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# Risk assessment for arsenic in drinking water: limits to achievable risk levels

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**Abstract**

The drinking water standard or maximum contaminant level (MCL) for arsenic is currently being re-evaluated by the US Environmental Protection Agency. The health risk associated with exposure to arsenic through consumption of drinking water is a primary concern in setting a new arsenic MCL. This paper examines the implications of arsenic chemistry, occurrence, and routes of exposure for risk assessment. In order to illustrate the relative importance of exposure through consumption of food and drinking water, the contribution of dietary intake to human exposure to inorganic arsenic was estimated as 2 μg/d. This estimate is based on a total dietary intake of arsenic of 40 μg/d and a 5% contribution of inorganic arsenic to the total dietary intake. This estimated value for dietary intake of inorganic arsenic (2 μg/d) is comparable to the exposure that would result from consumption of 2 l/d of drinking water containing 1 μg/l inorganic arsenic. At lower concentrations of arsenic in drinking water, daily intake of inorganic arsenic becomes increasingly dominated by the dietary contribution. Evaluation of standards for arsenic in drinking water should include careful consideration of exposure through other routes, particularly food consumption.

*Keywords:* Arsenic; Health risk; Risk assessment; RPHL; RPHG

## 1. Introduction

In the last few years, increasing attention has been focused on the possible long-term health risks associated with ingestion of low levels of arsenic in drinking water. In the United States, the current standard or maximum contaminant level (MCL) for arsenic in drinking water is 50 μg/l (50 ppb). This standard was originally promulgated in 1942 by the US Public Health Service and was adopted in 1975 by the US Environmental Protection Agency (USEPA) under the Safe Drinking Water Act

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[1]. Adverse health effects have been convincingly demonstrated for exposure to concentrations of arsenic in drinking water significantly above the current MCL. Recent assessments suggest that exposure at or below the current MCL may also pose unacceptable health risks to the exposed population [2–5]. In 1993, the World Health Organization recommended a provisional guideline value of 10 μg/l for arsenic based on both risk assessment and the practical quantitation limit (also estimated as 10 μg/l) [6]. With the statutory requirement for review of the arsenic MCL under the 1986 amendments to the Safe Drinking Water Act, serious consideration is being given to a significant decrease in the arsenic MCL. The range currently under consideration by the USEPA is from 2 to 20 μg As/l [1]. Compliance with these more stringent standards is expected to be very costly especially for small water supply systems [7]. Although many factors, including the practical quantitation limit and the availability of feasible treatment technologies, will be evaluated in determination of the new standard, it is clear that accurate assessment of the health risks associated with exposure to arsenic through consumption of drinking water and through other routes of exposure is crucial to this process.

Recently, a Recommended Public Health Level (RPHL) for arsenic in drinking water has been proposed by the California Environmental Protection Agency (Cal/EPA) to achieve a $10^{-6}$ lifetime excess skin cancer risk [5]. Note that, in 1994, the term RPHL was changed to Recommended Public Health Goal (RPHG); the statutory definition of the term, however, was not revised [8]. The proposed RPHL (now RPHG) is extremely low, 0.002 μg/l, a value that is lower than the current MCL by a factor of 40 000. Although a number of questions can be raised concerning such risk assessments, this paper will address the implications of arsenic chemistry, occurrence, and routes of exposure for the calculation of the RPHL (now RPHG) and the issue of the lower limit to the risk level achievable by decreasing the arsenic MCL.

## 2. Health implications of arsenic chemistry

The geochemistry of arsenic in natural waters has been extensively reviewed [9–14]. Of significance here is that arsenic is present in the environment in several chemical forms. Arsenic commonly occurs in environmental media (i.e., water, soil, air, and biota) in two oxidation states, +III and +V. The thermodynamically stable form under oxic conditions is As(V). Arsenic(III) is stable under reducing conditions and is the dominant oxidation state of arsenic in reducing groundwaters. However, As(III) may persist under oxic conditions as a metastable species as a result of slow oxygenation kinetics. Low steady-state concentrations of As(III) in oxic waters may also be maintained by biological reduction of As(V). Of the inorganic forms of arsenic, As(III) is considered more toxic [4]. There is, however, significant evidence of biologically mediated interconversion of As(III) and As(V) [7] and no distinction between these forms is made in setting water quality standards.

Arsenic also occurs in numerous organic forms of biogenic or, in the case of arsenical herbicides such as monosodium methyl arsonate, synthetic origin [10]. In

natural waters, the methylated and dimethylated species, monomethylarsonic acid $CH_3AsO(OH)_2$ and dimethylarsinic acid $(CH_3)_2AsO(OH)$, have been reported though they usually contribute only a small fraction of the total arsenic [15]. In the biota, however, arsenic is present largely in organic forms, including more complex organic species such as arsenosugars and arsenobetaine [10]. The organic forms of arsenic are unquestionably less toxic to humans than the inorganic forms [4] and, although recent evidence suggests that dimethylarsinic acid acts as a cancer promoter in rats [16], exposure to organic arsenic is not considered in assessing health risks.

## 3. Routes of exposure to inorganic arsenic

Arsenic in water is predominantly, if not entirely, inorganic. Thus, consumption of drinking water is clearly a route of exposure to inorganic arsenic and the level of exposure a function of the arsenic concentration in the water. Dietary intake of inorganic arsenic is less easily assessed. Foods vary considerably both in their total arsenic content and in the proportion of total arsenic in inorganic form. A recent comprehensive total diet study conducted in Canada has provided an estimate for the daily intake of total arsenic; the mean value obtained in this study for males and females age 12 and above was 40 μg/d [17]. This mean value is very close to those obtained for Dutch 18-yr-old males (38 μg/d) [18] and from a study of 1982–1984 total diet samples in the US (34 μg/d) for males and females age 14 and above [19].

These studies, however, report only total arsenic and not the contribution of inorganic arsenic to the total arsenic intake. In a Japanese study of dietary intake and excretion of arsenic, inorganic arsenic contributed an average of 5.7% of the mean daily intake of 182 μg total arsenic per day [20]. The higher total arsenic intake in Japan can be largely attributed to consumption of seafood; although seafood often contains quite elevated concentrations of total arsenic, the proportion of inorganic arsenic is generally low [10, 20–21]. In a study of arsenic speciation in Dutch total diet, dietary intake of total arsenic ranged from < 5 to 950 μg/d. The contribution of inorganic arsenic was assayed for samples containing elevated total arsenic (40–950 μg/d). On average, inorganic arsenic contributed 30% of the total dietary intake; the reported range was from 5% to 50% inorganic arsenic in the diet [22]. The USEPA has relied on values for inorganic arsenic in foods established by the Canadian Ministry for the Environment, which indicate that approximately 20–25% of the total dietary arsenic intake is as inorganic arsenic [23]. It is clear that dietary intake represents some finite contribution to human exposure to inorganic arsenic and that the exposure through food consumption, unlike exposure through consumption of drinking water, is independent of the concentration of arsenic in drinking water.

In a recent Canadian evaluation of the health risks of exposure to inorganic arsenic, the daily intake through the diet was estimated to be approximately equal to intake through drinking water consumption for the general population [24]. In this study, arsenic exposure through air inhalation was also evaluated but was considered to

be negligible. The relative contributions of food and water consumption to inorganic arsenic exposure can be calculated as a function of the concentration of arsenic in drinking water (which is assumed to be entirely inorganic) if a reasonable value for the dietary intake of inorganic can be estimated. Here, the daily dietary intake of inorganic arsenic is assigned a value of 2 μg/d based on a 5% contribution of inorganic arsenic to a daily dietary intake of total arsenic of 40 μg/d. Note that this estimate of dietary exposure is conservative relative to other estimated and reported values [20, 22–23] including the Canadian estimate [24] of 5.6 μg/d (reported as 0.08 μg/kg d for an average body weight of 70 kg). The estimated value used here is also much lower than the provisional maximum tolerable daily intake of

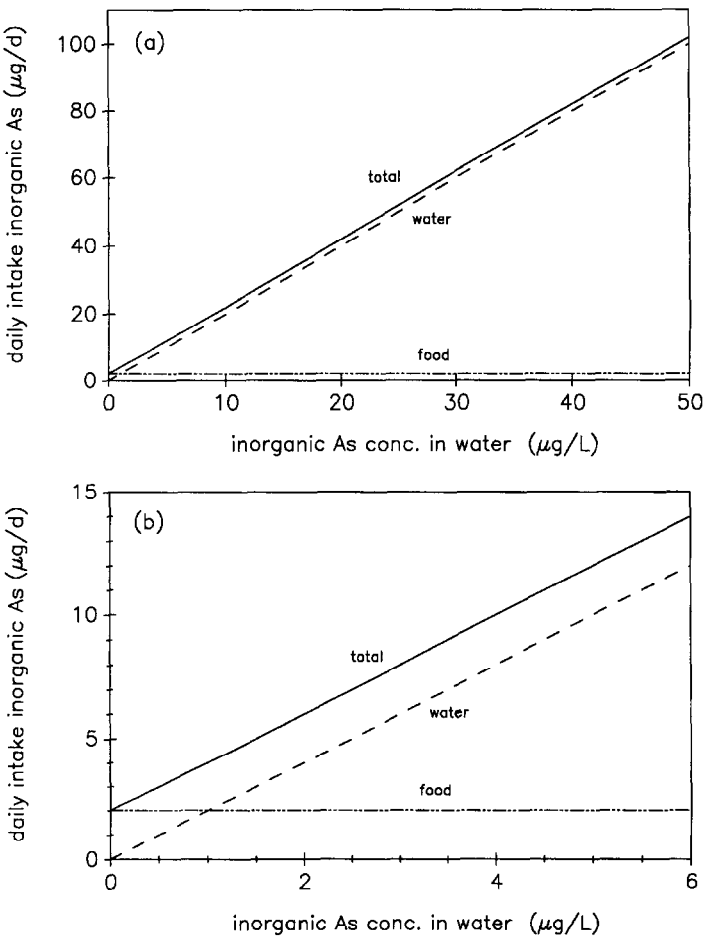


Fig. 1. Contributions of food and water consumption to the total daily intake of inorganic arsenic shown as a function of the arsenic concentration in drinking water (assumed to be 100% inorganic arsenic). Daily intake from food and water calculated based on water consumption of 2 l/d and daily dietary intake of 40 μg/d total arsenic of which 5% is inorganic. (a) Concentration range shown up to current MCL of 50 μg/l; (b) expanded concentration scale.

inorganic arsenic of 2 μg/kg body weight or 150 μg/d for an average body weight of 70 kg established by the Joint FAO/WHO Expert Committee on Food Additives (JECFA) [6].

With this conservative estimate (2 μg/d) for dietary intake of inorganic arsenic, exposure through consumption of drinking water clearly overshadows exposure through food consumption if the arsenic concentration in drinking water is at or near the current MCL of 50 μg/l as shown in Fig. 1(a). The scale of this figure, however, obscures the relative importance of dietary intake when the arsenic concentration in drinking water is low. As shown in Fig. 1(b), the estimated daily intake through consumption of water and food are calculated to be equal when the arsenic concentration in drinking water is 1 μg/l; below this value, the contribution from diet becomes more important than the contribution from drinking water consumption. An alternative way of examining this information is to consider the percentage of the inorganic arsenic intake that is derived from drinking water consumption. As shown in Fig. 2, this value approaches 100% at the current MCL but drops dramatically at low concentrations of arsenic in drinking water. In this calculation, the concentration of arsenic in drinking water for which exposure through food and water are equally important is obviously sensitive to the estimated dietary intake of inorganic arsenic. In Table 1, results of this calculation are shown for varying estimates of dietary intake of inorganic arsenic. Clearly, the value of the current MCL is most consistent with the value of the provisional maximum tolerable daily intake for inorganic arsenic in food.

Even on the expanded (linear) concentration scale of Figs. 1(b) and 2(b), it is difficult to illustrate the potential benefit (or lack of benefit) that might be obtained by the imposition of extremely low drinking water standards for arsenic. At the proposed RPHL (now RPHG) of 0.002 μg/l, the contribution of drinking water consumption to the total, daily intake of inorganic arsenic is clearly insignificant compared to food consumption (Fig. 3); even conservatively estimated, dietary intake is more than a factor of 100 greater than intake through drinking water consumption. Obviously, the total exposure to inorganic arsenic cannot be decreased

Table 1
Concentrations of arsenic in drinking water at which exposure through consumption of drinking water is equal to exposure through consumption of food

| Dietary intake of inorganic As (μg/d) | Basis for dietary intake value | Inorganic As concentration in water giving equal exposure through water[a] and food (μg/l) |
|---|---|---|
| 2 | 40 μg/d total arsenic [17–19]<br>5% inorganic arsenic [22] | 1 |
| 5.6 | Canadian estimate [24] | 2.8 |
| 150 | Tolerable daily intake[b] | 75 |

[a] Water consumption 2 l/d.
[b] Based on JECFA provisional maximum daily tolerance [6].

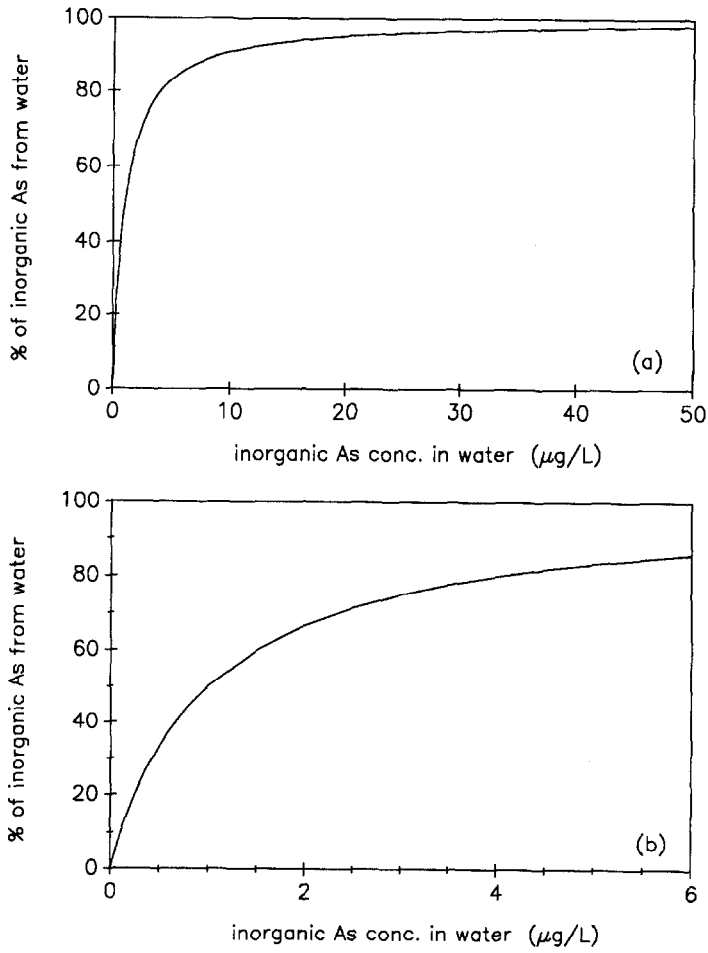


Fig. 2. Percent of total daily intake of inorganic arsenic derived from drinking water consumption as a function of the arsenic concentration in water. All parameter values are as in Fig. 1. (a) Concentration range shown up to current MCL of 50 μg/l, (b) expanded concentration scale.

below the level of exposure from consumption of food even at vanishingly small arsenic concentrations in drinking water.

## 4. Consequences for calculation of the RPHL (now RPHG)

Calculation of the RPHL (now RPHG) as described by Brown and Fan [5] assumes that the fraction of environmental arsenic exposure due to drinking water consumption (F) has a fixed value (of 0.2 or 20%) that is independent of the arsenic concentration in drinking water. Comparison with Fig. 2(b) indicates that this approach is likely to provide an underestimation of the exposure due to drinking

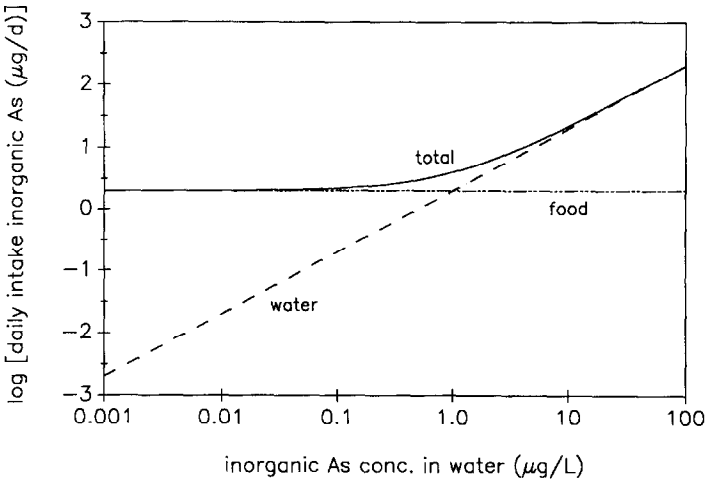


Fig. 3. Contributions of food and water consumption to the total daily intake of inorganic arsenic as a function of the arsenic concentration in drinking water shown on a logarithmic scale to emphasize the low concentration range. All parameter values are as in Fig. 1.

water consumption at or near the current MCL and an overestimation at very low arsenic concentrations in drinking water. Rearranging the formula for calculation of the RPHL (now RPHG) given by Brown and Fan [5] yields the expression for excess risk, $R$,

$$R = \frac{(C)(W)(q)}{(BW)(F)}$$

as a function of the arsenic concentration in drinking water ($C$ in μg/l), daily water consumption ($W$ = 2 l/d), human cancer potency ($q = 5.3 \times 10^{-3}$ (μg/kg d)$^{-1}$), average body weight (BW = 70 kg) and $F$ (fraction of environmental arsenic exposure due to drinking water consumption).

As shown in Fig. 4, the calculated excess risk is markedly affected by whether the factor $F$ is assumed to be constant or is taken to be a function of the arsenic concentration in drinking water (cf. Fig. 2). The contributions of exposure through consumption of food and drinking water are also shown separately (based on the daily intake values shown in Fig. 1); risks from these two exposure pathways are assumed to be additive. Again, the results of these calculations are shown on a logarithmic scale to emphasize the low concentration range. The significance of these results is that a calculated excess risk of $10^{-6}$, the target risk level for the proposed RPHL [5], can be achieved at extremely low concentrations of arsenic in drinking water (ca. 0.002 μg/l) only if the fraction of environmental arsenic exposure due to drinking water consumption ($F$) is assumed to be constant. This risk level cannot be attained if the factor $F$ is calculated as a function of the arsenic concentration in drinking water given a constant value for the (conservatively estimated) daily dietary intake of inorganic arsenic. Thus, at (or even

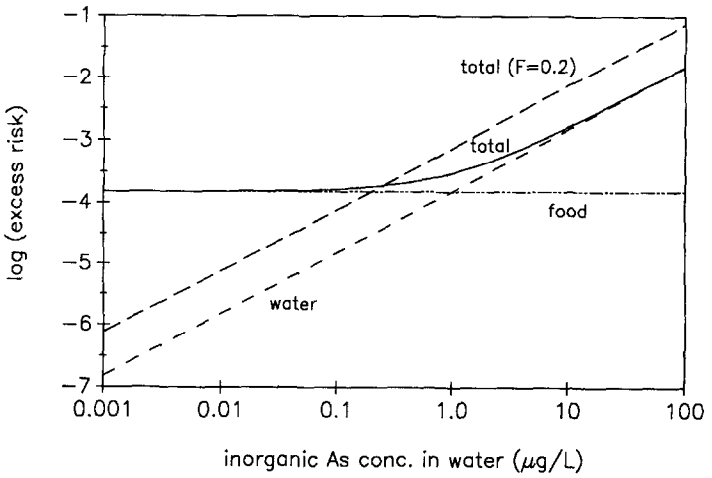


Fig. 4. Calculated excess risk as a function of arsenic concentration in drinking water. Excess risk calculated based on parameters from Fig. 1, human cancer potency of $5.3 \times 10^{-3}$ $(\mu g/kg\ d)^{-1}$, average body weight of 70 kg. Risks derived from exposure through consumption of food (– – –) and water (– –) are assumed to be additive in calculating total risk (—). Calculation of total risk assuming fixed value of $F$ (of 0.2) also shown (— —) after Ref. [5].

100 times above) the proposed RPHL, the minimum achievable risk is that due to exposure through food consumption and this risk level cannot be significantly improved by lowering the drinking water standard for arsenic. A previous calculation of annual cancer cases in the US from arsenic ingestion based on a higher dietary contribution to exposure similarly indicated that even the complete elimination of arsenic from drinking water could effect only a limited reduction in the expected cancer rate; estimated uncertainties in these calculations were significant [25].

## 5. Further questions concerning the RPHL (now RPHG) calculation

The argument made above concerning the minimum achievable risk assumes that the daily dietary intake of inorganic arsenic is constant and independent of the arsenic concentration in drinking water. This assumption is not unreasonable since arsenic in foods may be derived from soils, directly in the case of plants or ingestion of soil by grazing animals or indirectly by transfer through the food chain, from untreated agricultural irrigation water, or from medicated animal feeds. The currently allowed use of arsenicals as medicating agents for animal feeds and drinking water to increase weight and control disease in poultry and swine [26] should be re-evaluated. The calculated value of the minimum achievable risk level (1.5 in 10 000) is directly dependent on the estimated daily dietary intake of inorganic arsenic. The estimated value used here (2 μg/d) was chosen conservatively for illustrative purposes and is certainly subject to debate. Although the bioavailability of arsenic in food

has been extensively documented [20, 22, 26, 27], the assumption, implicit in this work, that inorganic arsenic in water and food are absorbed in the gastrointestinal tract with similar efficiencies should be carefully examined. It is clear, however, that Draconian standards for arsenic in drinking water should not be imposed without careful consideration of exposure through other routes, particularly food consumption.

The value for the human cancer potency for inorganic arsenic used here is also open to question. Some of the uncertainties regarding this human cancer potency value are discussed by Brown and Fan [5]. Clearly, this parameter is crucial to the estimation of risk but discussion of the uncertainties in this parameter are beyond the scope of this paper. The general conclusion of this paper regarding minimum achievable risk will, however, be valid even if the value of the human cancer potency is re-assessed if the assumptions that (1) the same value for human cancer potency can be applied for inorganic arsenic ingested in water and food and (2) the risk associated with ingestion of inorganic arsenic in either food or water is linearly related to daily intake are not invalidated.

## 6. Concluding remarks

This paper examines the implications of alternative routes for exposure to inorganic arsenic for accurate risk assessment and evaluation of the RPHL (now RPHG). Exposure to inorganic arsenic through consumption of food and drinking water is considered. With a conservative estimate of the daily dietary intake of inorganic arsenic, consumption of food and of drinking water are calculated to contribute equally to exposure to inorganic arsenic at a concentration of arsenic in drinking water of 1 μg/l. Below this value, the daily intake of inorganic arsenic becomes increasingly dominated by dietary intake. As a consequence, the minimum achievable risk level is determined by the dietary intake of inorganic arsenic and becomes independent of the concentration of arsenic in drinking water.

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