SIGNIFICANCE OF ARSENIC IN DRINKING WATER NEAR HIDDEN LANE LANDFILL

STERLING, VIRGINIA

Letter Health Consultation

February 23, 2017

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Dear Dr. Goodfriend:

In response to concerns about Hidden Lane Landfill contaminating drinking wells, the Virginia Department of Health prepared a Letter Health Consultation on November 23, 2016. This Letter Health Consultation responds to your follow-up questions regarding the levels of arsenic found in the water and whether residents should be advised to find alternate sources of drinking water.

BACKGROUND

The U.S. Environmental Protection Agency (EPA) maximum contaminant level (MCL) for arsenic is 10 µg/L. There has been considerable controversy over this limit, with some arguing that this limit is protective of health, while others suggest that it should be lower. This controversy is seen in the EPA drinking water unit risk (5×10^{-5} per µg/L) for arsenic, which suggests that the MCL of 10 µg/L has an associated 5/10,000 increased cancer risk. Therefore, in a group of 10,000 people drinking water containing 10 µg/L arsenic over 70 years, the EPA expects that five people would develop cancer due to arsenic exposure. The EPA considers this a high increased risk for cancer.

The average water arsenic concentration measured in wells near Hidden Lane Landfill was approximately 2 µg/L. This is lower than the EPA MCL of 10 µg/L, but still has an associated risk of cancer of 1/10,000, which the EPA considers high risk. Most drinking water systems in the U.S. have an arsenic concentration of 1–2 µg/L. While MCLs are based upon cost concerns in addition to health consequences, the 1 in 10,000 increased cancer risk for 2 µg/L arsenic seemed to be unusually high to be tolerated over so much of the US.

DISCUSSION

A 1989 study by Wu et al. is the foundation for our knowledge of arsenic cancer risk.[1] This study was carried out in Taiwan, where artesian wells often contain extremely high concentrations of arsenic. In the United States, most groundwater has a concentration of 1–2 µg/L. By contrast, in Wu's study the low exposure well water had <150 µg/L arsenic, while the high exposure well water had over 600 µg/L.[1] The scientific consensus is that water containing ≥500 µg/L arsenic poses a significant risk to health, primarily from skin, bladder, and lung cancers. The cancer mortality risk from drinking water with arsenic concentrations this high is 1 in 10 people. However, early estimates of the cancer risks of arsenic in drinking water at lower concentrations may be overestimating the risks due to several factors presented below.

Confounding Factors

Difference in baseline health status between Taiwanese and U.S. populations

The population studied in Taiwan may have been at increased risk for arsenic toxicity due to poor nutrition. A study of skin cancer and nutritional status found risk factors in addition to arsenic exposure were undernourishment and liver dysfunction secondary to hepatitis B infection.[2] One analysis found the increased risk due to malnutrition so high the authors recommended addressing the problem of nutrition prior to trying to reduce water arsenic levels below 50 µg/L and recommended the WHO scale back their target standard of 10 µg/L in developing nations.[3]

Nonlinear correlation of cancer risk at water arsenic levels <150 µg/L

The cancer risks for the Taiwanese population at the low water arsenic level do not appear to correlate linearly with water arsenic exposure and are likely confounded by other factors not accounted for in the original analysis.[4,5,6] This means an extrapolation from this data to low water arsenic is probably overestimating the cancer risk. Lamm et al. suggested a threshold model for arsenic exposure and bladder cancer mortality with bladder cancer risk not increasing at water arsenic concentrations of <100–200 µg/L.[4,5]

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[1] Wu MM, Kuo TL, Hwang YH, Chen CJ. Dose-response relation between arsenic concentration in well water and mortality from cancers and vascular diseases. Am J Epidemiol. 1989 Dec;130(6):1123-32.
[2] Hsueh YM, Cheng GS, Wu MM, Yu HS, Kuo TL, Chen CJ. Multiple risk factors associated with arsenic-induced skin cancer: effects of chronic liver disease and malnutritional status. Br J Cancer. 1995 Jan;71(1):109-14.
[3] Smith AH, Smith MM. Arsenic drinking water regulations in developing countries with extensive exposure. Toxicology. 2004 May 20;198(1-3):39-44.
[4] Lamm SH, Engel A, Penn CA, Chen R, Feinleib M. Arsenic cancer risk confounder in southwest Taiwan data set. Environ Health Perspect. 2006 Jul;114(7):1077-82.
[5] Lamm SH, Robbins S, Chen R, Lu J, Goodrich B, Feinleib M. Discontinuity in the cancer slope factor as it passes from high to low exposure levels—arsenic in the BFD-endemic area. Toxicology. 2014 Dec 4;326:25-35. doi: 10.1016/j.tox.2014.08.014. Epub 2014 Sep 18.
[6] Lamm SH, Robbins SA, Zhou C, Lu J, Chen R, Feinleib M. Bladder/lung cancer mortality in Blackfoot-disease (BFD)-endemic area villages with low (<150 µg/L) well water arsenic levels—an exploration of the dose-response Poisson analysis. Regul Toxicol Pharmacol. 2013 Feb;65(1):147-56. doi: 10.1016/j.yrtph.2012.10.012. Epub 2012 Nov 5.

Mechanism of carcinogenesis not consistent with linear extrapolation of risk

Historically there has been controversy regarding the dose-response curve appropriate for carcinogens, but researchers from the mid-1970's have tended towards a linear dose-response curve based upon single-hit kinetics.[7] While the existence of DNA repair mechanisms was known at the time, it was thought that these processes would generally not appreciably affect the linearity of the curve.

By the time the EPA was making their determination regarding the MCL of arsenic, it was known that arsenic did not act through simple formation of DNA adducts, but through several other mechanisms,[8] and from population data that the dose-response curve was not strictly linear. In their analysis of cancer risk, the EPA used a multi-stage model with time and used both linear and quadratic fitting. [9] The EPA cancer risk estimation for arsenic in drinking water is nonlinear at higher concentrations, but at concentrations <150 µg/L is principally linear. The EPA recognized this was an approximation but was limited by available data:

> Eastern Research Group, under contract to EPA, convened an Expert Panel on Arsenic Carcinogenicity on May 21 and 22, 1997 (Eastern Research Group, 1997). The Expert Panel believed that, "it is clear from epidemiological studies that arsenic is a human carcinogen via the oral and inhalation routes (p. 20)." They also concluded, "that one important mode of action is unlikely to be operative for arsenic". The panel agreed that arsenic and its metabolites do not appear to directly interact with DNA (pp. 30-31)." In addition, the panel agreed that, "for each of the modes of action regarded as plausible, the dose-response would either show a threshold or would be nonlinear (p. 31)". The panel agreed, however, "that the dose-response for arsenic at low doses would likely be truly nonlinear, i.e., with a decreasing slope as the dose decreased. However, at very low doses such a curve might be linear but with a very shallow slope, probably indistinguishable from a threshold (p. 31)."[9]

While the risk analysis done anticipated nonlinear risk and a likely dosage threshold, the unit risk projection extracted from the risk analysis is by definition linear. This linear extrapolation is likely overly cautious at low arsenic concentrations. A better way to evaluate risk is to look to recent research on low-level arsenic exposure in drinking water.

Cancer risks of low-concentration (<100 µg/L) arsenic in U.S. drinking water

A 1995 study of Utah residents with a mean water arsenic concentration of 5 µg/L found no association between bladder cancer and cumulative exposure to arsenic. A 2005 study of populations in California and Nevada with drinking water with nearly 100 µg/L arsenic showed

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[7] Zeise L, Wilson R, Crouch EA. Dose-response relationships for carcinogens: a review. Environ Health Perspect. 1987 Aug; 73: 259–306.

[8] Rossman TG. Mechanism of arsenic carcinogenesis: an integrated approach. Mutat Res. 2003 Dec 10;533(1-2):37-65.

[9] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS) Chemical Assessment Summary: Arsenic, inorganic; CASRN 7440-38-2. Feb 10, 1988. https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0278_summary.pdf, accessed December 19, 2016.

similar results, with no clear association between arsenic in drinking water and bladder cancer.[10] There did appear to be some association between smoking and increased risk in bladder cancer at 200 µg/day, but it would be impossible to reach this daily dosage by drinking water that met the EPA MCL for arsenic. In 2008, another ecologic study found no association between bladder cancer and arsenic at concentrations near the MCL.[11] Most recently, in 2010, a Michigan population study found no increased risk in bladder cancer for smokers and nonsmokers with water concentrations >10–100 µg/L by comparison to <1 µg/L.[12]

At high concentrations, arsenic in drinking water is a risk factor for lung and skin cancers. There are no adequate studies to determine cancer risk at low arsenic water concentration. The skin cancers caused by arsenic tend to be squamous and basal cell carcinomas, which are not generally fatal, therefore, they are not included in cancer mortality studies. No direct studies of low concentration of arsenic in drinking water and cancer risk have been done, but studies of U.S. populations have shown that individuals with the highest concentration of arsenic in toenails have an increased risk of basal and squamous cell cancer[13] and small-cell and squamous cell lung cancer.[14] It is difficult to interpret these studies since we do not know the source of the arsenic exposure, which could be from drinking water, food, or occupational exposure.

A 1999 cohort mortality study in Utah provides direction for future research, but is unlikely to be illuminating of risks for water arsenic concentrations below the EPA MCL given the observed arsenic concentrations spanned 3.5–620 µg/L.[15] The authors defined a "low" exposure category of <1,000 µg/L-years. For a water arsenic concentration of 10 µg/L, a person would reach this limit after 100 years. No increased risk for bladder or lung cancer were detected for this exposure group, however, women in this exposure group had a large increased risk for death due to melanoma.[15] This is an unexpected finding since arsenic has not been implicated as a risk factor for melanoma and is probably a spurious correlation. Supporting this conclusion, no correlation was found between the medium or high exposure groups and death due to melanoma, in either men or women.[15]

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[10] Steinmaus C, Yuan Y, Bates MN, Smith AH. Case-control study of bladder cancer and drinking water arsenic in the western United States. Am J Epidemiol. 2003 Dec 15;158(12):1193-201.

[11] Han YY, Weissfeld JL, Davis DL, Talbot EO. Arsenic levels in ground water and cancer incidence in Idaho: an ecologic study. Int Arch Occup Environ Health. 2009;82: 843-849. doi:10.1007/s00420-008-0362-9

[12] Meliker JR, Slotnick MJ, AvRuskin GA, Schottenfeld D, Jacquez GM, Wilson ML, Goovaerts P, Franzblau A, Nriagu JO. Lifetime exposure to arsenic in drinking water and bladder cancer: a population-based case-control study in Michigan, USA. Cancer Causes Control. 2010 May;21(5):745-57. doi: 10.1007/s10552-010-9503-z. Epub 2010 Jan 19.

[13] Karagas MR, Stukel TA, Morris JS, Tosteson TD, Weiss JE, Spencer SK, Greenberg ER. Skin cancer risk in relation to toenail arsenic concentrations in a US population-based case-control study. Am J Epidemiol. 2001 Mar 15;153(6):559-65.

[14] Heck JE, Andrew AS, Onega T, Rigas JR, Jackson BP, Karagas MR, Duell EJ. Lung cancer in a U.S. population with low to moderate arsenic exposure. Environ Health Perspect. 2009 Nov;117(11):1718-23. doi: 10.1289/ehp.0900566. Epub 2009 Jul 2.

[15] Lewis DR, Southwick JW, Ouellet-Hellstrom R, Rench J, Calderon RL. Drinking water arsenic in Utah: A cohort mortality study. Environ Health Perspect. 1999 May;107(5):359-65.

Non-cancer risks of low-concentration (<10 µg/L) arsenic in drinking water

Effects on adults

A study in Michigan found there might be an association between increasing arsenic water concentrations and stroke hospitalization.[16] The median arsenic concentration was 7.78 µg/L, which is low enough to be more directly comparable to the concentrations typically encountered. However, the risk found was very small (an increased relative risk of 1.03 (95% confidence interval 1.01–1.05) per 1 µg/L increase in arsenic concentration). Since this study tracked hospitalization incidents rather than individuals and used county and zip code data to estimate arsenic intake, a follow-up study utilizing individual data is needed to confirm the association.

A 2012 study of a Texas population with mean water arsenic concentrations of 6.2 µg/L showed a possible weak association between coronary heart disease and hypertension and water arsenic concentrations.[17]

The Utah drinking water cohort mortality study mentioned above found that the low exposure category had approximately doubled incidences of mortality due to hypertensive coronary disease and nephritis or nephrosis. [15]

Effects on children

The major concern for children exposed to arsenic is the effects on their neurological development. One study on U.S. children drinking well water with elevated concentrations of arsenic found that an increase in water arsenic concentration from <5 µg/L to ≥5 µg/L resulted in a 5–6 point drop in IQ.[18] However, there was no correlation between toenail arsenic concentrations and IQ in these children.[18] They did not observe a correlation between arsenic concentrations and IQ below 5 µg/L and suggested there may be a threshold for neurological harm.[18]

Future evaluations may result in a lower MCL

The EPA is re-evaluating the evidence for risks of arsenic exposure and may further reduce the MCL in the future. Evidence suggestive of some harm at arsenic concentrations near the current MCL in increased cancer risk for smokers, cardiovascular risks, and risk of neurological harm to children could justify reducing the MCL to 5 µg/L to increase the safety margin.

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[16] Lisabeth LD, Ahn HJ, Chen JJ, Sealy-Jefferson S, Burke JF, Meliker JR. Arsenic in drinking water and stroke hospitalizations in Michigan. Stroke. 2010 Nov;41(11):2499-504. doi: 10.1161/STROKEAHA.110.585281.
[17] Gong G, O'Bryant SE. Low-level arsenic exposure, AS3MT gene polymorphism and cardiovascular diseases in rural Texas counties. Environ Res. 2012 Feb;113:52-7. doi: 10.1016/j.envres.2012.01.003. Epub 2012 Feb 15.
[18] Wasserman GA, Liu X, Loiacono NJ, Kline J, Factor-Litvak P, van Geen A, Mey JL, Levy D, Abramson R, Schwartz A, Graziano JH. A cross-sectional study of well water arsenic and child IQ in Maine schoolchildren. Environ Health. 2014 Apr 1;13(1):23. doi: 10.1186/1476-069X-13-23.

CONCLUSIONS

VDH used accepted methods in its Letter Health Consultation dated November 23, 2016 for calculating cancer risk for arsenic concentrations in well water. However, the linear unit risk determined at moderate arsenic concentrations overestimates risk when extrapolated to very low arsenic concentrations. Also, in evaluating populations in the U.S. exposed to arsenic concentrations in drinking water less than the EPA MCL of 10 µg/L it appears that the MCL is generally protective against cancer and non-cancer health risks.

RECOMMENDATIONS

There are no recommendations at this time. VDH will revisit this issue in future in light of new research or in response to a decision by the EPA on the new MCL.

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