Interpretation of drinking water quality guidelines – The case of arsenic

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Abstract

Drinking water quality guidelines are often interpreted by the non-expert as make or break cut-off values below which drinking water is absolutely safe, and above which it is totally unacceptable. In reality there is no such knifelike cut-off limit, and there is a large grey area between safe water and undrinkable water. The uncertainty of the boundaries of the grey area for each constituent presents a serious problem, both in the creation of sound drinking water quality guidelines or standards, and in the problem of how to interpret the risk to human health when guideline values are exceeded. In this paper this problem is discussed using the case of arsenic, where the definition of the boundaries of the grey area is particularly uncertain.

Keywords: drinking water quality, guideline interpretation, arsenic, uncertainties

Introduction

In order to ensure safe drinking water, and gauge the quality of treated drinking water, guidelines or standards, in terms of concentration of determinands, are customarily defined with which to evaluate the safety of drinking water (SANS, 2005; WHO, 2004). Water service providers are obliged to comply with appropriate water quality standards (DWAF, 2005). The conventional drinking water treatment process of flocculation, sedimentation, filtration and chlorination, is at times hard-pressed to remove trace constituents to levels which satisfy the South African Class 0 (ideal) of the SABS 241:2001 drinking water standard, which in the case of arsenic is set at 10 µg/ℓ (SABS, 2001). According to the SABS 241:2001 classification system the Class 1 acceptable standard of 10 to 50 µg/ℓ is also deemed safe for lifetime use. Drinking water authorities are put under great pressure by the public to try and achieve Class 0 (ideal) drinking water quality. Yet the reaching of the stricter level, particularly where the source water contains significant concentrations of arsenic from typically natural causes, presumably due to oxidation of arseno-pyrite, entails careful optimisation of the treatment process and particularly effective filtration to remove fine suspended particulate material. Such careful optimisation of the treatment process has significant cost implications, both in instrumentation needed for monitoring and analysis and costs of treatment chemicals as well as trained operators (DWAF, 2002).

This paper discusses the implications of the uncertainties regarding the relationship between concentrations of the substances in the water and their health effects in interpretation of drinking water quality guidelines. These uncertainties are particularly evident in the case of arsenic.

Health effects of arsenic

The subject of arsenic in the diet is one which is associated with dread by many, as it is a substance which has been the basis of many historical poisonings, including the death of Napoleon Bonaparte on the island of St. Helena (Weider and Fournier, 1999) and the madness of King George III of Britain (Cox et al., 2005). Arsenic is both a toxic substance in excess as well as a carcinogen by inhalation (EPA, 1992). Despite hundreds of studies on arsenic's health effects, the mechanism of action remains uncertain, partly due to the lack of a really suitable animal model, but also because of the contradictory findings in different studies which seem to characterise arsenic epidemiology, and it is still not clear whether arsenic is a primary carcinogen or a co-carcinogen, requiring a carcinogenic partner (Rossman, 2003). Arsenic can give rise to skin cancers, the most obvious with drinking water exposure to elevated arsenic concentrations, as well as to cancers of the lung, bladder and liver (Morales et al., 2000). Ingesting arsenic at concentrations from 300 to 30 000 µg/ℓ will cause noticeable health effects, and concentrations above 60 000 µg/ℓ can be lethal (ATSDR, 2000).

One of the major challenges in establishing valid dose-response relationships and especially safe threshold levels for arsenic is that there are not only large interspecies differences, but also genetic variations within humans as to how arsenic is metabolised, which strongly affect susceptibility to the toxic effects of arsenic (Vahter, 1994; Westerfeldt et al., 2001).

To make the understanding even more problematical is the fact that the previously well-accepted hypothesis that toxic inorganic trioxide is detoxified in the body by methylation, has been shown to probably do the reverse, i.e., that the methylation process actually activates arsenic as a toxin and (co)carcinogen (Styblo et al., 2002).

Drinking water guideline limits for arsenic

The need for sound drinking water guidelines for arsenic is nowhere better illustrated than in the tragedy of the mass chronic arsenic poisoning occurring in Bangladesh and West Bengal where millions of people have been exposed to drinking water containing in excess of 50 µg/ℓ arsenic and where many show symptoms of chronic arsenic toxicity with both skin and internal organ lesions (Rahman et al., 2001).

It is instructive that the WHO limit for arsenic was originally set at 200 µg/ℓ in 1958, based on avoidance of arsenic toxicity, but which was lowered to 50 µg/ℓin 1963, and still lower

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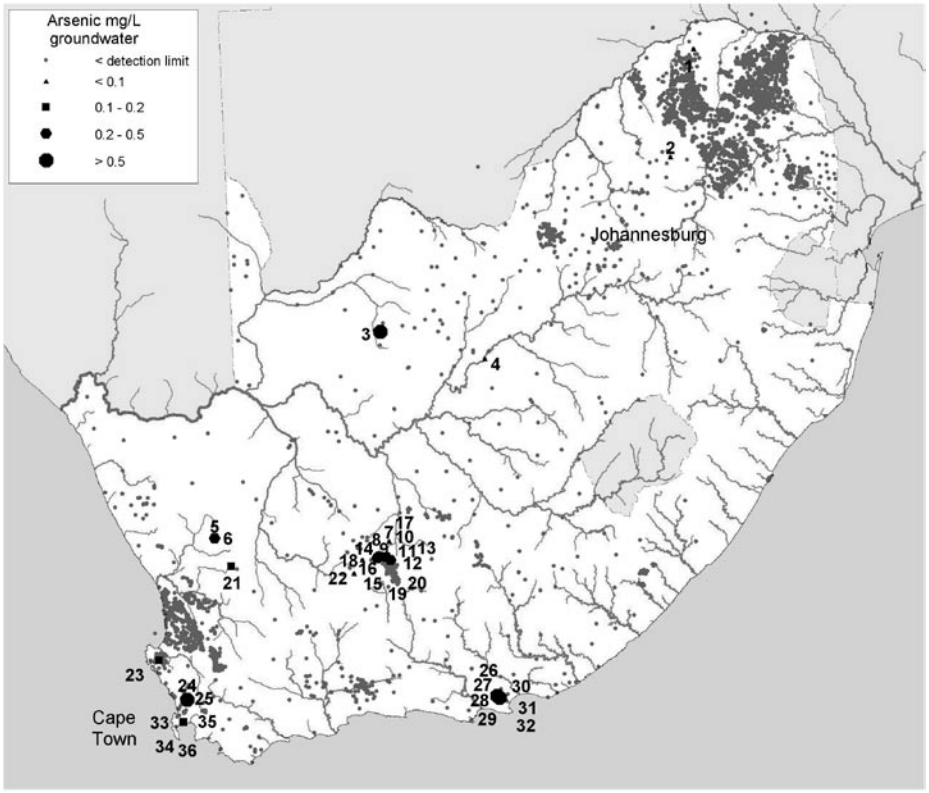

Figure 1
Groundwater arsenic data for South Africa

to the practical analytical quantitation limit of 10 µg/ℓ in 1993, which is still regarded as a provisional limit. This lowering of the drinking water guideline for arsenic has been largely due to the fear of an unknown risk of cancer induction, from animal studies which showed arsenic to be a proven carcinogen in animals (WHO, 2004; EPA, 1992). The European Union limit for arsenic in drinking water is also set at the WHO limit of 10 µg/ℓ (Massey, 2001), while Canada specifies 25 µg/ℓ(Health Canada, 2004).

There is considerable uncertainty on what the actual risk of cancer in humans is from low levels of arsenic in drinking water. By re-analysing the epidemiological data from the endemic region in Taiwan using several different models, Morales et al. (2000) found the uncertainty of the 1% excess cancer risk (ED_{01}) of lung cancer from arsenic in males to lie between 10 and 364 µg/ℓ arsenic in drinking water, and for bladder cancer the ED_{01} to be in the range of 21 to 633 µg/ℓ. Clearly it is important from the viewpoint of safeguarding human health to set the drinking water guideline limit at the lower limit of the observed range of 1% excess cancer risk, as other studies have shown that arsenic in combination with other carcinogen-inducing factors such as solar-UV radiation do show effects at lower concentrations of arsenic than isolated arsenic ingestion (Rossman, 2003). Thus the 1958 WHO guideline for arsenic of 200 µg/ℓ may be protective in the absence of concomitant carcinogens, but the current 10 µg/ℓ guideline is clearly necessary in view of the fact that humans are exposed to many potential carcinogens in their total diet, apart from solar-UV radiation.

The current South African standard for arsenic in drinking water is set at 10 µg/ℓ for Class 0 water (ideal), at 50 µg/ℓ for Class 1 water (acceptable) and at 200 µg/ℓ for Class 2 water (maximum allowable for a limited period of 3 months) (SABS, 2001). The South African drinking water standard is currently under revision and the indications are that the maximum limit for arsenic will be lowered to 50 µg/ℓ (SANS 241, 2005)

Monitoring of arsenic in South African water resources

There is currently no formal national monitoring programme in place to report on the status of and trends in arsenic levels in South Africa's water resources. Available data are based on a number of surveys that were done by the Department of Water Affairs and Forestry (DWAF) in surface water and groundwater. The data are thus quite patchy, both temporally and spatially. The ability to measure arsenic concentrations at the Class 0 level has also been inconsistent. From 1984 to 1993, the detection limit was 100 µg/ℓ. This improved to 50 µg/ℓ in 1993 but ageing analytical instruments resulted in a deterioration of the detection limit to 100 µg/ℓ (in 2000), 125 µg/ℓ(in 2002 to 2004) and 200 µg/ℓ (in 2005). The detection limit would need to be 20 µg/ℓor better for the data to be of use for epidemiological analysis.

A synoptic view of arsenic data for 8 380 groundwater samples is shown in Fig. 1. Most groundwater samples had no detectable arsenic (at the detection limits indicated above), and there are only a few sites which showed arsenic values in excess of 200 µg/ℓ which would be of immediate concern to health (see Table 1 for a complete list of samples that exceeded the detection limit at the time of analysis). The 4 sites that showed toxic levels of arsenic in the red class of >200 µg/ℓ were:

- Point 3, a borehole near Sishen: 520 µg/ℓ As on 1 January 1989
- In the Victoria West area, Point 14, Vingerfontein: 341 µg/ℓ As on 12 October 1998 and Point 10, Ruigtefontein: 207 µg/ℓ As
- Point 29, Site 3325CD00310 G & I in the Eastern Cape with arsenic in the range 134 to 1 664 µg/ℓ As for 14 samples taken over the period January 1997 to May 2001
- Point 24, Site 3318DA00363 in the Western Cape with 10 000 µg/ℓ As on 3 November 1999.

A synoptic view of arsenic data for 6 360 surface water samples is shown in Fig. 2. This shows that most of the instances of elevated arsenic in surface water have occurred in the industrialised area in and around Gauteng, due presumably to industrial effluent contamination events. Two incidents of note were an arsenic value of 850 µg/ℓ for the Roodeplaat Canal (Point 42) on 1st December 1989 and an arsenic value of 1233 µg/ℓ in water from Welbedacht Dam (Point 54) on 5 June 1984. Arsenic is common in arsenopyrite mineralisation in association with iron, so it can dissolve under anaerobic and acidic conditions to create temporary high spikes in observed arsenic concentrations.

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Discussion

In view of the uncertainty at which real risks to consumers arise from arsenic in drinking water, water treatment authorities need to understand the importance of ensuring that their treated supplies do not contain arsenic above the limits set by the South African Standard (SABS, 2001; SANS, 2005), and that they should aim at ensuring that they meet the standard of 10 µg/ℓ arsenic in drinking water, with the proviso that: excursions of up to 50 µg/ℓ are allowed, for a period of up to one year. While skin cancers arising from exposure to arsenic are easily treatable, this is not the case with lung and bladder cancers which lead to much suffering to affected individuals.

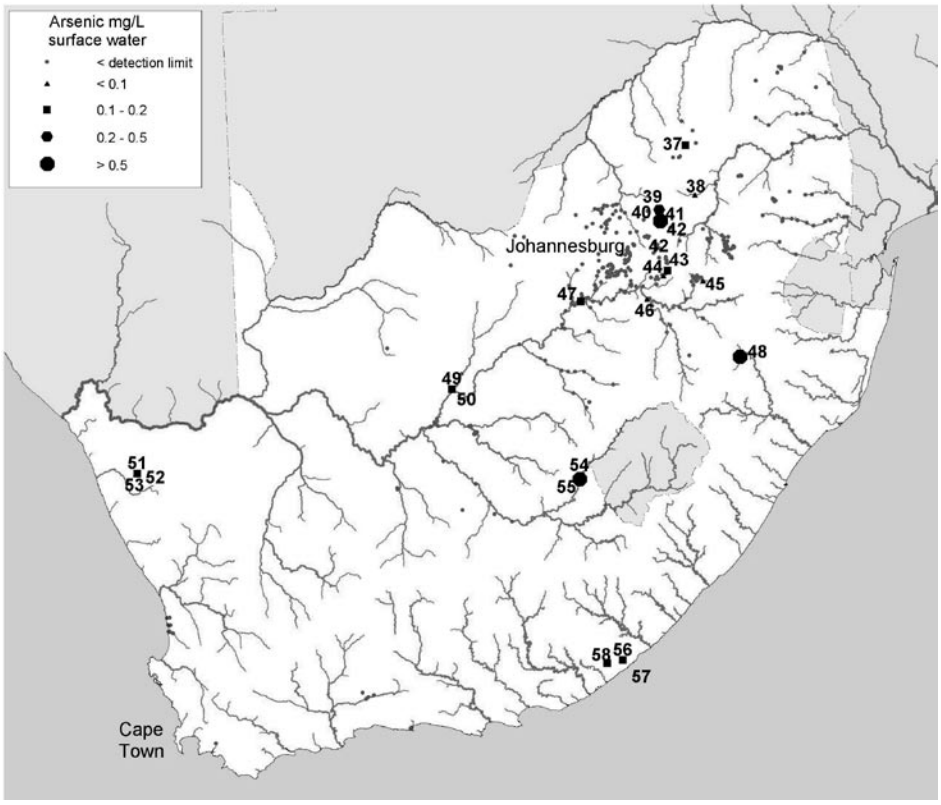

Figure 2
Surface water arsenic data for South Africa

Arsenic is not usually present in elevated concentrations in surface waters, unless these have been affected by mining and effluent discharges. Industrial water use and wastewater discharges are regulated in terms of the National Water Act (Act No 36 of 1998) and point sources of pollution are well under control. The real risk of arsenic presence in water lies with groundwater supplies, particularly in areas where arsenic-containing minerals are found. It has been a standing policy within our Department of Water Affairs and Forestry that all new boreholes intended for use for drinking water should be tested for acceptability for use, particularly with respect to the arsenic, nitrate and fluoride concentrations in addition to the usual total dissolved salts (DWAF, 1998).

A worrying feature of risk information published on arsenic is that the risk is determined for a very specific scenario and using a very specific epidemiological model. The assumptions used in the derivation of the risk figure are often forgotten and the numerical risk itself is thereafter taken as absolute truth. In the exhaustive review of arsenic carcinogenesis by Rossman (2003) many cases occurred where the threshold for observation of carcinogenesis in animals was strongly influenced by many factors, including genetic and dietary factors, as well as the exposure to co-carcinogens and vitamins. This implies that the risk factors cannot be used as generalisations. For instance, despite the hundreds of articles on observed arsenic carcinogenesis in animals, it is still not certain that arsenic is a primary carcinogen in humans (Rossman, 2003). Yet the modelled risk level for arsenic- induced cancer at 10 µg/ℓ has been quoted as being as high as 1 in 1 700 (Massey 2001) or 1 in 500 at 50 µg/ℓ (Morales et al., 2000). The uncertainties of risk assessment epidemiological models imply, if they are taken at face value, that arsenic in drinking water may be a leading cause of cancer in humans, as the safety factors for organic carcinogens such as pesticides are normally managed at the 1 in 1 000 000 risk level (Massey, 2001).

It is frustrating to say that neither the level at which arsenic carcinogenesis kicks in nor the mechanism of action is clearly understood (Rossman, 2003; Morales et al., 2000), nor the reasons for why a small percentage of individuals are particularly susceptible to the toxic effects of arsenic (Westerveldt Check spelling et al., 2001). Ideally epidemiological studies should be conducted to establish the applicability of available risk assessment models to the South African situation.

Despite these fears, arsenic does have potentially positive benefits in the medical and nutritional fields, and there is already strong evidence that arsenic may be essential in small quantities in certain animal species. It is, however, not yet known in what form the arsenic is best taken, nor what the exact beneficial amount is (Nielsen, 1998; Munshi et al., 2002). Tarello (2001a) has shown that chronic fatigue syndrome in cats can be rapidly brought into complete remission using treatment with small doses of arsenic. This syndrome, with elements of immune suppression resembling HIV disease in humans, can be effectively brought into remission in birds (Tarello, 2001b), and in dogs as well (Tarello, 2001c) using small-dose arsenic treatment. Arsenic has been used in human medicine for centuries especially for treating parasitic infections (Munshi et al., 2002). In relation to the human HIV/AIDS pandemic, Fincham et al. (2003) and Adams et al. (2005) have postulated that soil-transmitted helminthic infection could be a significant risk factor. The intriguing possibility thus exists that dietary arsenic intake in food and water may be a negative risk factor in the HIV infection. However, the form in which the arsenic is present is probably critical due to the large differences in metabolic pathways of different arsenic species (Styblo et al., 2002). Toxicologists believe that inorganic arsenic is mainly responsible for cancer whereas arsenobetaine or 'fish arsenic' is considered relatively non-toxic (Ryan et al., 2001). A typical Japanese fish-rich diet contributes as much as 195 µg/d arsenic (Yamauchi et al., 1992), whereas an American diet only contributes around 28 µg/d (Ryan et al., 2001).

TABLE 1
Raw data for all monitoring points where arsenic exceeded the current detection limit

Point	Description on DWAF database		Date	As (μg/ℓ)
1	ZQMALL2 2229AC00129 Alldays	(borehole)	1999-04-19	82
2	ZQMNAB4 2428DA00613 Groot Valley – Naboomspruit	(borehole)	1999-04-26	80
3	ZQM783HAL Borehole on Halliford near Sishen D4N783Q01	(borehole)	1985-08-05	126
			1989-06-01	520
4	Abramsyskraal	(borehole)	2000-02-07	93
5	3019CD00056 Loeriesfontein Meent	(borehole)	2000-03-17	136
			2000-03-17	155
			2000-03-18	209
			2000-03-20	149
6	3019CD00057 Loeriesfontein Meent	(borehole)	2000-03-25	162
7	3123AC00226 Victoria West Allotment	(borehole)	2000-03-29	74
			2000-03-29	64
8	Vingerfontein 162	(borehole)	1999-02-09	61
			1999-02-11	72
9	Vingerfontein 162	(borehole)	1999-02-03	100
			1999-02-05	280
10	Ruigtefontein 169	(borehole)	1999-06-11	207
11	Ruigtefontein 169	(borehole)	1999-07-23	202
12	3123AC00208 Ruigtefontein	(borehole)	1999-12-04	87
			1999-12-05	76
			1999-12-08	90
13	Ruigtefontein 169	(borehole)	1999-07-21	189
14	Vingerfontein	(borehole)	1998-10-10	78
			1998-10-12	222
			1998-10-12	341
15	Vingerfontein	(borehole)	1998-09-30	117
			1998-10-09	239
16	Vingerfontein	(borehole)	1998-10-13	154
17	3122BD00111 Vingerfontein	(borehole)	1999-11-25	66
18	Taaiboschfontein	(borehole)	1998-08-19	65
19	Victoria West Allotment	(borehole)	1999-04-26	150
			1999-04-27	241
20	Victoria West Allotment	(borehole)	1999-06-14	110
21	ZQMCLV1 3119BD00036 Calvinia Allotment - CT59	(borehole)	1999-05-03	140
22	Duikerfontein	(borehole)	1999-10-16	62
23	3218CC00387 Langeberg G46063	(borehole)	2001-05-04	116
24	ZQC000051 3318DA00363 Vryheid - 51/1B	(borehole)	1999-11-03	10 000
25	3318DA00118 Mikpunt 181/1B	(borehole)	2002-02-12	59
26	G & I	(borehole)	1996-10-21	267
27	Kruisrivier: G & I	(borehole)	1995-09-27	363
			1996-04-24	234
28	Kruisrivier: G & I	(borehole)	1999-09-09	400
29	3325CD00310 ZQCSKR3 G & I - G40036	(borehole)	1997-01-28	1 664
			1997-04-24	1 439
			1997-07-23	600
			1997-10-29	1 133
			1998-03-05	134
			1998-09-02	280
			1998-12-07	543
			1999-03-04	441
			1999-12-02	597
			2000-03-25	699
			2000-07-10	1 182
			2000-10-10	1 200
			2001-01-05	1 094
			2001-05-01	646
30	C G H	(borehole)	1996-08-23	469
			1996-10-23	444
31	3325CD00315 ZQCSKR9 C G H Ponds - G40042	(borehole)	1997-10-28	1 269
			1998-03-04	188
			1998-09-03	347
			1998-12-07	226
			1999-03-04	307
			1999-09-10	252
			1999-12-01	353
			2000-03-24	274
			2000-07-11	244
			2000-10-10	379
			2001-01-05	424
32	3325CD00313 ZQCSKR8 Uitenhage Sewage Works - G40041	(borehole)	1995-09-28	212
			1995-11-21	272
			1996-04-24	262
33	ZQM196MPL 3418BA00001 Mitchell's Plain - G32963 G2N196Q01	(borehole)	1986-03-13	121
34	ZQM199MPL 3418BA00018 Mitchell's Plain - G 32966 G2N199Q01	(borehole)	1986-03-13	114
35	ZQM200MPL 3418BA00037 Mitchell's Plain - G 32967 G2N200Q01	(borehole)	1986-03-13	139

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36	ZQM198MPL 3418BA00020 Mitchell's Plain - G032965 G2N198Q01	(borehole)	1986-03-13	139
37	A6H025R01 Makopane: Doorndraai Dam raw water	(WPW)	1989-12-06	150
38	B3H010R02 South Ndebele: Renosterkop Dam raw water	(WPW)	1993-03-01	57
39	A2H070R01 Klipdrift: Roodeplaat Dam raw water	(WPW)	1990-04-03	404
40	A2H070S01 Klipdrift: potable water	(WPW)	1984-12-05	105
			1990-04-03	177
41	A2H071S01 Wallmannsthal potable water	(WPW)	1989-11-01	950
			1989-12-01	780
			1990-02-28	271
			1990-04-03	526
42	A2H071R01 Wallmannsthal: Roodeplaat Dam raw water	(WPW)	1989-12-01	850
			1990-02-28	653
43	C2H183Q01 R42 Delmas-Nigel road bridge	(river)	1999-02-15	160
44	C2H185Q01 Poortjie Road on Blesbokspruit	(river)	1994-08-02	50
45	C1H033Q01 RESM 8 Klipspruit upstream Secunda	(river)	1995-08-23	50
46	C1H013R01 Vaal Dam raw water	(WPW)	1993-02-01	53
47	ZKOEK-BUF Koekemoer Spruit at Buffelsfontein	(river)	1994-11-07	171
48	V3H021R01 Ngagane Treatment Works - Chelmsford Dam raw water	(WPW)	1984-07-03	820
49	C9H012R01 Vaal Gamagara Treatment Works - Vaal River raw water	(WPW)	1984-02-02	120
50	C9H012S01 Vaal Gamagara Treatment Works - treated water	(WPW)	1984-06-02	115
51	6 Springbok Municipal Area SHC sump	(river)	2002-07-29	200
			2002-10-02	100
52	7 Springbok Municipal Area downstream of N7	(river)	2002-10-02	80
53	1 Springbok Municipal Area commonage boundary	(river)	1997-03-18	25
54	D2H021R01 Caledon / Bloemfontein: Welbedacht Dam raw water	(WPW)	1984-06-05	1 233
			1993-01-04	56
55	D2H021S01 Caledon / Bloemfontein: treated water	(WPW)	1984-01-10	427
			1984-12-03	101
56	R3H002R01Nahoon Dam raw water	(WPW)	1984-11-05	113
57	R3H002S01Nahoon Dam treated water	(WPW)	1984-11-05	111
			1984-12-03	106
58	R2H014S01 Middle Buffalo: treated water	(WPW)	1986-02-03	107

WPW = Water purification works
The complete data set is available on request from the South African Department of Water Affairs and Forestry.

If it is assumed, for the sake of argument, that the Japanese diet, high in fish intake, represents the dietary picture for early man who lived close to the sea, then on the basis of the safe drinking water arsenic intake being 10% of the total dietary intake, the safe arsenic drinking water limit would be around 10 µg/ℓ As for a 2 ℓ per day water intake for an adult, and 195 µg/d As from food intake (Yamauchi et al., 1992). Now this is exactly at the current WHO (2004) guideline for arsenic, which probably is confirmation that it is a reasonable limit in terms of safety with respect to the total diet. However, should the diet be deficient in fish intake, then 10 µg/ℓ. As may well be a deficiency level for arsenic and below the level of optimal benefit to human health and well-being, and the Canadian limit of 25 µg/ℓ may be more appropriate.

Knowing the typical food basket total dietary intake of arsenic is important in reaching balanced decisions on what the drinking water standard or guideline for a naturally occurring constituent should be. Unfortunately the role of naturally occurring arsenic in fish and water in human diets and health is not known, but evidence from animal and human studies shows that it may play a significant health role in small concentrations (Nielsen, 2000). There is an unfortunate trend in drinking water guidelines for naturally occurring substances to be reduced as the years pass, on the grounds of carcinogenicity at unrealistically high doses in animal experiments. Standard linear extrapolation models for calculating cancer risks at low dose exposure to carcinogens almost certainly seriously overestimate risk, as about half of chemicals tested for carcinogenicity in rodents give a positive response at high dose levels, and the response at low dose is fundamentally different (Ames and Gold, 1997). Where water is the vehicle for an essential trace element, excessively low drinking water guidelines based on linear extrapolation risk assessment models may well do the consumer a disservice, as well as raising untested and alarmist fears.

In view of the growing incidence of HIV infection in humans, as well as associated syndromes where immune dysfunction plays a role, such as chronic fatigue syndrome in humans and animals, it is important for water suppliers not to induce trace mineral deficiencies through drinking water that is too pure and devoid of minerals. This is particularly a risk from the modern popularity of point-of-use reverse osmosis units, which reduce the mineral content of water and particularly trace elements to very low levels. The ideal level of intake of naturally occurring mineral elements may be in a small window of concentration between deficiency and toxicity. For example, the element selenium, adjacent to arsenic on the periodic table, is toxic at mg/ℓ concentrations, but is required in the diet in small amounts (about 20 µg/ℓ: Oldfield, 1987).

Conclusion

This paper has shown the importance of interpreting drinking water guidelines on a very broad front, and the importance of not only looking at high-dose risk extrapolation data in evaluating what the safe dose is for naturally occurring elements in the total diet. There is a great danger that risk of cancer may force increasing pressure for standards bodies to reduce the drinking water limits of naturally occurring elements still further, and with the technology available to achieve chemically clean water, there is a real risk of inducing deficiency diseases in the population. This may already be happening with arsenic, for which there is growing evidence of a role in the immune system. The risk is real, as conventional water treatment already automatically removes arsenic by complexation to iron or aluminium flocculants, thus providing a water which is guaranteed almost free of arsenic. While erring on the side of caution in cancer prevention, we may quite unknowingly encourage the appearance of chronic deficiency diseases where the diet is limited. It is thus vitally important to maintain a balanced view of what constitutes a safe water and to promote the importance of proper nutri-

tion in the total diet, both from the viewpoint of food as well as from water. Ideally arsenic and other potentially harmful substances should be monitored on a long-term basis so that these data can be linked to epidemiological studies to make water quality guidelines more appropriate to South African conditions in order to ensure that the health of the South African population is protected optimally on a more site-specific basis.

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