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Arsenic drinking water regulations in developing countries with extensive exposure

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Abstract

The United States Public Health Service set an interim standard of 50 μg/l in 1942, but as early as 1962 the US Public Health Service had identified 10 μg/l as a goal which later became the World Health Organization Guideline for drinking water in 1992. Epidemiological studies have shown that about one in 10 people drinking water containing 500 μg/l of arsenic over many years may die from internal cancers attributable to arsenic, with lung cancer being the surprising main contributor. A prudent public health response is to reduce the permissible drinking water arsenic concentrations. However, the appropriate regulatory response in those developing countries with large populations with much higher concentrations of arsenic in drinking water, often exceeding 100 μg/l, is more complex. Malnutrition may increase risks from arsenic. There is mounting evidence that smoking and arsenic act synergistically in causing lung cancer, and smoking raises issues of public health priorities in developing countries that face massive mortality from this product. Also, setting stringent drinking water standards will impede short term solutions such as shallow dugwells. Developing countries with large populations exposed to arsenic in water might reasonably be advised to keep their arsenic drinking water standards at 50 μg/l.

Keywords: Arsenic; Water; Regulations; Developing countries; Cancer

1. Introduction

During the last 10 years, the widespread nature of human exposure to arsenic in drinking water has become apparent in many countries. Potential health risks, in particular, cancer risks are very high (Chen et al., 1992; Smith et al., 1992; NRC, 1999, 2001). At the same time, regulating arsenic concentrations in drinking water has been a controversial and protracted process (Smith et al., 2002). Even when the concentration in drinking water is reduced to 10 μg/l as the World Health Organization recommendation (WHO, 1993), potential cancer risks remain high (NRC, 2001). With this in mind, consideration needs to be given to susceptible sub-populations within overall communities, and dietary and genetic susceptibility are topics warranting attention. Mounting evidence of synergy between smoking and arsenic raises issues of differential risk estimation for smokers and non-smokers. While these and other topics call for research investigation, a pressing issue in developing countries with widespread exposure is which arsenic drinking water regulation to adopt in the light of widespread exposure in rural populations

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with limited resources. In this paper, we will consider the magnitude of cancer risks from arsenic in drinking water, evidence concerning potential susceptible sub-populations, and consider their implications for drinking water standards in countries with widespread population exposures.

2. Cancer risks from arsenic in drinking water

Inorganic arsenic can cause multiple outcomes in different organ systems but the best documented effects relate to cancers: in particular, skin, lung, and bladder cancer. The International Agency for Research on Cancer has classified arsenic in drinking water as an established cause of each of these cancers (IARC, 1980, 2002). While arsenic can cause many different non-malignant effects, cancer risks are of particular concern at low dose since they originate from events in a single cell, rather than toxic effects on an organ system as is the case for neurological, cardiovascular, respiratory, and other non-malignant outcomes. In addition, the evidence from Chile indicates that in the longer term at least, cancer mortality predominates over all other causes of death. For example, in the period 1989 to 1993, while lung cancer and bladder cancer were still greatly increased, the relative risk estimates for all other causes of death combined (excluding lung cancer, bladder cancer, kidney cancer, liver cancer, and skin cancer) were 1.02 (95% CI, 0.99–1.05) for men and 1.00 for women (95% CI, 0.97–1.03) (Smith et al., 1998). At the same time, due to peak population, weighted exposure averages to 570 μg/l of arsenic in water spanning over 15 years, close to one in 10 of all deaths in men were attributable to arsenic in drinking water, and about one in 20 of deaths among women. It is therefore reasonable to focus on cancer when considering the long term risks resulting from arsenic in drinking water.

The shape of the dose–response curve below concentrations at which cancer risks are established is a matter of debate as is the case for all carcinogens. While it is accepted that animal cancer experiments can only assess high doses, there is a tendency to think that human studies should establish the shape of the dose–response curve, establish a threshold if there is one, and be able to detect risks at all doses at which they occur. The reality is much different. With lower and lower doses, accompanied by lower and lower risks, the ability of epidemiological studies to determine if the risks are actually increased diminishes. When risks are small, study size must be very large and exposures accurately assessed over many years. Eventually, dose–response curve fades into uncertainty as doses get lower and lower, confidence intervals broaden, and questions of confounding and potential bias in effect measures increase as relative risk estimates get closer to unity. Linear extrapolation from established risks in Taiwan and Chile down to 50 μg/l yields cancer mortality estimates of roughly one in 100 persons exposed (Smith et al., 1992; NRC, 1999). Yet there is probably no population in the world sufficiently large, and with sufficient numbers of people exposed to such concentrations for the necessary decades of constant exposure required to establish whether this estimate is valid or not. Thus, regulations of carcinogens in drinking water need to consider margins of safety, or consider simple linear extrapolation of risks downwards without waiting for proof.

3. Potential susceptible sub-populations

One issue of concern is that there may be some persons in exposed populations who are more susceptible to the disease effects than the others. This may suggest interventions to reduce their susceptibility, or that drinking water standards are set more stringently to protect them.

3.1. Nutrition

Malnutrition is widespread in West Bengal in India, Bangladesh, and parts of China where arsenic effects are widespread. Evidence also suggests that malnutrition was a factor in arsenic effects seen in Taiwan (Hsueh et al., 1995; Hsueh et al., 1997). Yet the largest population with relatively uniform and well documented high exposure to arsenic is in Northern Chile and is relatively well nourished (Smith et al., 2000), yet it has very high mortality from arsenic in water. Developed countries also have pockets of malnourishment, and one would hope that a goal in drinking water standards was to protect them too. Nevertheless, in poorer countries such as India and Bangladesh, a tragedy of arsenic in water is that it

exposes people who are obviously undernourished, and it has been postulated that this undernourishment increases the risks of arsenic effects. So far there have been few systematic studies of this issue, although in our investigations in West Bengal we have found approximately a doubling of the prevalence of skin lesions in those with the lowest intake of certain nutrients (Mitra et al., submitted). If we assume for the moment that this is correct and that severe malnourishment might double arsenic effects, then, we have a resource allocation problem. With ample resources, we would want to provide access to arsenic-safe water and provide good nourishment. With limited resources, one might note that providing arsenic-safe water will prevent future arsenic problems but improving nourishment without providing easy access to arsenic-safe water might only halve the risk in those who were previously the most poorly nourished.

3.2. Genetic

Potential susceptibility factors also include genetic susceptibility (Ahsan et al., 2003; Chung et al., 2002) and the susceptibility related to arsenic methylation (Chen et al., 2003a,b). These will not be discussed in detail here. With regard to genetic susceptibility, arsenic caused disease from drinking water is now manifested in many widely divergent populations in the world with more being discovered each year. Increased genetic susceptibility may occur within populations, and perhaps explain why some with similar exposure get disease, and others do not. Genetic factors might also relate to the differences in arsenic metabolism such as the tissue concentrations of the most toxic forms, in particular MMA3. Yet, as with nutritional susceptibility, the fact that some people may be more susceptible than others means one should have even greater caution with drinking water exposure and drinking water standards in order to protect the most susceptible.

3.3. Smoking

As some countries are bent on wiping out terrorism, at the same time, they are also vigorous in their defense of their right to export cigarettes to the developing countries even knowing that they will make addicts of their consumers and eventually kill close to one in two of them. Unfortunately, smokers themselves are also at increased risk from arsenic in water, and probably form a susceptible sub-population. The strongest evidence for this comes from a lung cancer study in Chile, in which the relative risk for those who both smoked and had high arsenic in their water was about 30 times that of non-smokers with low arsenic concentrations in their water (Ferreccio et al., 2000). In contrast, the lung cancer risk of smokers without arsenic in their water was about six times that of non-smokers. This, along with the vascular effects of both arsenic and smoking (Tseng et al., 1996; Engel et al., 1994; Chen et al., 1996, 1988; Hertz-Picciotto et al., 2000) might suggest that those who have been exposed to arsenic in water should be advised not to smoke. However, since smoking kills nearly one in two persons without arsenic in their water, it hardly seems pertinent to point out that smokers who have arsenic in their water have even greater risks.

3.4. Human susceptibility

To conclude this section, it might be noted that the greatest puzzle concerning arsenic toxicity is the susceptibility of humans to arsenic in contrast to rodents. The doses given to animals in the experimental cancer studies are often more than 100 times greater than that experienced by humans, with little response (NRC, 1999). Knowing something about the mechanisms associated with this difference in susceptibility between humans and rodents might also help determine why some people may be more susceptible to arsenic effects than the others. Be that as it may, the implication of variation to susceptibility which may occur in humans means that one would want even lower concentrations of arsenic in the drinking water than would be the case if risks were more uniform. Malnourished populations, for example, may need greater protection from arsenic than others, especially if their malnourishment cannot be easily solved.

4. Widespread arsenic exposure and the difficulty of community interventions

The problem confronted by developing countries with widespread exposure is fundamentally different from populations where only a small minority is

exposed. In Bangladesh (Smith et al., 2000), West Bengal (India) (Rahman et al., 2001), and parts of China (Guo et al., 2001), millions of people have arsenic concentrations in their drinking water above 50 μg/l, with some exceeding 1000 μg/l (Chakraborti et al., 2003). Widespread exposure raises the need for urgent short term solutions which can be accomplished with limited resources throughout a population, and which are sustainable until long term solutions such as piped water systems are eventually installed. However, even short term solutions have been difficult to implement and are only slowly adopted in countries with significant exposure. One problem is that, although something like inhaling cigarette smoke into your lungs seems as if it might harm you, the idea that crystal clear water with excellent taste can actually be damaging to your health is difficult to swallow. The technocrats (usually from the cities) sometimes act as if one visit to the villagers to tell them not to drink the contaminated water should be sufficient. If the technocrat ever returns, the villagers know what he/she wants to hear about what they are now drinking and have learned from long experience, it is best to keep technocrats from cities happy. They have also learned from experience that it is often wise not to believe what the latest technocrats are currently telling them. We are, therefore, confronted with situations in which some of the main costs of interventions involve on-going community education but which are usually not included in the cost estimates of supposedly cheap interventions, such as, for example installing arsenic removal filters.

5. Implications of drinking water standards for intervention strategies

There are major differences between a drinking water standard of 10 μg/l and one of 50 μg/l with regard to the intervention strategies. Removal of arsenic to a level of 50 μg/l is easier than to a concentration of 10 μg/l, although in both instances there are

Fig. 1. Newly installed shallow dugwell with hand pump extraction of water to replace tubewell with water arsenic concentration of 220 μg/l. Arsenic concentration in this dugwell was 6 μg/l, but concentrations in such dugwells may be in range from 2 to 50 μg/l.

short and long term disposal problems with arsenic removal which tend to be ignored or brushed aside. Deep tube wells have been installed in some locations but may exceed 10 μg/l of arsenic with some cross-contamination between aquifers and may pose problems of access. Finally, inexpensive traditional shallow dugwells (Fig. 1) provide low arsenic water (Smith et al., 2003), but which can be in a range of less than 10 μg/l up to 50 μg/l. This short term intervention which might be put in place in some areas, until long term water systems are eventually installed, would have to be ruled out with a drinking water standard of 10 μg/l. Reducing water concentrations from 50 to 10 μg/l might reduce long term cancer risk from one in 100 to one in 500, but would be self-defeating if an implication of this policy was that high exposures continued until the long term solutions achieving less than 10 μg/l are eventually put in place, perhaps decades from now.

It is concluded that setting drinking water standards in developing countries with widespread exposure to arsenic in drinking water needs to be done with considerable care. World Health Organization Guidelines currently advise a concentration of 10 μg/l. However, it might be advisable to make the Guideline 50 μg/l for developing countries with widespread population exposure currently above 50 μg/l.

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