Public Health Goal for
FLUORIDE
in Drinking Water

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PREFACE

Drinking Water Public Health Goal of the
Office of Environmental Health Hazard Assessment

This Public Health Goal (PHG) technical support document provides information on health effects from contaminants in drinking water. The PHG describes concentrations of contaminants at which adverse health effects would not be expected to occur, even over a lifetime of exposure. PHGs are developed for chemical contaminants based on the best available toxicological data in the scientific literature. These documents and the analyses contained in them provide estimates of the levels of contaminants in drinking water that would pose no significant health risk to individuals consuming the water on a daily basis over a lifetime.

The California Safe Drinking Water Act of 1996 (amended Health and Safety Code, Section 116365) requires the Office of Environmental Health Hazard Assessment (OEHHA) to adopt PHGs for contaminants in drinking water based exclusively on public health considerations. The Act requires OEHHA to adopt PHGs that meet the following criteria:

1. PHGs for acutely toxic substances shall be set at levels at which scientific evidence indicates that no known or anticipated adverse effects on health will occur, plus an adequate margin-of-safety.
2. PHGs for carcinogens or other substances which can cause chronic disease shall be based solely on health effects without regard to cost impacts and shall be set at levels which OEHHA has determined do not pose any significant risk to health.
3. To the extent the information is available, OEHHA shall consider possible synergistic effects resulting from exposure to two or more contaminants.
4. OEHHA shall consider the existence of groups in the population that are more susceptible to adverse effects of the contaminants than a normal healthy adult.
5. OEHHA shall consider the contaminant exposure and body burden levels that alter physiological function or structure in a manner that may significantly increase the risk of illness.
6. In cases of scientific ambiguity, OEHHA shall use criteria most protective of public health and shall incorporate uncertainty factors of noncarcinogenic substances for which scientific research indicates a safe dose-response threshold.
7. In cases where scientific evidence demonstrates that a safe dose-response threshold for a contaminant exists, then the PHG should be set at that threshold.
8. The PHG may be set at zero if necessary to satisfy the requirements listed above.
9. OEHHA shall consider exposure to contaminants in media other than drinking water, including food and air and the resulting body burden.
10. PHGs adopted by OEHHA shall be reviewed periodically and revised as necessary based on the availability of new scientific data.

PHGs adopted by OEHHA are for use by the California Department of Health Services (DHS) in establishing primary drinking water standards (State Maximum Contaminant Levels, or MCLs). Whereas PHGs are to be based solely on scientific and public health considerations without regard to economic cost considerations, drinking water standards adopted by DHS are to consider economic factors and technical feasibility. For this reason PHGs are only one part of the
information used by DHS for establishing drinking water standards. PHGs established by OEHHA exert no regulatory burden and represent only non-mandatory goals. By federal law, MCLs established by DHS must be at least as stringent as the federal MCL if one exists.

PHG documents are developed for technical assistance to DHS, but may also benefit federal, state and local public health officials. While the PHGs are calculated for single chemicals only, they may, if the information is available, address hazards associated with the interactions of contaminants in mixtures. Further, PHGs are derived for drinking water only and are not to be utilized as target levels for the contamination of environmental waters where additional concerns of bioaccumulation in fish and shellfish may pertain. Often environmental water contaminant criteria are more stringent than drinking water PHGs, to account for human exposures to a single chemical in multiple environmental media and from bioconcentration by plants and animals in the food chain.
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SUMMARY

A Public Health Goal (PHG) of 1 ppm (1,000 ppb) is developed for fluoride in drinking water. This level is intended to be an approximate year-round average. The U.S. Environmental Protection Agency’s (U.S. EPA’s) Maximum Contaminant Level (MCL) for fluoride is 4 mg/L. U.S. EPA’s MCL was set to protect against crippling skeletal fluorosis, with a secondary MCL of 2 mg/L to protect against dental fluorosis (in mild cases, fluorosis is a slight discoloration of teeth, in more severe cases it can lead to pitting and breaking of the teeth). Moderate to severe dental fluorosis is rare when the drinking water fluoride level is in the range of 1 mg/L, but begins to become significant at concentrations close to 2 mg/L. The PHG is based on a no-observed-adverse-effect-level (NOAEL) of 1 mg/L for dental fluorosis in children. A relative source contribution of 100% (1) was applied yielding a calculated PHG of 1 mg/L. This level is judged to be the optimum level for reducing the prevalence of dental fluorosis while providing protection against dental caries. In reviewing the available data on health effects of fluoride, studies have been found which provide some indication that there may be a causative relationship between lifetime consumption of fluoridated drinking water and increased incidence of hip fracture in the elderly. However, this health endpoint is not sufficiently established at present to provide the basis for calculating a PHG. Therefore, OEHHA calculates a PHG of 1 mg/L (1 ppm) for fluoride in drinking water.

INTRODUCTION

The purpose of this document is to develop a PHG for fluoride in drinking water. Fluoride can be distinguished from other chemicals for which PHGs are developed in that it can be present as an intentional additive rather than a “contaminant.” Fluoride is added to drinking water at levels close to 1 mg/L in order to protect children and adults against dental caries. Individuals who live in areas where the water is fluoridated are exposed to fluoride from this source. In addition, virtually everyone is exposed to fluoride in toothpaste, food and other sources described in this report. It is the cumulative exposure to fluoride from all of these sources that determines the likelihood of health effects such as dental fluorosis. Nonetheless, good correlations have been established between the drinking water level of fluoride and the prevalence of dental fluorosis. Drinking water standards or regulations can only control one source of fluoride exposure, but this one source has been demonstrated repeatedly to have a crucial effect on the prevalence of dental fluorosis.

In 1995, California enacted AB 733 (Speier) which requires the California Department of Health Services (DHS) to adopt regulations requiring public drinking water systems with more than 10,000 service connections to implement fluoridation as a health measure for preventing dental caries. This fluoridation requirement will be phased in between January 1, 1997 and January 1, 2000. Prior to enactment of this legislation, only 17% of Californians were supplied with fluoridated drinking water (Speier, 1995).

CHEMICAL PROFILE

Fluorine is the ninth element on the periodic table. It has an atomic weight of 18.9984. It is the lightest and most reactive member of the halogen family. Fluorine reacts with other elements to produce such ionic compounds as hydrogen fluoride (HF), sodium fluoride (NaF) and many others. When these ionic compounds are dissolved in water, the ions dissociate and fluorine is present as the negatively charged ion fluoride. Fluoride, usually as the sodium salt, is added to drinking water.
for the prevention of dental caries, as discussed throughout this report. Fluoride salts are also naturally occurring in geological formations, and therefore in some sources of drinking water.

ENVIRONMENTAL OCCURRENCE AND HUMAN EXPOSURE

Air and Soil

No data were found on fluoride levels in ambient air or residential soil. Fluoride can enter the air from sea spray, and might therefore be expected to be highest near the coast (U.S. Public Health Service, 1991). Human exposure to fluoride from ambient air has been estimated to be only about 1 to 4 \( \mu g/\text{day} \) (U.S. Public Health Service, 1991). This is insignificant compared to other sources of exposure. Fluoride exposures by inhalation are therefore not significant except in special occupational settings (Medvedeva, 1983; Desai, 1986). The use of fluoridated water for watering of lawns and gardens might be expected to add fluoride to air and soil, but this does not appear to have been systematically studied.

Water

Some sources of drinking water, especially surface water, are naturally low in fluoride (< 0.3 mg/L). In many communities fluoride is added to the water supply in order to bring its concentration into the range of 0.7 to 1.2 mg/L, which is deemed optimal for prevention of dental caries. In other communities fluoride need not be added because it is naturally present in this range, or even higher, up to about 3 mg/L (U.S. Public Health Service, 1991).

Children drink about one liter of water per day, and adults drink about two liters, depending on the ambient temperature. Because water consumption is higher in areas with higher ambient temperatures, the recommended amount of fluoride to be added to drinking water has been made temperature-dependent. A study in Senegal indicated that the World Health Organization (WHO) guideline of 1.5 mg/L is not sufficiently protective against dental fluorosis in children or skeletal fluorosis in adults in a hot and dry climate (Brouwer et al., 1988). In some locations in California, the climate is very hot with low percentage of humidity where water consumption may be similar to that in Senegal. In areas of California where the ambient temperature is high, the recommended fluoride level in the water would be 0.7 mg/L (California Health and Safety Code, Title 22, Article 4, Section 64435).

Most consumers use water from their tap for drinking water. In addition they use this water for preparing beverages such as orange juice, coffee and tea. The estimated intakes of one to two liters per day includes beverages as well as drinking water.

Food

Although raw foods contain some fluoride, the major source of fluoride in the diet is added to foods when they are cooked or processed with fluoridated water. Among the foods that are high in fluoride are tea and ocean fish containing bones or bone meal. Based on market basket diet studies the amount of fluoride in a typical adult's diet has been estimated by several researchers (Singer et al., 1980; Kramer et al., 1974; Marier and Rose, 1966; U.S. Public Health Service, 1991). For communities with low fluoride levels in drinking water the food contribution to fluoride exposure for adults ranges from 0.3 to 1 mg/day. For adults in communities with fluoridated water the...
corresponding fluoride intakes are 0.3 to 3.4 mg/day. To estimate the intakes for children, the adult intakes were divided approximately in half to yield intakes ranging from 0.1 to 0.5 mg/day and 0.1 to 1.7 mg/day for nonfluoridated and fluoridated communities, respectively.

**Toothpaste, Mouthwash and Fluoride Supplements**

**Toothpaste**

Over 90% of the toothpastes sold in the United States (U.S.) are fluoridated, with fluoride concentrations ranging from 1 mg/gram to 1.5 mg/gram (Whitford, 1989; U.S. Public Health Service, 1991). When a person brushes with a fluoride toothpaste, some of the fluoride is absorbed directly into the tooth enamel. Children may swallow 0.2 to 0.8 grams of toothpaste per day, whereas adults are estimated to swallow only approximately 0.02 to 0.1 grams per day (Whitford, 1989; U.S. Public Health Service, 1991). The ranges of fluoride exposures from toothpaste shown in Table 1 were calculated by multiplying the amounts swallowed by the fluoride concentration of the toothpaste.

**Mouthwash**

Mouthwashes contain 0.23 to 0.97 mg/gram fluoride (Whitford, 1989). These are probably used, and swallowed, more frequently by adults than by children. OEHHA is estimating that an adult would swallow about 1 gram of mouthwash per day, and a child would swallow about 0.5 grams. The range of fluoride exposures from mouthwashes indicated in Table 1 are based on these estimates of the amount swallowed multiplied by the range of fluoride concentrations found in mouthwashes.

**Fluoride Supplements**

Fluoride tablets are used to provide fluoride for children who live in areas with nonfluoridated water. These tablets provide 0.5 mg/day of fluoride to the child. Fluoride supplements are not recommended for children in areas with fluoridated water, or for adults.

**Total Fluoride Exposure**

The total fluoride intake for children and adults is given in Table 1, by summing the contributions from each of the significant exposure sources. Ambient air is not included in Table 1 because the contribution from air is only about 1 to 4 μg/day, which is negligible compared to the other sources.

For children, the estimated daily fluoride intake ranges from 1 to 4.6 mg. For adults the estimated range is 0.7 to 7 mg. For both children and adults the range of exposures is very broad, and is determined by many different factors including the drinking water source, the amount of water consumed, the kinds of foods eaten and the use of dental products and supplements. The total intake can be affected (but not controlled) by manipulating only one or two of these factors. In order to ensure the desired intake of fluoride, all factors must be taken into account.
METABOLISM AND PHARMACOKINETICS

Fluoride enters the body mainly by ingestion. Seventy-five to 90% of ingested fluoride is absorbed into the blood through the gastrointestinal tract (NRC, 1993). Ingested fluoride appears in the plasma 30 to 60 minutes after ingestion. Fluoride is present unbound in the plasma. There does not appear to be any homeostatic control of plasma fluoride levels. Fluoride is taken up into all the tissues of the body, but is retained and accumulated only in the bones and teeth. Ninety-nine percent of the “body burden” of fluoride resides in these tissues, tightly but reversibly bound into the crystalline structure.

Plasma fluoride is excreted mainly in the urine, but also in the feces and sweat. Fluoride readily enters breast milk and is thereby passed on to the nursing child. Clearance of fluoride from the plasma is the sum of uptake by the bones and teeth together with elimination in the urine and feces (NRC, 1993).

Table 1. Estimated Fluoride Intakes for Children and Adults

<table>
<thead>
<tr>
<th>Fluoride Intake (mg/kg)</th>
<th>Drinking Water</th>
<th>Food</th>
<th>Toothpaste</th>
<th>Mouthwash</th>
<th>Supplement</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Children</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 0.3</td>
<td>0.1 to 0.3</td>
<td>0.1 to 0.5</td>
<td>0.2 to 1.2</td>
<td>0.1 to 0.5</td>
<td>0.5</td>
<td>1.0 to 3.0</td>
</tr>
<tr>
<td>0.7 to 1.2</td>
<td>0.7 to 1.2</td>
<td>0.1 to 1.7</td>
<td>0.2 to 1.2</td>
<td>0.1 to 0.5</td>
<td>0</td>
<td>1.1 to 4.6</td>
</tr>
<tr>
<td><strong>Adults</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 0.3</td>
<td>0.2 to 0.6</td>
<td>0.3 to 1.0</td>
<td>0.02 to 0.15</td>
<td>0.2 to 1.0</td>
<td>0</td>
<td>0.7 to 2.8</td>
</tr>
<tr>
<td>0.7 to 1.2</td>
<td>1.4 to 2.4</td>
<td>0.3 to 3.4</td>
<td>0.02 to 0.15</td>
<td>0.2 to 1.0</td>
<td>0</td>
<td>1.9 to 7.0</td>
</tr>
</tbody>
</table>

TOXICOLOGY

Toxicological Effects in Animals

Noncarcinogenic Effects

When administered orally to laboratory animals, fluoride has adverse health effects on bones and teeth as well as the kidneys and stomach. The exposure levels at which these effects occur are much higher than those associated with water fluoridation as discussed below. Oral administration
of fluoride at high levels affects survival and body weight gain. Potential effects of fluoride exposure on reproduction and development are also discussed in this section.

**Body Weight**

In a two-year rat feeding study by Procter and Gamble (Maurer et al., 1990) there were four dose groups: controls, 1.8, 4.5 or 11 mg/kg-day. The high-dose (11 mg/kg-day) rats exhibited significantly depressed weight gain in both males and females compared to the controls. The low-dose groups (1.8 and 4.5 mg/kg-day) exhibited no effect on body weight gain. The controls that were fed lab chow had significantly reduced body weight gain relative to the low-fluoride diet controls; this was true for both male and female rats. However, the depression in weight gain was much greater (30%) for the high-dose rats than for the lab chow control rats (15%). The reason for the depression in weight gain in the lab chow controls remains unexplained. The depression in weight gain for the high-dose rats appears to be attributable mainly to reduced diet consumption. The reason for this reduced diet consumption is not also explained.

**Survival**

In the Procter and Gamble study (Maurer et al., 1990), there were no significant differences in survival between any of the groups of male rats. In female rats, survival was significantly depressed for the high-dose group and for the low-fluoride diet group relative to the other groups. The lab chow controls (both male and female) had significantly higher survival than any of the experimental groups.

**Teeth**

Rodents, with their large ever-growing incisors, have been used as a model for the effects of fluoride on tooth enamel. Fluoride had several dose-related adverse effects on teeth in the Procter and Gamble study (Maurer et al., 1990), including ameloblastic dysplasia, fracture and/or malformation and enamel hypoplasia. Ameloblasts are columnar epithelial cells in the "enamel organ" which secrete the material that forms the enamel. In moderate dysplasia the ameloblastic layer is disrupted and lays down an irregular layer of enamel. In severe dysplasia ameloblasts may be completely absent in some areas and the enamel layer is very irregular.

Ameloblastic dysplasia first appeared in the lowest dose group (1.8 mg/kg-day) and increased with dose in both male and female rats. Fractures and/or malformations appeared in the controls and in all doses in both sexes, but increased in a dose-related manner in both sexes. Enamel hypoplasia first appeared in the lowest dose group in both sexes, and increased with dose in both sexes.

In a National Toxicology Program (NTP) study (NTP, 1990) incisors of rats given drinking water with higher concentrations of fluoride (45 or 79 mg/L) were described as "chalky white" in appearance. Other teeth of these animals showed discoloration and abnormalities of shape. The study was not reported in such a way that a lowest-observed-adverse-effect-level (LOAEL) could be determined from the data.

**Bones**

Orally administered fluoride has been shown to affect bone calcification in every animal species tested, including rodents, dogs and cattle (U.S. Public Health Service, 1991). Rats given high
doses of sodium fluoride (4.5 mg/kg-day or more) exhibited bones that were described as white and thick with roughened surfaces (Maurer et al., 1990). The most common microscopic effect was subperiosteal hyperostosis, a diffuse irregular deposition of mature lamellar bone in the subperiosteal regions leading to excessively thick cortices.

This effect was most pronounced in crania, but was also observed in femurs, cervical vertebrae, premaxillae and maxillae. In all of these bones subperiosteal hyperostosis appeared in a dose-related manner in both males and females. In crania the effect was seen at all doses, but increased in a dose-related manner. In femurs it was observed only in the highest dose group (11 mg/kg-day) in both males and females.

Subperiosteal hyperostosis may be an adverse effect, since it would tend to change the mechanical properties of the bone surface, possibly affecting the performance of joints. As this effect has been observed in even the lowest dose group, an NOAEL cannot be determined from this study. The lowest dose group (1.8 mg/kg-day) may tentatively be considered an LOAEL for this effect.

The National Research Council (NRC) reviewed animals studies of the effect of bone fluoride on the susceptibility of bones to fracture. It was observed that the studies yielded "all possible outcomes," and the NRC report noted that the published studies are fraught with many technical problems that diminish their value for predicting fluoride effects in humans (NRC, 1993).

Kidneys

Kidney damage in rodents and other animals exposed to very high levels of fluoride in drinking water (100 to 380 mg/L) has been reported by a number of investigators (U.S. Public Health Service, 1991). These exposures are two orders of magnitude greater than those that humans would expect to encounter from drinking water.

The specific gravity of the urine increased in a dose-dependent manner in male and female of rats, in the Procter and Gamble study (Maurer et al., 1990). This may be an indication of a decreased ability of the kidneys to concentrate urine, however no damage to the kidneys was observed in any of the dose groups.

Stomach

All forms of fluoride are converted to hydrogen fluoride in the stomach. Fluoride administration to animals has been observed to cause lesions in the stomach only at concentrations many times higher (300 mg/L) than those likely to be encountered in drinking water used by humans (U.S. Public Health Service, 1991).

Fluoride appeared to be irritating to the stomachs of rats given high doses (25 mg NaF/kg-day) of fluoride in their food (Maurer et al., 1990). Irritation-related endpoints (mononuclear cell infiltration, chronic inflammation in the glandular region, regeneration in the glandular region, hyperkeratosis and acanthosis) appeared in the controls and in all dose groups, but increased in a dose-related manner in both male and female rats. No minimum dose for stomach irritation can be determined from these data.
Genetic Toxicity

The ability of fluoride to cause mutations has been tested in bacteria and cultured mammalian cells. Li *et al.* (1987) tested sodium fluoride in the Ames *Salmonella* assay with and without microsomes at concentrations ranging from 0.1 to 100 mg/L fluoride. Sodium fluoride did not increase the number of revertants over the solvent control. However, sodium fluoride was mutagenic in mouse lymphoma L5178Y cells at 400 mg/L and in human lymphoblasts at 440 mg/L (Caspary *et al.*, 1988). Potassium fluoride caused mutations at the thymidine kinase locus in mouse lymphoma cells at 500 to 700 mg/L (Caspary *et al.*, 1988).

Sodium fluoride at concentrations of 25 to 100 µg/mL was found to cause chromosome damage (e.g., deletions, gaps) at the G2 phase of the cell cycle in Chinese hamster ovary (CHO) cells (Aardema *et al.*, 1989). The authors speculated that fluoride may inhibit DNA synthesis or repair. Sodium fluoride also caused chromosome aberrations in cultured human lymphocytes at 20 to 40 mg/L (Albanese, 1987). Fluoride has been negative in most recent studies of sister-chromatid exchange (Tong *et al.*, 1988).

In general, fluoride has been found to be clastogenic in vitro only at concentrations higher than 10 mg/L. This activity may be due to an effect on enzymes involved in DNA synthesis or repair, rather than a direct effect on DNA (ATSDR, 1991; Caspary *et al.*, 1987 and 1988).

Sodium fluoride at 75 or 100 µg/mL has been shown to cause morphological and neoplastic transformation in Syrian hamster embryo cells in culture. The transformed cells developed the ability to produce anaplastic fibrosarcomas when injected into newborn hamsters (Tsutsui *et al.*, 1984). While this may be viewed as supporting evidence for the potential carcinogenicity of fluoride, the NRC has noted in its report that hamster embryo cells are unusually sensitive to the induction of transformation, leading them to conclude that "the overall significance of the fluoride transformation data are subject to question" (NRC, 1993).

Hydrogen fluoride at 1 mg/m³ did not cause dominant lethal mutations when tested by inhalation in the mouse C57B1 system (Voroshilin *et al.*, 1975). Sodium fluoride and hydrogen fluoride have been tested in rodents for clastogenic activity (chromosome aberrations, sister-chromatid exchange, sperm abnormality, micronuclei, DNA strand breaks) with mixed results. Four studies reported positive results, while seven studies were negative. The positive results occur only at doses that are toxic to the animals. One explanation would be that the clastogenic effect may be secondary to a more general effect on metabolism and synthesis of macromolecules (U.S. Public Health Service, 1991). Controversy over the potential clastogenicity of fluoride is one of the major unresolved research issues at present (Zeiger *et al.*, 1993).

Reproduction and Development

Reproductive capability was not affected in rats administered up to 23 mg fluoride/kg-day as sodium fluoride in the diet (Marks *et al.*, 1984), or in mice administered 13 mg fluoride/kg-day as sodium fluoride for three generations (Tao and Suttie, 1976).

A later study described changes in seminiferous tubules in rats administered 13 mg/kg-day in the diet as sodium fluoride with fewer offspring from the treated animals (Araibi *et al.*, 1989). The question of the effect of fluoride on reproduction in animals remains unresolved.
In a study of potential developmental effects, bone morphology of weanling rats was not affected when dams were given 21 mg fluoride/kg-day for 10 weeks before breeding and during gestation (Ream et al., 1983). Schellenberg et al. (1990) administered fluoride to dogs (460 mg/kg in feed), and observed no changes in rates of major congenital anomalies. These and other studies suggest that fluoride is not likely to be teratogenic in animals.

Carcinogenicity

There have been two recent rodent carcinogenicity studies of fluoride, one by the NTP (1990) and one by Procter and Gamble (Maurer et al., 1990). Both were conducted at approximately the same time, and both involved oral administration of fluoride to rats and mice.

NTP Carcinogenicity Study

In the NTP bioassay, fluoride was administered to rats and mice in the drinking water (see Table 2). The drinking water concentrations of fluoride were 11, 45 or 79 mg/L. There was also a control group with drinking water that was free of fluoride. All the rats and mice had a specially formulated low-fluoride diet. The amount of fluoride in the low-fluoride diet was not determined.

Three of the male rats in the highest dose group developed osteosarcomas. One male rat in the intermediate dose group also developed an osteosarcoma. These results are suggestive of a dose-related effect in male rats. Indeed, the trend test on these data is statistically significant, even though none of the data points differs statistically from the controls. Osteosarcomas are uncommon in rats. No osteosarcomas or other malignant tumors were reported in the female rats or in male or female mice.

Procter and Gamble Carcinogenicity Study

In the Procter and Gamble study, sodium fluoride was administered to rats and mice in the solid diet, at amounts calculated to yield predetermined doses of 0, 1.8, 4.5 or 11 mg/kg-day (see Table 2). On a mg/kg-day basis, the doses in the NTP study are calculated to be 0, 0.6, 2.3 and 3.9 mg/kg-day. It is noteworthy that the highest dose in the Procter and Gamble study was about three times the highest dose in the NTP study.

One female rat in the low-dose group in the Procter and Gamble study developed an osteosarcoma. This tumor was judged to be non-treatment-related because it was in the lowest dose group, and because no similar tumors were found in the highest dose groups. As stated above, osteosarcomas are uncommon in rats; no osteosarcomas or other malignant tumors were reported in the female rats or in male or female mice.

The mouse portion of the Procter and Gamble study was compromised by the presence of a type-C retrovirus in all the mice. No malignant tumors were detected in any of the mice, but osteomas were reported. Osteomas are a type of benign neoplasm arising from osteoblasts. The investigators concluded that the mouse study was of no value in determining the carcinogenic potential of fluoride in mice.

NRC asked the Armed Forces Institute of Pathology (AFIP) to evaluate the significance of the osteomas in the Procter and Gamble study (NRC, 1993). AFIP convened a committee of
pathologists who reviewed the study and a sample of the histolological slides. They concluded that the osteomas in question were not malignant and would not have progressed to a malignant state. They also stated that the presence of retroviral particles in the osteomas suggests that these viruses were involved in the induction of the osteomas (NRC, 1993).

Comparison of NTP and Procter and Gamble Studies

Table 2 provides a comparison of these two recent rodent bioassays.

Table 2. Two-year Rodent Carcinogenicity Studies on Fluoride

<table>
<thead>
<tr>
<th>Study</th>
<th>Animals</th>
<th>Doses</th>
<th>Tumor Outcomes</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>NTP (1990)</td>
<td>F344/N Rats</td>
<td>0, 0.6, 2.3, 3.9 mg/kg-day in water n = 80, 50, 50, 80/sex/group</td>
<td>45 mg/L: osteosarcoma in one male 79 mg/L: osteosarcomas in 3 males</td>
<td>&quot;equivocal evidence&quot; of carcinogenicity in male rats; no evidence of carcinogenicity in female rats</td>
</tr>
<tr>
<td>NTP (1990)</td>
<td>B6C3F1 Mice</td>
<td>0, 0.6, 2.3, 3.9 mg/kg-day in water n = 80, 50, 50, 80/sex/group</td>
<td>no tumors in either males or females</td>
<td>no evidence of carcinogenicity in mice of either gender</td>
</tr>
<tr>
<td>Procter and Gamble (1990)</td>
<td>S.D. Rats</td>
<td>0, 1.8, 4.5, 11 mg/kg-day n = 60/sex/group</td>
<td>1.8 mg/kg-day: osteosarcoma in 1 female, no tumors in males</td>
<td>no evidence of carcinogenicity in male or female rats</td>
</tr>
<tr>
<td>Procter and Gamble (1990)</td>
<td>CD-1 Mice</td>
<td>0, 1.8, 4.5, 11 mg/kg-day; n = 60/sex/group</td>
<td>no malignant tumors, some osteomas</td>
<td>uninformative because of type-C retrovirus infection</td>
</tr>
</tbody>
</table>

The NTP and Procter and Gamble rodent bioassays were comparable in that both involved oral administration of fluoride to rats and mice for a period of two years. The route of administration was somewhat different in that the NTP study involved administration of the chemical in drinking water whereas the Procter and Gamble study involved administration in the diet, making it difficult to accurately compare dosage groups between the two studies. Administration of fluoride in the diet might result in poorer absorption than administration in water.

Both studies observed comparable effects on teeth and bones in the animals administered intermediate and high doses of fluoride. Whereas the Procter and Gamble study did not find dose-dependent evidence for carcinogenicity in male or female rats, the NTP study did find some evidence of dose-dependent carcinogenicity in male rats based on three osteosarcomas in the high-dose (79 mg/L) group and one osteosarcoma in the intermediate dose (45 mg/L) group.
One osteosarcoma appeared in a female rat of the lowest dose group in the Procter and Gamble study which received 1.8 mg/kg-day. This is comparable to the intermediate dose (45 mg/L) group in the NTP study which received 2.3 mg/kg-day. Therefore, the dose to the female rat that developed osteosarcoma in the Procter and Gamble study was close to the dose to one of the male rats that developed osteosarcoma in the NTP study. Considered together these two studies may suggest that doses of sodium fluoride in this range may induce a small number of osteosarcomas, although the lack of osteosarcomas in the higher dose groups in the Procter and Gamble study cannot be explained.

The presence of a type-C retrovirus in the mouse colony for the Proctor and Gamble study compromised the integrity of the study and rendered the results of little value for risk assessment purposes.

**Toxicological Effects in Humans**

**Noncarcinogenic Effects**

**Teeth**

Excess oral exposure to fluoride can lead to dental fluorosis (Figure 1). Fluorosis occurs during the period of tooth formation as fluoride is incorporated into the crystalline structure of the enamel in such a way as to increase its porosity. The teeth are not discolored at the time of eruption, but subsequently become stained and discolored as a consequence of the increased porosity. There are varying degrees of dental fluorosis. Mild fluorosis is characterized by light tooth mottling. More severe fluorosis is characterized by darker and more widespread tooth mottling, as well as discoloration and pitting of the teeth. In severe cases the teeth are misshapen and apt to break (U.S. Public Health Services, 1991).

Humans who live in areas with high levels of fluoride in the drinking water have increasing degrees of dental fluorosis (U.S. Public Health Service, 1991). Tooth mottling occurs when fluoride concentrations in the drinking water exceed the levels which are optimal for prevention of dental caries (estimated to be 0.7 to 1.2 mg/L, see Figure 1). The degree of fluorosis depends on the level of fluoride in the water.

U.S. EPA considers tooth mottling a cosmetic effect, but there is some concern that it may have social and psychological consequences (Welbury, 1990). The cosmetic effects can sometimes be corrected. Increasing fluoride levels in the drinking water does not appear to add any protection against dental caries beyond the optimal level (Dean, 1942; U.S. Public Health Service, 1991; NRC, 1993). Some water systems in California have enough naturally occurring fluoride (3 to 4 mg/L) to cause increases in noticeable dental fluorosis.
The relationship between water fluoridation (or natural high fluoride content) and dental fluorosis has been studied in over 40 ecological epidemiological studies. Drinking water is not the only source of fluoride. For fetuses and children, an estimated 50% of the fluoride intake comes from the drinking water; the other half is from diet and/or toothpaste. For adults, the dietary contribution to total fluoride is generally greater than the drinking water contribution (see Table 1). The epidemiological studies assume that dietary sources of fluoride would be about equal for groups living in areas with or without fluoridated water.

Trend studies have indicated that the prevalence of dental fluorosis is increasing. Six such trend studies were reviewed and summarized by the U.S. Public Health Service in 1991. According to their review, the prevalence of dental fluorosis, in areas with low (< 0.4 mg/L) natural fluoride, increased from approximately 1% in 1939 to approximately 6% by 1985 (U.S. Public Health Service, 1991). A study of children aged 7 to 17 years in New York State found that the prevalence of dental fluorosis had increased three and a half-fold in nonfluoridated areas and two-fold in fluoridated areas (Leverett, 1986). The cause of this increase in unknown.

**Bones**

Chronic exposure of humans to fluoride in drinking water and food affects the structure of the bones. Fluoride affects the structure of long bones by increasing the deposition of bone material at the epiphyseal plates. These fluoride effects are analogous to the bone morphology changes already described for animals (U.S. Public Health Service, 1991). A histomorphometric analysis of bones from cadavers in Finland revealed that individuals whose drinking water contained greater than 1.5 mg/L of fluoride (only slightly higher than the 1 mg/L usual level in areas with...
fluoridation) had a greater amount of unmineralized bone relative to mineralized bone. These histomorphometric changes were correlated with bone fluoride content (Arnala et al., 1985). The health significance of these bone structure changes is not clear and needs further investigation.

Several ecological epidemiological studies have indicated that long-term exposure to fluoride may increase the risk of hip fracture in individuals over 65 years of age. Other studies have found no association between water fluoridation and hip fracture. A study by Jacobsen et al. (1990) found what the authors described as a "weak positive association" between the percent of county residents who receive fluoridated water and hip fracture incidence in white women over 65. In this study, soft water, poverty, reduced sunlight exposure and rural location were also positively correlated with risk of hip fracture. Danielson et al. (1992) observed a small but significant increase in the risk of hip fracture in both men and women exposed to artificial fluoridation at 1 mg/L, suggesting that levels of fluoride that are optimal for prevention of caries may increase the risk of hip fracture in the elderly.

Cooper et al. (1990, 1991) conducted a study to test the hypothesis that fluoridation of water would protect against hip fractures in the elderly. The idea that fluoridation would protect against hip fractures was suggested by laboratory observations that fluoride induced bone formation. These investigators studied hospital discharge rates for men and women 45 year of age and older in England and Wales. Initially, they found no correlation between water fluoridation and rate of hospital discharge for proximal hip fractures. After seeing the results of Jacobsen et al. (1990), they were prompted to reexamine their data. In their reexamination they weighted the data from each county according to the population of the county, giving greater weight to those counties with higher population, based on the idea that these counties would produce more precise data. As a result of this reanalysis they found a positive correlation between fluoride levels and discharge rates for hip fracture for both men and women. When hospital discharge rate was plotted against fluoride level (discharges per thousand/milligram per liter of fluoride) the regression coefficient was 0.65 for women and 0.23 for men. This work demonstrates that there is a positive dose-response relationship between fluoride level and hip fracture rate.

A study by Sowers et al. (1991) demonstrates a doubling in the risk of fractures of all sites in elderly women in a community in rural Iowa with 4 mg/L of naturally occurring fluoride compared with a community with 1 mg/L of fluoride in their water. These individuals were exposed to higher levels of fluoride in drinking water than individuals in the other studies, and the results are more pronounced.

Riggs et al. (1990) studied 163 postmenopausal women who were given 75 mg/day fluoride as treatment for osteoporosis, compared with 136 women in a control group who were given placebos. They found that the fluoride treatment increased bone mass, but at the same time decreased bone strength. The number of nonvertebral fractures was much higher in the treatment group (72 vs. 24). They concluded that fluoride treatment was not effective for postmenopausal osteoporosis.

The reason for an increase in the risk of hip fracture in the elderly due to fluoride exposure has been examined. Apparently fluoride does increase the amount of bone formation, especially in perimenopausal women, but the extra bone that is formed is weaker. Riggs et al. (1990) after studying the effect of fluoride treatment on bone mass and bone mineral density in postmenopausal women with osteoporosis, concluded that fluoride treatment increased the formation of cancellous bone but decreased cortical bone mineral density. These changes in bone structure affect the fracture risk in some bones more than others. For example, individuals treated with fluoride have
been found to have higher rates of hip fracture, but similar rates for vertebral fractures (Riggs et al., 1990). On the other hand, Sowers et al. (1991) found that women who lived in areas with very high fluoride content in their water (4 mg/L) had higher frequency not only of hip fractures, but also of fractures of the wrist and spine.

**Kidneys**

The kidney is a potential target organ because it absorbs fluoride from the blood and excretes it in the urine. Lantz et al. (1987) observed renal failure in an individual who drank two to four liters of mineral water daily with 8.5 mg/L of fluoride for approximately twenty years, and who had other symptoms of fluoride poisoning. However, several epidemiological studies on people who have consumed drinking water with as much as 8 mg/L fluoride have revealed no increased prevalence of kidney disease (U.S. EPA, 1985a). Kidney effects are not likely to be caused by drinking water with fluoride concentrations below 8 mg/L.

**Stomach**

Chronic gastritis has been reported in two studies of workers exposed to fluoride by inhalation (Medvedeva, 1983; Desai, 1986). The amount of fluoride in the air was not measured in either of these studies. In these instances fluoride must have been inhaled and then cleared from the respiratory tract into the gastrointestinal tract. There have been no reports of gastritis from nonoccupational exposures including drinking water (U.S. Public Health Service, 1991). The levels of exposure in the occupational studies were probably significantly higher than those which would be encountered from drinking water exposure.

**Reproductive Effects**

Freni (1994) studied the relationship between fluoride concentrations in drinking water and birth rates. Prior to this study there had been virtually no data on the effect of fluoride on human reproduction. The Freni study used data on fluoride in drinking water obtained from the Centers for Disease Control (CDC). Counties were identified with 3 mg/L or greater fluoride in drinking water. These counties were studied together with other counties in the same regions that had lower fluoride in drinking water. Data were obtained for each of the regions on the total fertility rate (TFR) of women in the age range 10 to 49 years. When the TFR was regressed on the fluoride concentration in drinking water, a negative slope was obtained. Fertility (as measured by TFR) was lower in counties with higher fluoride in drinking water. The consensus combined p value for this relationship was 0.0002 to 0.0004 depending on the meta-analysis method used.

This is a preliminary study only. Although suggestive epidemiological studies such as this cannot prove a causal relationship, and cannot rule out the presence of one or more unidentified confounders. This is a study on population data which should be followed by studies on individual women.

**Carcinogenicity**

There have been more than 50 epidemiological studies of the potential carcinogenic effects of fluoride in drinking water (U.S. Public Health Service, 1991; NRC, 1993). The National Cancer Institute (NCI) has recently analyzed the cancer data collected in the Surveillance, Epidemiology and End Results (SEER) program (U.S. Public Health Service, 1991). NCI found that the
frequency of osteosarcomas in adult males (over 20 years of age) increased during the period 1973 to 1987. The increase was greater in areas with fluoridated water. However, the increase in osteosarcomas did not correlate with the time of introduction of fluoridation for the areas studied, suggesting that something other than fluoridated water was responsible for the increase in osteosarcomas (U.S. Public Health Service, 1991, Appendix F). Nevertheless, the tumor type is identical to that seen in rats administered fluoride raising a risk health concern about these findings.

DOSE-RESPONSE ASSESSMENT

Noncarcinogenic Effects

When administered orally to laboratory animals, fluoride has adverse health effects on bones and teeth as well as the kidneys and stomach. Oral administration of fluoride at high levels affects survival and body weight gain. The skeletal effects of long-term exposure to fluoride are not completely understood. Experiments in laboratory animals suggest that fluoride exposure has subtle effects on the surface properties of bones. It seems plausible that changes in the mechanical surface properties of bones would have some potential health consequences but this mechanism for toxicity does not appear to have been adequately researched.

Experiments in Laboratory Animals

In a two-year rat feeding study by Procter and Gamble (Maurer et al., 1990) there were four dose groups: controls, 1.8, 4.5 or 11 mg/kg-day. The high-dose (11 mg/kg-day) rats exhibited significantly depressed weight gain in both males and females compared to the controls. The low-dose groups (1.8 mg/kg-day and 4.5 mg/kg-day) exhibited no effect on body weight gain. The depression in weight gain for the high-dose rats appears to be attributable mainly to reduced diet consumption. The no-observed-effect level (NOEL) for reduced body weight gain is therefore 4.5 mg/kg-day.

Fluoride also caused several dose-dependent adverse effects on teeth (Maurer et al., 1990), including ameloblastic dysplasia, fracture and/or malformation and enamel hypoplasia. Ameloblastic dysplasia was observed in controls and in all dose groups, but the frequency of the effect increased with dose in both male and female rats. Enamel hypoplasia also appeared in the lowest dose group in both sexes (1.8 mg/kg-day), and increased with dose in both sexes.

Orally administered fluoride has also been shown to affect bone calcification in every animal species tested, including rodents, dogs and cattle (U.S. Public Health Service, 1991). Rats given high doses of sodium fluoride (4.5 mg/kg-day or more) exhibited bones that were described as white and thick with roughened surfaces (Maurer et al., 1990). The most common microscopic effect was subperiosteal hyperostosis, a diffuse irregular deposition of mature lamellar bone in the subperiosteal regions leading to excessively thick cortices. Subperiosteal hyperostosis might lead to a change in the mechanical properties of the bone surface, possibly affecting the performance of joints.

OEHHA concludes that subperiosteal hyperostosis and ameloblastic dysplasia may be adverse effects which occurred at the lowest dose tested in this study. A no-observed-adverse-effect-level (NOAEL) cannot be determined from these data. Therefore, a lowest-observed-adverse-effect-level (LOAEL) of 1.8 mg/kg-day is identified.
Human Studies

Non-dental effects

Epidemiological studies (Riggs et al., 1990; Sowers et al., 1991; Cooper et al., 1990 and 1991; Arnala et al., 1985) have suggested that older people who have been exposed to fluoride throughout much of their lives may be more at risk for hip fractures. There is still much debate about this association in the scientific community. As bones and teeth have similar crystalline structure, it would be plausible that fluoride would affect the hardness and brittleness of bones, as it affects teeth (Arnala et al., 1985). No NOAELs or LOAELs have been identified from these studies.

The effects of fluoride on the kidneys and stomach appear to be limited to doses much higher than people would be exposed to from fluoridation of drinking water. Reproductive and developmental effects also do not seem to be a cause of concern at fluoride levels below 3 mg/L in drinking water, although more study is needed.

Dental Fluorosis

The prevalence of dental fluorosis appears to be rising (U.S. Public Health Service, 1991), possibly as a consequence of increased environmental levels of fluoride from fluoridation of municipal water systems. Dental fluorosis should not be dismissed as merely a cosmetic problem, because it may have social and psychological consequences for the development of children, and because severe dental fluorosis also affects the mechanical properties of teeth, making them more vulnerable to chipping or breaking.

The original study of the dose-response relationship between water fluoride content and dental fluorosis was done by Dean in the 1930’s and 40’s (Dean, 1942). The author reported that “mild and very mild” dental fluorosis increased from 5% to 25% as the fluoride content of the water increased from 0.4 to 1.3 mg/L. Moderate and severe dental fluorosis begins to appear at 2 mg/L, and rises to 40% at 4 mg/L (approximate numbers based on a graphical display of data). Later studies confirmed these findings, but showed dental fluorosis appearing at lower levels of fluoridation. Data collected in the 1980’s (U.S. Public Health Service, 1991; NRC, 1993) showed that the mean prevalence of dental fluorosis in four cities with optimally fluoridated water supplies was about 22% (17% very mild, 4% mild, 0.8% moderate and 0.1% severe). In another city with a water fluoride concentration in the range of 1.8 to 2.2 mg/L, dental fluorosis prevalence was 53% (23% very mild, 17% mild, 8% moderate and 5% severe).

In summary, moderate to severe dental fluorosis is rare when the drinking water fluoride level is in the range of 1 mg/L, but begins to become significant at concentrations close to 2 mg/L.

Carcinogenic Effects

The data from two rodent bioassays are equivocal. The NTP study presented evidence for an increasing trend in osteosarcomas in male rats, and osteomas were increased in the Procter and Gamble study. While osteomas are nonmalignant, they represent a phenotypic change in
osteoblasts that could have been induced by the action of fluoride or retrovirus or both acting together. This question can only be resolved by further experimentation. Given the present data, the potential for carcinogenicity of fluoride in rodents is not resolved.

The epidemiological database also raises questions about osteosarcoma in adult human males who have been exposed to fluoride in drinking water. Given the present data, one interpretation is that something other than fluoride was responsible for the differences in occurrence of osteosarcomas in young males when comparing fluoridated with nonfluoridated geographical areas. On the other hand, firm conclusion that fluoride in drinking water was not a contributing factor in these observed differences cannot be drawn at this time. The fact that slight increases in osteosarcomas were observed in animal experiments and in human studies raises the possibility of a similar carcinogenic effect across species. In view of this possibility, limiting human exposures to fluoride to those that are optimal to produce the beneficial effects would seem to be prudent.

**CALCULATION OF PHG**

The PHG (in mg/L) for fluoride can be calculated using the general formula for noncarcinogenic endpoints:

\[
PHG = \frac{\text{NOAEL} \times \text{BW} \times \text{RSC}}{\text{UF} \times \text{L/day}} = \text{mg/L}
\]

where,

- **NOAEL** = No-observed-adverse-effect-level (1 mg/L)
- **BW** = Body weight, not applicable because NOAEL is expressed in units of mg/L
- **RSC** = Relative source contribution of 100% (1)
- **UF** = Uncertainty factor of 1
- **L/day** = Volume of daily water consumption in units of L/day, not applicable in this case because NOAEL is expressed in units of mg/L.

OEHHA concludes that the critical adverse health effect for fluoride toxicity is dental fluorosis in children. In part, our determination is based on the extent of the database for fluorosis and the limited data available for other, more severe toxicological endpoints such as brittleness of bones in the elderly. Studies from 1942 onward (Dean, 1942; U.S. Public Health Service, 1991; National Research Council, 1993) have provided estimates that 1 mg/L is optimal for protection against dental caries. Above this concentration dental fluorosis begins to become significant (see Figure 1). For the purposes of developing a PHG for fluoride in drinking water, OEHHA has selected the NOAEL of 1 mg/L fluoride for the onset of dental fluorosis in children.

OEHHA assumes that the populations under study exhibited comparable dietary and hygiene exposures to fluoride. Therefore, the relative source contribution factor is 100% to account for equal exposure of study populations to fluoride in sources other than drinking water.

A UF of one is applied in the development of the PHG. The studies are of human populations and are of good quality. The most sensitive individuals to the effects of dental fluorosis (i.e., children) were included in the study population.
Therefore,

\[
\text{PHG} = \frac{1 \text{ mg/L} \times 1}{1} = 1 \text{ mg/L} = 1 \text{ ppm}.
\]

The purpose of the PHG for fluoride is to protect against dental fluorosis in children, while preserving the beneficial effect of fluoride in preventing tooth decay. Therefore, OEHHA develops a PHG of 1 mg/L (1 ppm) for fluoride in drinking water. As the PHG is based on a long-term effect (not an acute effect) the level is meant to be an approximate year-round average.

**RISK CHARACTERIZATION**

Fluoride can be distinguished from other chemicals for which PHGs are being prepared in that it may be present in drinking water not only as a “contaminant” but also a chemical which is voluntarily added because of its beneficial health effect, prevention of tooth decay. Fluoride is, however, also naturally occurring. In considering the best public health practice, the PHG should not be set so low as to unduly reduce or eliminate the health benefit, nor must it be set so high as to incur undesirable health effects. The statute allows for flexibility consideration of public health, and OEHHA interprets this to mean that health risks and health benefits of a drinking water contaminant can and should be considered in developing a PHG for fluoride.

The PHG for fluoride in drinking water is based on dental fluorosis in children as the critical effect. Dental fluorosis is an adverse health effect because in moderate form it affects children’s appearance, and in the more severe cases affects the brittleness of teeth. Moderate dental fluorosis should be avoided because it can affect children’s social interactions and self-esteem. Severe dental fluorosis can result in damaged or lost teeth. These are not life-threatening health effects.

Based on the literature (Dean, 1942; U.S. Public Health Service, 1991; NRC, 1993), 1 mg/L in drinking water is considered optimal for protection against dental caries and in the avoidance of excessive dental fluorosis (Figure 1). The dose-response curves for the beneficial effects and the adverse effects of fluoride are steep and it is difficult to determine the optimal exposure levels from these data. Therefore, OEHHA estimated that the NOAEL for fluorosis and the optimal preventative level for dental caries is 1 mg/L. This level of fluoride is also the PHG. Therefore, the PHG offers little to no margin-of-safety for variation in human sensitivity or for health effects that might occur at lower doses. If daily oral exposure to fluoride did not have a public health benefit, a lower PHG might be developed. However, because fluoride offers a public health benefit and the adverse effect at levels just above the PHG is mild fluorosis (slight discoloration of teeth), OEHHA has determined that little or no margin-of-safety is acceptable for this special case.

Decades of research and experience with naturally and artificially fluoridated drinking water in communities all over the nation and the world support the conclusion that routine use of fluoride can help prevent the formation of dental caries. However, these values are based on extrapolation from data that contain some potential for error. In addition, uncertainties still exist in several areas because individuals consume different amounts of water depending on individual habits, availability of alternatives and local climate. Individual variability might lead to a wide range of exposures that are not accounted for in the development of this PHG.
Another source of uncertainty is the added exposure to fluoride from other sources including diet, toothpaste, mouthwash and dental supplements. Parents of young children should be cautioned against exposing them to too many sources of fluoride. Excessive exposure to fluoride should also be avoided by pregnant women, especially in the latter weeks of pregnancy when the teeth of the fetus are beginning to form.

The scientific database, while extensive, raises numerous areas of public health concern regarding excess fluoridation of drinking water. In laboratory animals, there is some evidence although equivocal, that fluoride causes malignant osteosarcomas. Incidences of benign osteomas were also elevated in mice, although the authors of the study disregarded the results because of a reported viral infection. In humans, there are some data suggesting increased risk of osteosarcomas in areas with fluoridated water compared to nonfluoridated water, but validity of these results have been debated in the scientific community.

Other toxicological effects, such as effects on bone calcification have been described following oral exposure to fluoride in animals. In humans, there is suggestive evidence linking excess fluoride consumption with increased risk of hip fracture in the elderly. The evidence on this effect is not at this time sufficient to be the basis of a PHG, but if more data become available in the future, this question should be reviewed in the context of reconsideration of the PHG for fluoride in drinking water. In addition, toxicological issues such as clastogenicity and reproductive effects remain unresolved.

OTHER STANDARDS AND REGULATORY LEVELS

U.S. EPA’s MCL for fluoride in drinking water is 4 mg/L. This level was set to protect against crippling skeletal fluorosis, with a secondary MCL of 2 mg/L to protect against dental fluorosis which U.S. EPA considers a "cosmetic" rather than adverse effect (U.S. EPA, 1986). The California MCL is 1.4 to 2.4 mg/L depending on the ambient temperature (California Health and Safety Code, Title 22). The state has argued that the U.S. EPA should lower its MCL since dental fluorosis is more likely to occur at the higher level. The state argued that noticeable fluorosis is not merely "cosmetic" but may have psychological and social effects. Some communities in California have higher levels of naturally occurring fluoride in their drinking water sources, usually in the range of 2.5 to 3 mg/L. These communities have been granted exemptions from the State MCL to spare them the expense of removing fluoride from their water.
REFERENCES


California Health and Safety Code, Title 22, Article 4, Section 64435.


