

March 26, 2010

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Re: Comments to the US33 Science Advisory Board on IRIS Toxicology of Inorganic Arsenic (Cancer) (External Review Draft) (75 Federal Register 7477); Docket ID No. EPA-HQ-ORD-2010-0123

Dear Dr. Shallal-Al-Mudullal:

The Albuquerque Bernalillo County Water Utility Authority (ABCWUA) appreciates the opportunity to submit comments on the Integrated Risk Information System (IRIS) Toxicology of Inorganic Arsenic (Cancer) (External Review Draft) (75 Federal Register 7477).

Arsenic is ubiquitous at low levels in the nation's soils and natural waters. For this reason, the hazard assessment has to be sound, particularly at potentially low concentrations in drinking water. As USEPA moves from hazard assessment to exposure assessment and risk management under the Safe Drinking Water Act (SDWA), Superfund and other statutes, the role of uncertainty and how uncertainty is addressed in the hazard assessment amplifies both the projected benefits and costs of potential risk management options. The SAB's careful review of the External Review Draft is greatly appreciated.

In 2003, USEPA undertook revision of the IRIS toxicology document for inorganic arsenic. As a part of that revision process, USEPA requested a review by the USEPA's Science Advisory Board (SAB). During 2005 – 2007 a review was completed and SAB recommendations were published in 2007. The review did not focus on the entire IRIS document for inorganic arsenic, but rather on specific charge questions. USEPA subsequently revised the document that will be under a second SAB review in 2010. For the second SAB review, the entire document will again not be reviewed, but will focus on specific USEPA charge questions in response to the 2007 SAB review.

The SAB has been provided specific charges, including the request that the SAB evaluate USEPA's treatment of available epidemiology data. We have reviewed the IRIS document and are concerned that USEPA continues to rely on the controversial Taiwanese data. It is our continued concern that USEPA may have overlooked

additional studies on arsenic which demonstrate a much lower health risk due to arsenic in drinking water

Although the USEPA dismisses the Lamm et al. (2006) study, this study clearly calls attention to the weakness of the Taiwanese data. Lamm et al. performed a quantitative analysis for the risk of human cancer from the ingestion of inorganic arsenic that was based on the reported cancer mortality experience in the blackfoot disease (BFD)–endemic area of southwest Taiwan. Linear regression analysis shows that arsenic as the sole etiologic factor accounts for only 21% of the variance in the village standardized mortality ratios for bladder and lung cancer. A previous study had reported the influence of confounders (township, BFD prevalence, and artesian well dependency) qualitatively, but they have not been introduced into a quantitative assessment. In this six-township study, only three townships (2, 4, and 6) showed a significant positive dose–response relationship with arsenic exposure. The other three townships (0, 3, and 5) demonstrated significant bladder and lung cancer risks that were independent of arsenic exposure. The data for bladder and lung cancer mortality for townships 2, 4, and 6 fit an inverse linear regression model ($p < 0.001$) with an estimated threshold at 151 micrograms per liter ($\mu\text{g/L}$ (95% confidence interval, 42 to 229 $\mu\text{g/L}$). Such a model is consistent with epidemiologic and toxicologic literature for bladder cancer. Exploration of the southwest Taiwan cancer mortality data set has clarified the dose–response relationship with arsenic exposure by USEPA ruling out township as a confounding factor.

Further, Lamm et al. (2004) analyzes the relationship between arsenic exposure through drinking water and bladder cancer mortality in the U.S. The county-specific white male bladder cancer mortality data (1950-1979) and county-specific groundwater arsenic concentration data were obtained for 133 U.S. counties known to be exclusively dependent on groundwater for their public drinking water supply. No arsenic-related increase in bladder cancer mortality was found over the exposure range of 3 to 60 $\mu\text{g/L}$ using stratified analysis and regression analyses (both unweighted and weighted by county population and using both mean and median arsenic concentrations). These results, which provide a direct estimate of arsenic-related cancer risk for U.S. residents, exclude the National Research Council's 2001 risk estimate that was based on Southwest Taiwan data and required adjusting for differences between the body mass and water consumption rates of U.S. and Taiwanese residents. Clearly this study again points to the weakness of the Taiwanese data and studies.

In the U.S. arsenic epidemiological studies have not corroborated the Taiwanese data. The USEPA's own study in Fallon, NV did not find any excess cancers for this population of around 7,600 people. In Fallon, the arsenic in the drinking water supply was over 100 $\mu\text{g/L}$. If the risk of cancer were truly as high as the USEPA evaluation of the Taiwanese data suggests, then excess cancers would certainly be found in Fallon with an arsenic level of 100 $\mu\text{g/L}$. This USEPA study and data from Fallon NV appears to have been overlooked in the IRIS document.

Omitted from the IRIS document is the epidemiological study by Buchet and Lison (1998) who report: "The dose-response relationship for lung carcinoma and other cancers at low doses of arsenic is highly uncertain because it is based on modeling data collected in populations with a high daily intake of the element." They studied causes of mortality in the Belgian general population finding that a moderately increased absorption of

arsenic, leading to a 3- to 4- fold higher urinary excretion (35 µg/day as compared with 6-10 µg/day in nonexposed subjects) did not increase the mortality from diseases of the nervous system, liver and heart, and cancers, including lung and bladder cancer. Buchet and Lison concluded that a low to moderate level of environmental exposure to inorganic arsenic (0.3 µg/m³ of air; 20-50 µg/L of drinking water) did not seem to affect the causes of mortality and suggested a nonlinearity of the dose-response relationship for arsenic and cancer. They also felt that smoking may act in synergy with arsenic in the induction of lung cancer.

Evidence from epidemiological studies in the U.S. and Europe suggests that the risk of bladder cancer, lung cancer and cardiovascular disease are not consistent with the Taiwanese data. Additional studies should be conducted in the ET AL. to obtain more information about cancer risks in U.S. populations that have been exposed to low to moderate levels of arsenic in drinking water (less than several hundred micrograms per liter for 20 to 40 years). Incidence studies, in addition to mortality studies, should be considered where such information is available. If the combined cancer risk of waterborne arsenic exposure in the U.S. is "on the order of 1 in 100" as estimated by the Subcommittee on Arsenic in Drinking Water, National Research Council (NRC, 1999, page 8), and a higher risk shown in the IRIS document, epidemiological studies in the U.S. should be able to detect an increased cancer risk. The Subcommittee (NRC, 1999, page 3) also recommended additional epidemiological studies: "Additional epidemiological evaluations are needed to characterize the dose-response relationship for arsenic-associated cancer and noncancer endpoints, especially at low doses. Such studies are of critical importance for improving the scientific validity of risk assessment." These studies have not been done by the USEPA.

The NRC also discussed a number of other issues that affect the extrapolation of the Taiwanese data to the U.S. population, including: limitations of the use of ecological data; the effects of measurement error from use of ecological data; and the impact of the choice of models to assess the dose-response relationship (NRC, 1999). NRC specifically warns about the inadequate information available to assess exposures in Taiwan: "It is important to keep in mind that the considerable variability in the arsenic concentrations detected in multiple wells within some of the villages leads to considerable uncertainty about exposure concentrations in the Taiwanese data" (NRC, 1999, page 294). We are concerned that the possible misclassification of drinking water arsenic exposures has biased the relationship (i.e., the observed risks are higher than the actual risks in the Taiwanese study). If so, health benefits associated with lower arsenic levels may be overestimated. The NRC strongly recommended that further research be conducted to better characterize the exposure-response relationship at low exposures to arsenic in drinking water.

Morales et al. (2000) published a risk assessment for cancers of the bladder and lung for arsenic in drinking water, based on data from 42 villages in the Taiwan epidemiological study. They showed that arsenic exposure-response assessments using the Taiwan data depend highly on the choice of the mathematical model and whether or not a comparison population is used in the analysis. Since the Taiwan study was ecological in design, individual drinking water arsenic exposures were not available, and Morales et al. conducted their risk assessment assuming that all persons in each village were exposed to the same arsenic concentration. Arsenic levels in the village wells were measured in

1964-66, and all wells are now closed. Morales et al. acknowledged that individual waterborne arsenic exposures can vary widely in a village and that in their risk assessment they could not account for dietary intake of inorganic arsenic in food, poor nutritional status and other possible confounders. If they are not considered, all of these factors can affect the results of the risk assessment. Morales et al. found that risk estimates were particularly affected by whether or not a comparison population was used and different risk estimates were obtained for the two comparison Taiwanese populations in the analysis. These findings illustrate the magnitude of the uncertainty associated with the USEPA's risk estimates. The effects of additional uncertainties (e.g., dietary intake of inorganic arsenic in food, poor nutritional status and other possible confounders) could further increase the magnitude of the variability in the risk estimates. The findings of Morales et al. also show the importance of conducting epidemiological studies in the U.S. to determine whether the theoretical risks at low to moderate exposures estimated from the Taiwan study can be confirmed in the U.S. population. If the cancer risks are as high as estimated, they should be able to be detected in an epidemiological study of a U.S. population.

If waterborne arsenic is the cause of increased bladder cancer risk as observed in the Taiwan study (Guo et al., 1997), one would expect that persons who developed bladder cancer had high exposures because they consumed water with higher arsenic levels than the median exposure for their village or that they consumed more water. Persons with bladder cancer may also have consumed much higher levels of arsenic than were measured in any well in a village, since wells with the highest arsenic levels may have been closed and not in use at the time of the water sampling. Although these wells would be unavailable for testing, study participants may have used them prior to the study. These factors may have caused researchers to underestimate the actual arsenic exposure of Taiwan cancer cases (Brown et al., 1997a). An underestimate of arsenic exposures would overestimate the dose-response relationship for arsenic and cancer, resulting in an overestimate of the risk of cancer from low drinking water exposures in the U.S.. A similar exposure-assessment problem was noted in studies of arsenic-related bladder cancer in South America (Brown and Beck, 1996).

Another area of uncertainty is whether the results from a study of arsenic-related health effects in Taiwan can be extrapolated to U.S. populations. In the Taiwanese populations studied, arsenic exposures from sources other than drinking water, such as food, were much higher than in the U.S. This would also result in overestimating the cancer risk from waterborne arsenic in U.S. populations. The USEPA did not address how these results that suggested a synergistic effect might affect the exposure-response curve.

In addition, socioeconomic factors may have influenced the relationship between arsenic and cancer. Income levels of residents in the area of Taiwan with high waterborne arsenic levels were very low. Other diseases and dietary deficiencies also may have influenced the relationship between arsenic exposure and cancer. For example, the diets for Taiwanese living in this area have very low levels of selenium; deficiencies in selenium may increase the toxicity of arsenic. Also, Hepatitis B infections, common in Taiwan and much of Asia, appear to increase the risk of arsenic-caused skin cancer and may have also influenced the risk of arsenic-related internal organ cancers.

The problems in extrapolating the Taiwan findings to the U.S. are difficult based on the concerns mentioned above. However, in 1999 a cohort study was completed in Utah that reported findings inconsistent with the Taiwan study results (Lewis et al., 1999). This study included 4,045 people living in Millard County, Utah, who were exposed to drinking water arsenic at levels of 14 to 166 $\mu\text{g}/\text{L}$. The cohort was assembled from Church of the Latter Day Saints records and participants were followed to determine if they had died. If so, the cause of death was identified. Efforts were made to determine the waterborne arsenic exposures (arsenic level and number of years the person drank the water) for each cohort member.

Of these people, 2,203 had died at the time of the study. Findings were reported as SMRs where the number of deaths in the Millard County cohort were compared to the number expected based on mortality rates for the entire state of Utah. An SMR of greater than 1.00 indicates that more deaths were observed than were expected, suggesting an increased risk. An SMR less than 1.00 indicates that the number of deaths observed was less than the number expected. An SMR of 1.00 indicates the number of deaths observed were equal to those expected (i.e., no increased risk). Confidence intervals provide a measure of SMR stability; if 1 is included within the confidence interval, the SMR is indistinguishable from 1.00 (not statistically significant).

Lewis et al. observed no association between arsenic exposure in drinking water and mortality due to bladder, lung, liver, or kidney cancer in the Utah cohort. No increased risk of death was found for bladder cancer. The SMR for bladder cancer mortality among cohort members with the highest arsenic exposure levels was approximately 1.00. It was also indistinguishable from 1.00 for the lowest exposure level.

The respiratory cancer SMRs for Utah cohort males and females, rather than being elevated, were statistically significantly less than 1.00 (e.g., cohort members had a lower risk of respiratory cancer than expected). For males in the highest drinking water arsenic exposure category, the SMR for lung cancer was 0.44 and for females in the highest drinking water arsenic exposure category, it was 0.22. These SMRs were based on an expected mortality rate from lung cancer in Utah and, therefore, reflects the lower smoking prevalence of residents of that state.

These findings raise questions about whether the risks of arsenic-related bladder and lung cancer found in Taiwan can be extrapolated to U.S. populations. Based on the Taiwanese data, the USEPA predicts that arsenic may raise the lifetime risk of bladder and lung cancer by 1 in 100 for each 50 $\mu\text{g}/\text{L}$ of arsenic in the drinking water. If these risks are applicable to U.S. populations, a waterborne arsenic exposure of greater than 150 $\mu\text{g}/\text{L}$, which was experienced by some of the Utah study participants, should have tripled the risk of bladder cancer for females in the highest waterborne arsenic exposure category (an SMR of 3.0) and doubled it for males in the highest waterborne arsenic exposure category (an SMR of 2.0).

Bates et al. (1995) also studied a Utah population. Data from Utah respondents to the National Bladder Cancer Study conducted in 1978 were used to evaluate associations in a U.S. population exposed to measurable, but low levels of drinking water arsenic. This analytical epidemiological study of 117 bladder cancer cases and 266 population-based controls was conducted in areas where 92 percent of towns had arsenic levels near the

proposed MCL (<10 µg/L); one town had >50 µg/L). Subjects were interviewed and individual exposures to arsenic in drinking water were estimated by linking residential history information with water sampling information.

Two indices of cumulative arsenic exposure were used, total cumulative exposure and intake concentration. Exposures were in the range 0.5 to 160 µg/L (mean, 5.0 µg/L). There was no overall increase in bladder cancer risk with increasing exposure to arsenic in drinking water considering either cumulative dose or intake concentration. However, Bates et al. reported that among “cigarette smokers there was a nonsignificant elevation in risk that was not dose related.” Among smokers only, positive trends in risk were found for exposures estimated for decade-long time periods, especially in the 30- to 39-year period prior to diagnosis. The following presents information about the magnitude of relative risks, none of which were statistically significant, among all participants using cumulative dose of arsenic from water. In a case-control study, the odds ratio (OR) is interpreted as a relative risk; that is, the cancer risk for exposed persons is relative to risk for persons who are unexposed or have low exposure (the baseline or OR=1). An OR=1 is interpreted as no increased risk; an OR>1.00 suggests an increased relative risk, and an OR<1.00 suggests a decreased relative risk. Confidence intervals provide a measure of OR stability; if 1 is included within the confidence interval, the OR is indistinguishable from 1.00 (not statistically significant).

As shown in Table 1, persons in the study that had cumulative waterborne arsenic exposures of less than 19 mg were considered as the baseline; persons with exposures of 19 less than or equal to 33 mg had 56 percent higher risks than those with exposures of less than 19 mg. However, persons with exposures of 33—<53 mg had 5 percent less risk and those with the highest exposure had 41 percent higher risks. Relative risks did not increase with increased exposure, and none were statistically significant.

TABLE 1
Bates et al. Utah Study

Cumulative Dose of Arsenic	Bladder Cancer Risk OR (95% CI) ^a
< 19 mg	1
19 – <33 mg	1.56 (0.8, 3.2)
33 – <53 mg	0.95 (0.4-2.0)
>53 mg	1.41 (0.7-2.9)

^a No statistically significant associations were observed. Risks were adjusted for gender, age, smoking, and other possible confounders.

The USEPA should evaluate how the Bates et al. results compare with estimated exposures in other epidemiological studies. Bates et al. (1995) noted that previous U.S. studies of arsenic have involved small communities and none reported elevated health risks. Bates et al. also noted that “...there is a striking difference in risk estimates found between this study (Utah) and the Taiwanese investigations.... Arsenic in artesian water from the Taiwan study area ranged from 0.35-1.14 mg/L with a mean of 0.78 mg/L....In view of the contrast between our findings and those from Taiwan, it is useful to consider possible noncausal reasons for our apparent associations” of increased bladder cancer

among smokers with 30 to 39 years of exposure to arsenic; "chance may have played a role."

Bates et al. also note that "the discrepancy of our bladder cancer risk estimates with those from Taiwan and patients treated with Fowler's solution raises the possibility of bias in these data that can only be resolved by further carefully conducted studies in exposed populations." We agree that additional epidemiological studies of an analytical design are needed in populations exposed to arsenic at levels lower than found in Taiwan, Chile, and Argentina. These studies should be conducted in the U.S.

Another epidemiological study (Kurttio et al., 1999) considered low drinking water arsenic exposures. Like the Bates et al. study in Utah, this is a case-control study, and the USEPA should note the strengths of these studies, which are more informative than ecological and SMR studies. Individual exposures and confounding factors are considered for cases and controls.

Kurttio et al. assessed the levels of arsenic in drilled wells in Finland and studied the association of arsenic exposure with the risk of bladder and kidney cancers. Study participants were selected from a register-based cohort of all Finns who had lived at an address outside the municipal drinking-water system during 1967-1980 (n = 144,627). The final study population consisted of 61 bladder cancer cases and 49 kidney cancer cases diagnosed between 1981 and 1995, as well as an age-and-sex-balanced random sample of 275 subjects, which were the reference cohort.

Water samples were obtained from the wells used by the study population at least during 1967-1980. The total arsenic concentrations in the wells of the reference cohort were low (reported median = 0.1 µg/L; maximum = 64 µg/L, and 1 percent exceeded 10 µg/L). Arsenic exposure was estimated as arsenic concentration in the well, daily dose, and cumulative dose of arsenic. None of the exposure indicators was statistically significantly associated with the risk of kidney cancer.

Kurttio et al. (1999) reported that bladder cancer tended to be associated with arsenic concentration and daily dose during the 3rd to 9th years prior to the cancer diagnosis, but the risk for bladder cancer was statistically significant only for water arsenic concentrations >0.5 µg/L after a short latency period. This is shown in Table 2.

TABLE 2
 Kurttio et al., 1999, Bladder Cancer Risk

Water Arsenic	Bladder Cancer Risk OR (95% CI)	
	Short Latency (exposure 3 to 9 years before diagnosis)	Long Latency (10 years and earlier)
<0.1 µg/L	1	1
0.1-0.5 µg/L	1.53 (0.8-3.1)	0.81 (0.4-1.6)
>0.5-64 µg/L	2.44 (1.1-5.4) ^a	1.51 (0.6-1.6)

^a p <0.05.

Additional evidence of no increased risk of bladder cancer is provided by a second analysis by Kurttio et al. (1999) that considered a different estimate of exposure; in this

analysis, no statistically significant results were seen with cumulative water arsenic dose at short or long latencies. This is shown in Table 3.

TABLE 3
Kurttio et al., 1999, Additional Data on Bladder Cancer Risk

Cumulative Arsenic Dose	Bladder Cancer Risk OR (95% CI)	
	Short Latency (exposure 3 to 9 years before diagnosis)	Long Latency (10 years and earlier)
<0.5 mg	1	1
0.5 – 2.0 mg	1.61 (0.7, 3.5)	0.81 (0.4-1.7)
>2.0 mg	1.50 (0.7-3.2)	0.53 (0.3, 1.1)

The USEPA should investigate the significance of these results. We believe the statistically significant association observed with a short latency period is merely a statistical anomaly and that the results do not provide evidence of an increased risk associated with low arsenic exposures. Bates et al. (1995) noted in the discussion of results from their study in Utah that Taiwanese exposed to arsenic for 40 or more years had the highest relative risks and that other studies had also found long latencies for bladder cancer. Thus, we feel that the correct interpretation of the increased risk observed by Kurttio et al. for a short latency period is statistical chance.

Kurttio et al. (1999) felt there was a suggestion of a synergistic effect (interaction of arsenic and smoking to increase risk) of arsenic; smoking-elevated arsenic exposure tended to increase bladder cancer risk among smokers. The investigators noted: "Experimental studies also suggest that arsenic compounds promote the carcinogenicity and genotoxicity of the known carcinogens and genotoxic compounds." "Exposures earlier than 10 years before cancer diagnoses did not show an association with bladder cancer risk. Hence, relatively recent arsenic exposure appears to be more relevant for bladder cancer risk. This is in concordance with the hypothesis that arsenic compounds act as promoters and/or co-carcinogens in the late stage of carcinogenesis."

The USEPA did not address how these results that suggested a synergistic effect might affect the exposure-response curve. Here we have human data on risks at low levels and a suggestion about mechanism or mode of action, but no discussion of its importance by the USEPA. How important is latency? If short-term exposure is more important than long-term exposure, does this suggest that some populations may be able to more effectively excrete (methylate) arsenic after chronic low-level exposure? The USEPA should investigate all of these issues.

Kurttio et al. noted: "More studies are needed to confirm the possible association between arsenic and bladder cancer risk at such low exposure levels." The study in Finland was conducted in a population exposed to arsenic drinking water levels less than found in the Utah studies. Although we feel the results may be due to statistical chance rather than suggesting an association, we certainly agree with Kurttio et al. that more analytical epidemiological studies are needed, especially case-control or cohort studies of persons with exposures to low arsenic levels in water. Why is the USEPA not conducting these studies?

It is also interesting to compare the results of studies in Utah by Bates et al. (1995) and studies in Finland by Kurttio et al. (1999), since both evaluated latency and low arsenic exposures. The following information shows no statistically significant increased risks of bladder cancer associated with cumulative arsenic doses ranging from <0.5 mg to >10.2 mg. The USEPA should investigate these results in terms of their importance to health risk.

Is there really an increased risk of bladder cancer at 50 µg/L based on the current epidemiological information? The data presented in Table 4 suggest no increased risk.

TABLE 4
 Risk of Bladder Cancer

Cumulative Arsenic Dose	Bladder Cancer Risk OR (95% CI)	
	Short Latency	Long Latency
Kurttio et al. (1999)	(<9 years)	(10 years and earlier)
<0.5 mg	1	1
0.5 – 2.0 mg	1.61 (0.7, 3.5)	0.81 (0.4-1.7)
>2.0 mg	1.50 (0.7-3.2)	0.53 (0.3, 1.1)
Bates et al. (1995)	(<9 years)	(40 to 49 years)
<4.4 mg	1	1
4.4 – <6.9 mg	1.18 (0.6-2.3)	0.52 (0.2-1.8)
6.9 – <10.2 mg	0.97 (0.5-2.0)	0.68 (0.2-2.1)
10.2 and >	1.11 (0.6-2.2)	0.65 (0.2-2.4)

Omitted in the IRIS document is how smoking may affect the cancer risk. Smokers may be more susceptible to bladder and lung cancer when exposed to high arsenic levels. There is some evidence to support this. Moore et al. (1997) investigated the relationship between arsenic ingestion and genetic damage to the urothelium in two cross-sectional biomarker studies, one in Nevada and one in Chile.

In both studies, they found that increased levels of micronucleated cells (MNCs) in exfoliated bladder cells were associated with elevated concentrations of arsenic in drinking water, suggesting that arsenic induces genetic damage to bladder cells. To further investigate this relationship, they conducted an intervention study in a subset of highly exposed men (n = 34) from the cross-sectional study in Chile. Subjects whose usual source of water contained about 600 µg/L arsenic were supplied with water lower in arsenic (45 µg/L) for 8 weeks, allowing ample opportunity for renewal and exfoliation of bladder epithelial cells.

Mean urinary arsenic levels decreased during the intervention. Bladder MNC prevalence also decreased (p <0.05). Among smokers, MNC prevalence decreased from 4.45 MNCs/1,000 cells pre-intervention to 1.44 MNCs/1,000 cells post-intervention (p = 0.002). Among nonsmokers, the decrease was much smaller: 2.04 MNCs/1,000 cells pre-intervention to 1.90 MNCs/1,000 cells post-intervention (p = 0.25), suggesting that smoker's bladder cells could be more susceptible to genotoxic damage caused by arsenic. The reduction in bladder MNC prevalence with reduction in arsenic intake provides

further evidence that arsenic is genotoxic to bladder cells but is the increased risk confined to smokers as some epidemiological studies suggest.

The Bates et al. (1995) Utah data also raise the possibility that smoking potentates the effect of arsenic on risk of bladder cancer. The Lewis et al. (1999) cohort study, which included only nonsmokers, also found no association between arsenic exposures in water and lung or bladder cancer. In Finland, if one accepts that the results are not due to a statistical anomaly, smoking was found to affect bladder cancer risk among smokers who were exposed to low drinking water arsenic levels for 3 to 9 years (Kurttio et al., 1999).

In summary, we are very concerned that the continued use of the Taiwanese data as the only source of epidemiological data will result in an overestimation of the health risk of arsenic in drinking water. The USEPA should consider the weaknesses of the Taiwanese data and further evaluate the health risk of arsenic in drinking water in the US through proper studies. It is clearly evident that the health risk studies of arsenic in drinking water in the US do not corroborate the Taiwanese data.

The SAB is also charged with evaluation of USEPA's modeling effort. It is not intuitive how the current health risk assessment could utilize essentially the same data as previous work and yet arrive at twice the anticipated cancer risk associated with drinking water exposure.

Recommendations

In addition to recommendations made above, ABCWUA requests that USEPA consider the following:

1. The SAB should comment on the validity of expressing unit risk in drinking water at sub-microgram per liter levels. While some labs may have the capability to detect arsenic at the levels expressed for unit risk in drinking water (0.14 µg/L for women and 0.21 µg/L for men), the technology is limiting and therefore there is a higher margin of error.

2. USEPA's 2003 public involvement policy (www.USEPA.gov/policy2003/policy2003.pdf) states that USEPA should distribute materials to make the public aware as soon as such information is available and that "the more complex the issue and greater the potential for controversy or misunderstanding, the earlier the Agency should distribute the materials." The IRIS toxicological review document for arsenic is both complex and lengthy. USEPA should extend the public comment period for both the SAB and the public beyond the less than 60 days allowed.

3. ABCWUA strongly advocates for effective research planning to support timely and appropriate regulatory decisions. USEPA's Office of Water recently demonstrated a similar interest by publishing its National Water Program Research Strategy (<http://www.USEPA.gov/waterscience/strategy/>). A recent paper by Seidel and Roberson (2009) in Journal AWWA found that only one third of the research in USEPA's Arsenic Research Strategy was incorporated into the 2001 arsenic regulation. As demonstrated,

a substantial portion of the research that was not completed would have informed this hazard assessment. Either completion of the other two thirds of this research, or development and implementation of an updated arsenic research plan needs to be completed prior to the Agency contemplating any potential revisions to the 2001 SDWA arsenic regulation.

USEPA has not provided for expert review of the entire draft of the IRIS inorganic arsenic document. We recommend that the SAB provide expert review of the document in its entirety. Similarly, USEPA should provide an opportunity for public comment on the entire draft document.

Finally, while the opportunity to comment on the document is appreciated, ABCWUA looks forward to the opportunity for an extended public comment period to allow the in-depth review that the entire draft IRIS inorganic arsenic hazard assessment warrants.

Sincerely,



Mark S. Sanchez
Executive Director

C: John M. Stomp III, P.E., Chief Operating Officer
Tom Curtis, AWWA

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