IS THE PROPOSED NEW ARSENIC WATER STANDARD OF 10 UG/L SUFFICIENTLY PROTECTIVE OF PUBLIC HEALTH?

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On January 22, 2001, the U.S. Environmental Protection Agency announced in the Federal Register that: "Today EPA is establishing a health-based, non-enforceable Maximum Contaminant Level Goal (MCLG) for arsenic of zero and an enforceable Maximum Contaminant Level (MCL) for arsenic of 0.01 mg/L (10 μg/L)" (www.epa.gov/fedrgstr/EPA-WATER/2001/January/Day-22/w1668.htm).

To reach their decision about the MCL, the EPA adapted one of the results from a risk assessment (Morales et al., 2000), which was based on an epidemiological study of ecological design in an arsenic-exposed area of Taiwan, involving cancer deaths in the period 1973-86. Morales et al. used 12 different statistical models for data analysis. They estimated arsenic doses associated with a 1% increased risk of cancer deaths (ED01). For lung cancer, the water arsenic concentration associated with a 1% increased risk of lung cancer ranged from 11 to 364 ug/L for males, and from 8 to 396 ug/L for women, for the twelve models used. To establish the MCL, the EPA used results from the sub-linear model (stated by them to be linear because it had a linear term in the mathematical function used) that gave the lowest lung cancer risks of all 12 results presented. They then estimated cancer risks to be about 1 in 10,000 for arsenic water concentrations of 10 ug/L. This 1 in 10,000 risk estimate happens to fall in the range of acceptable risks, which for EPA is from one in ten thousand to one in one million.

Several other publications include low dose lung cancer risks estimated by extrapolation of risks found in highly exposed populations. Chen et al., 1992, calculated cancer potency indices based on a multistage model analysis of the same ecological village study from Taiwan used in the Morales publication. Potency estimates for lung cancer in this analysis were 1.2 per 100 for men and 1.3. per 100 for women, which corresponded approximately to consumption of drinking water containing 200 ug/L of arsenic. Concurrent with this work, we published the findings of our risk assessment based on linear extrapolation of relative risks from the same Taiwan data (Smith et al., 1992). We estimated that lung cancer risks for consumption of 1 liter/day of water containing 50 ug/L of arsenic could be about 2.3 per 1000 for men and 5.1 per 1000 for women (Smith, et al., 1992). Both these studies yielded risk within the range of those calculated by Morales et al., but considerably higher than the risk estimates chosen by the EPA. The NRC committee concluded that the combined cancer risks, including bladder and lung cancer, could be of the order of 1 in 100 for consumption of water containing arsenic at 50 ug/L (NRC, 1999), an estimate which also suggests risks considerably higher than reported by EPA.

The above estimates are all based on one ecological study in Taiwan. However risk extrapolations can, and should, include results from studies in other exposed populations.

The following studies include lung cancer results: an ecological study in Chile (Smith et al., 1998); an ecological study in Argentina (Hopenhayn-Rich et al., 1999); a small cohort study from Japan (Tsuda et al., 1989); and a case-control study with individual exposure data (in contrast to ecological exposure data) from Chile (Ferreccio et al, 2000). Taken together, the results of these studies show a linear dose-response relationship for lung cancer risks with arsenic concentrations in drinking water. Simple linear regression (weighting makes little difference) gives a lung cancer relative risk estimate of about 1.07 for 10 ug/L of arsenic in water. With background lung cancer mortality of the order of 5% of all deaths in the U.S., this would translate to an increased lung cancer mortality risk from lifetime constant daily exposure to the arsenic MCL of the order of 3-4 per 1000 exposed persons. Without taking into account risks for other arsenic-caused cancers, this suggests that the EPA may have underestimated cancer risks from arsenic in drinking water by at least a factor of 10.

Low dose effects of arsenic also receive support from genotoxic effects on human bladder cells with low population exposure (Moore et al, 1997), and DNA hypermethylation when human lung cell cultures are exposed to low levels of arsenite commensurate with urinary concentrations in populations with quite low exposure to arsenic (Mass and Wang, 1997). On the other hand, it is possible that the risk of lung cancer due to arsenic in drinking water is largely confined to cigarette smokers. This possibility is raised by evidence of synergy between arsenic and smoking in Chile (Ferreccio et al., 2000). Furthermore, the presence of inorganic arsenic in food means that there are diminishing returns from reducing arsenic concentrations in drinking water below 10 ug/L.

And does diet protect against arsenic health effects so that it is only a problem in developing countries such as India (Guha Mazumder et al., 1998) and Bangladesh (Smith et al., 2000)? The arsenic-exposed populations in Argentina and Chile are quite well-nourished, yet there is no evidence that their risks are lower than those in Taiwan, where it has been postulated that low selenium and poor diet may have contributed to cancer risks. The highest population cancer risks from any environmental carcinogen have been reported for the arsenic-exposed region of Chile where about 7% of all adult deaths are attributable to arsenic in drinking water (Smith et al., 1998).

The possibility that cancer risks are much higher than predicted by the EPA in the promulgation of the new drinking water standard means that priority should be given to undertaking further research in populations with medium to low exposures. While it is impossibility to prove if risks of the order of 1 in 1000 are present at 10 ug/L, studies with additional data in the lower exposure range including 50-100 ug/L could be of considerable value. If well-designed studies with good statistical power fail to find increased cancer risks at these concentrations, we can have some assurance that the margin of safety associated with 10 ug/L is sufficient. On the other hand, if increased risks commensurate with linear extrapolation are found, then the drinking water standard should be re-examined in the light of such findings.

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