contact with enamel) increase with concentration up to about 2 mg of fluoride per litre of drinking-water; the minimum concentration of fluoride in drinking-water required to produce it is approximately 0.5 mg/litre.

However, fluoride can also have an adverse effect on tooth enamel and may give rise to mild dental fluorosis (prevalence: 12–33%) at drinking-water concentrations between 0.9 and 1.2 mg/litre (Dean, 1942); the period of greatest susceptibility is at the time of mineralization of the secondary upper central incisor teeth at about 22–26 months of age.. This has been confirmed in numerous subsequent studies, including a recent large-scale survey carried out in China (Chen et al., 1988), which showed that, with drinking-water containing 1 mg of fluoride per litre, dental fluorosis was detectable in 46% of the population examined. The extent of exposure from food was not clear in these studies. In general, dental fluorosis does not occur in temperate areas at concentrations below 1.5–2 mg of fluoride per litre of drinking-water. In warmer areas, because of the greater amounts of water consumed, dental fluorosis can occur at lower concentrations in the drinking-water (IPCS, 1984; US EPA, 1985a; Cao et al., 1992). It is possible that in areas where fluoride intake via routes other than drinking-water (e.g., air, food) is elevated, dental fluorosis will develop at concentrations in drinking-water below 1.5 mg/litre (Cao et al., 1992).

Elevated fluoride intakes can also have more serious effects on skeletal tissues. Skeletal fluorosis (with adverse changes in bone structure) may be observed when drinking-water contains 3–6 mg of fluoride per litre. Crippling skeletal fluorosis usually develops only where drinking-water contains over 10 mg of fluoride per litre (IPCS, 1984). The US EPA (1985b) considers a concentration of 4 mg/litre to be protective against crippling skeletal fluorosis. The relation between exposure and response for adverse effects in bone has been considered by IPCS (2002), which concluded:

A few investigations on skeletal fluorosis or the risk of fractures include quantitative estimates of the dose–response relationship. Studies in China and India report an increased prevalence of skeletal fluorosis above the level of 1.4 mg fluoride/litre in drinking-water (Jolly et al., 1968; Choubisa et al., 1997; Xu et al., 1997). However, such studies suffer from limitations: the diagnostic criteria are not always specified or consist of self-reported symptoms, and only drinking-water is considered as a source of exposure. The latter problem is likely to be important, since other studies (Liang et al., 1997; Ando et al., 1998) estimate that, at least in some regions of China and India, the contribution from food can greatly exceed that from water. Therefore, one cannot rule out that high rates of skeletal fluorosis associated with a level greater than 1.4 mg/litre in drinking-water are due to other exposures. While there is a clear excess of skeletal fluorosis in these studies for a total intake of 14 mg/day, the quantitative relationship between total intake of fluoride from different sources and the risk of skeletal fluorosis cannot be estimated because of substantial uncertainties in the prevalence of effects in the range of intakes between 3 and 14 mg/day.

The studies on fractures are also difficult to interpret, but for different reasons:

- Few studies report an interpretable range of exposures to fluoride.
- Results tend to be contradictory, with no clear-cut trend in both men and women.
- Total intake of fluoride is not estimated.

One exception is represented by a study in China (Li et al., 2001) in which different sources of exposure have been considered and an estimate of total intake is presented. In this study, there is an upward trend for the risk of total fractures above an exposure of 1.45 mg fluoride/litre in drinking-water, but only for the highest level of exposure (i.e., >4.32 mg fluoride/litre in drinking-water) was the relative risk statistically significant (RR = 1.47; P = 0.01). In the concentration range of 1.45–2.19 mg fluoride/litre in drinking-water, corresponding to a total intake of 6.54 mg/day, there was a relative risk for all fractures of 1.17 and for hip fractures of 2.13 (both not statistically significant).

In summary, estimates based on studies from China and India indicate that:

- for a total intake of 14 mg/day, there is a clear excess risk of skeletal adverse effects; and
- there is suggestive evidence of an increased risk of effects on the skeleton at total fluoride intakes above about 6 mg/day.

Several epidemiological studies are available on the possible association between fluoride in drinking-water and cancer rates among the population. IARC evaluated these studies in 1982 and 1987 and considered that they provided inadequate evidence of carcinogenicity in humans. Subsequently, IPCS (2002) considered all new data and concluded that overall the evidence of carcinogenicity in laboratory animals is inconclusive and that the weight of evidence does not support the hypothesis that fluoride causes cancer in humans; however, the data on bone cancer are relatively limited.

The results of several epidemiological studies on the possible adverse effects of fluoride in drinking-water on pregnancy outcome indicate that there is no apparent relationship between the rates of Down syndrome or congenital malformation and the consumption of fluoridated drinking-water (IPCS, 1984, 2002; US EPA, 1985a; Janssen et al., 1988).

It is known that persons suffering from certain forms of renal impairment have a lower margin of safety for the effects of fluoride than the average person. The data

available on this subject are, however, too limited to allow a quantitative evaluation of the increased sensitivity to fluoride toxicity of such persons (US EPA, 1985a; Janssen et al., 1988).

7. GUIDELINE VALUE

There is no evidence to suggest that the guideline value of 1.5 mg/litre set in 1984 and reaffirmed in 1993 needs to be revised. Concentrations above this value carry an increasing risk of dental fluorosis, and much higher concentrations lead to skeletal fluorosis. The value is higher than that recommended for artificial fluoridation of water supplies, which is usually 0.5–1.0 mg/litre (Murray, 1986).

In setting national standards or local guidelines for fluoride or in evaluating the possible health consequences of exposure to fluoride, it is essential to consider the intake of water by the population of interest and the intake of fluoride from other sources (e.g., from food and air). Where the intakes are likely to approach, or be greater than, 6 mg/day, it would be appropriate to consider setting a standard or local guideline at a concentration lower than 1.5 mg/litre.

A range of treatment technologies are available; however, in some areas with high natural fluoride levels in drinking-water, the guideline value may be difficult to achieve, in some circumstances, with the treatment technology available.

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