APPENDIX F

EPA RESPONSES TO COMMENTS
MAY 2003 WELLS G&H SUPERFUND SITE
OU-3 ABERJONA RIVER STUDY
BASELINE HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT REPORT

June 28, 2004

See attached address list

Re: EPA responses to comments on the May 2003 Wells G&H Superfund Site OU-3 Aberjona River Study Baseline Human Health and Ecological Risk Assessment

Dear addressees:

Thank you for preparing and submitting comments on EPA's Wells G&H Superfund Site OU-3 Aberjona River Study Baseline Human Health and Ecological Risk Assessment (BRA), dated May 2003, which focused on potential risks from surface water, sediments and soils along the Aberjona River from Route 128 to the Mystic Lakes. EPA initially released the baseline human health risk assessment in March 2003 and then followed up with the combined human health and ecological risk assessment in May 2003. During the Spring and Summer 2003 time period, EPA held numerous public meetings explaining the BRA results.

By November 2, 2003, EPA received approximately 128 pages of comments on the BRA from the following parties:

A. Gradient Corporation (contractor to Solutia, Inc., and Stauffer Management Company, LLC who act on behalf of the primary settlers to the Industri-Plex Superfund Site 1989 Consent Decree);
B. S.R. Hansen & Associates (contractor to Solutia, Inc., and Stauffer Management Company);
C. Solutia, Inc., Stauffer Management Company, LLC, and their legal councels Hush & Eppenberger, LLC, and Ropes & Gray, LLP, respectively;
D. Aberjona Study Coalition (prepared by Cambridge Associates, Inc., Tufts University, and Eco-Solutions, Inc.);
E. City of Woburn (prepared by University of Connecticut, Technical Outreach Services to Communities Program);
F. Town of Winchester; and
G. Other Comments

Please find attached, EPA's responses to each of the parties' comments in the order presented above (e.g., A. Gradient Corporation, B. S.R. Hansen & Associates, etc.,). EPA's responses pertaining to the Massachusetts Department of Environmental Protection (DEP) were prepared in consultation with the DEP. The responses are inserted immediately after every comment in the text of each party's original comment letter. The responses are denoted in bold italic text and begin with "***EPA Response.***"

Based upon EPA's responses to comments, EPA will revise the BRA. Notable aspects of the revision will include:

- § Recalculation of exposure point concentrations based on the use of EPA's updated software program ProUCL (version 3.0);
- § Evaluation of recent sediment and floodplain soil samples collected along the Aberjona River in Winchester, south of Beacon Street (station AJRW);
- § Evaluation of recent sediment core data collected from nine locations along the Aberjona River between Route 128 and the Mystic Lakes (SC05 through SC13); and
- § Evaluation of recent surface water baseflow and storm event data collected from 5 surface water gauging stations along the Aberjona River between Route 128 and the Mystic Lakes (SW05 through SW10).

EPA expects to release the revised document this summer. The revised BRA will be incorporated into a Comprehensive Remedial Investigation (RI) Report that will provide a detailed discussion on the fate and transport of contamination along the entire Aberjona River from the Industri-Plex Superfund Site (North of Route 128) in Woburn to the Mystic Lakes in Winchester and Medford. The RI will further explain potential human health and ecological risks along the river. EPA's next public meetings on the Aberjona River will take place after the release of the Comprehensive RI Report.

If you have further questions on the EPA's responses to comments, please contact Angela Bonarrigo at (617) 918-1034, or me at (617) 918-1323.

Sincerely,

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October 13, 2003

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1 Overview

This report presents Gradient's comments on EPA's "Baseline Human Health Risk Assessment Report, Wells G&H Superfund Site, Aberjona River Study, Operable Unit 3, Woburn, MA", dated March 2003. In general, we believe that EPA's risk assessment overestimates risk due to the use of several overly conservative and unrealistic exposure assumptions. The end result is that the calculated risks significantly overestimate the actual risks likely to be experienced by the local population. In this report we discuss the overly conservative nature of some exposure assumptions, and also show that the use of more realistic (yet still conservative) exposure assumptions leads to risks that are within EPA's range of acceptable risk levels.

EPA Response: See responses to specific comments below.

Chapter 2 presents our specific comments on the text and appendices. The most problematic exposure parameters that have been overestimated include the exposure frequency, the sediment ingestion rate, and the exposure point concentrations, as discussed below.

- The exposure frequencies used by EPA were based solely on professional judgment. However, it is important to note that the frequency with which a receptor might contact sediment would be far less than the frequency with which he or she might visit an area to take a walk. The exposure frequencies have been overestimated because they do not reflect the fact that the Wells G&H wetland and the Cranberry Bog are unattractive and undesirable areas for wading. These areas are overgrown with 10-ft high reeds, have soft sediment, and have mosquitoes during the summer months. It is difficult to access the sediment at stations WH and CB-03 due to the presence of dense vegetation, including vines and brambles. At some of the sample locations in CB-03, it is necessary to descend a steep embankment covered in dense vegetation to access the sediment in the bog, which itself is densely filled with tall reeds. To access these points, we found that a 6-ft tall adult was waist-deep in brush. The three southernmost points at CB-03 appear to be located in an undesirable channel of stagnant water that is choked with decaying leaves, and to access these sample points, it is necessary to walk through a dense tangle of vines and brambles. For these reasons, it is highly implausible that a young child, ages 1 to 7 years old, would contact the sample locations at WH or CB-03 at all, let alone at the frequencies assumed by EPA. Even for a more plausible adolescent receptor, EPA's assumption that wading would occur 4 days/week for 6 months/year (104 days/year) at the Cranberry Bog, and 3 days/week for 6 months/year (78 days/year) in the Wells G&H wetland is a significant overestimate, due to the lack of accessibility and desirability of these areas. Even if a boardwalk were constructed at the Wells G&H wetland in the future, it is highly unlikely that a young child would leave the boardwalk to contact sediment with a frequency of 78 days/year. Moreover, it should be noted that a recent article in the Woburn Daily Times Chronicle (8/26/03) indicated that it is possible that <u>no</u> nature trail will be built in this area. EPA's overestimated exposure frequencies overestimate risk in these areas.

EPA Response: Exposure frequencies for the exposure stations are based on current land use as well as future land use assumptions and do factor in professional judgment. However, the intent of the selected exposure frequencies is to adequately protect human health without being unrealistically conservative. All the samples applied to the human health risk assessment were thoroughly investigated by the Agency and risk assessors and considered reasonably accessible. The Cranberry Bog and Wells G&H wetland are well utilized areas by the neighborhood and

community. Future plans by the City of Woburn include development of the Wells G&H wetland into a passive recreational area. The Cranberry Bog is surrounded by residences, making it plausible that young children living in these residences may contact sediments and soils in areas adjacent to their yards. No fencing is in place to prevent a child from wandering from their yard into the wetland, which in some locations is a distance of as little as 5 to 10 feet. Therefore, for the Cranberry Bog, it is not unreasonable to assume an exposure frequency approaching one used in a residential setting. The Wells G&H wetland has periodically been used by community groups as a paint ball range. During each visit to these areas, adults and children were observed utilizing these areas (e.g., walking dogs, playing in groups, sliding down the embankments). It is plausible that a child engaged in a game of capture the flag or while participating in a paint ball activity would slide down a steep embankment or run through an area of stagnant water filled with decaying leaves. In addition, children and adults are naturally attracted to the edge of wetlands and surface water to observe the environment. Samples located in areas overgrown with reeds, vines, brambles or with excessively soft sediments, and considered not accessible, were not quantitatively evaluated for human exposures. For some samples, it was found that access may be more difficult via one path, but easier if approached from a different direction. For example, some of the CB-03 sampling locations may be more difficult to access by simply descending the bank, but could be easily accessed after entering the wetland in a less steep and drier area, and then traveling through the wetland to these sampling locations. Should the City of Woburn elect not to construct a boardwalk (station BW) into the Wells G&H wetland, then such future exposure assumptions used for station BW may over estimate risk.

- EPA's assumption that a child obtains 50% of his daily soil/sediment ingestion from the site is based solely on professional judgment. Since soil ingestion is believed to occur sporadically throughout the day as a consequence of hand-to-mouth activity, the assumption that a child obtains 50% of his daily soil/sediment ingestion from the site implies that the child is ingesting sediment for a significant portion of the day. Since sediment in the Cranberry Bog and the Wells G&H wetland is difficult to access due to the dense vegetation, and the areas are undesirable as wading or play areas, the duration of any sediment exposure event is likely to be very brief. Thus the assumption that a child obtains 50% of his daily soil/sediment ingestion from the site overestimates risk.

EPA Response: Since sediments adhere to body surfaces, it is not unrealistic to assume that 50% of a receptor's intake would occur from the study area. This assumption acknowledges that human receptors would not spend 100% of their recreational time in impacted areas.

- Recent soil ingestion rate studies suggest that the average and high-end soil ingestion rates for children are lower than the values used by EPA (200 mg/day for a child and 100 mg/day for an adult) based on 1994 Region I Guidance. EPA's 1997 Exposure Factors Handbook recommends soil ingestion rates of 100 mg/day for a child and 50 mg/day for an adult (USEPA, 1997). Use of these soil ingestion rates would decrease risks by a factor of two.

EPA Response: The child and adult soil ingestion values recommended in the 1997 Exposure Factors Handbook represent central estimate values and are appropriate for use in a central tendency evaluation. The Exposure Factors Handbook does not recommend upper percentile values for use with a reasonable maximum scenario. Therefore, EPA Region I values, recommended for use in a

reasonable maximum scenario, were selected for use. These values are consistent with ingestion rates recommended by MADEP and, as stated in the Exposure Factors Handbook, are within the range of ingestion estimates from published studies. The central tendency ingestion rates utilized are the same as those recommended in the Exposure Factors Handbook for use in a central tendency evaluation.

- At several stations, EPA has based risks on a highly uncertain estimate of the average arsenic concentration that people might be exposed to, resulting in an overestimate of risk. The exposure point concentrations (EPCs) for several exposure areas (WH, CB-03, 13/TT-27) are highly uncertain and based on skewed data sets. This is especially problematic in that these datasets, as analyzed by EPA, yielded risks of potential concern. These stations are discussed below.

EPA Response: See overall response provided below for these three data sets.

- At station WH, the dataset is highly skewed due to the inclusion of one sample with a high arsenic concentration. This sample (SD-12-01-ME with 3230 mg/kg) is the southernmost sample that EPA included in the WH area (Figure 1). The skewed dataset strongly suggests that this exposure area is not well delineated, and thus that this dataset may be inappropriate for use in risk management decisions. In addition, it is an unexpected observation that two samples with the same ID, taken by different contractors (SD-12-01-ME and SD-12-01-FW), appear to have been collected from two different locations (Figure 1).

EPA Response: SD-12-01-FW was collected in 1995 while SD-12-01-ME was collected in 1997. The samples were originally intended to be co-located. However, when the area was returned to in 1997, the sampling location was moved slightly to include a depositional area that had not been previously characterized. These sample locations are illustrated on Figure 2-3.

- At station CB-03, on the western side of the Cranberry Bog, EPA used an arsenic EPC equal to the maximum concentration (1410 mg/kg) detected in this exposure area, because the 95%UCL exceeds the maximum concentration. EPA's use of the maximum concentration as the EPC indicates that the dataset for CB-03 is skewed, with the maximum concentration located at the southernmost sample (Figure 2). The sediment concentrations in 11 of the 12 samples at CB-03, ranging from 9.1 to 510 mg/kg, are much lower than the maximum concentration, and the average concentration of all 12 samples is only 272 mg/kg. Based on this dataset, if a person visited each sample location with equal frequency, then, on average, that individual would be exposed to an average concentration much lower than 1410 mg/kg. Therefore, using an EPC of 1410 mg/kg in all likelihood overestimates the risks for CB-03. In addition, the sediment in the CB-03 exposure area is not very accessible because these sample locations are located in areas with dense vegetation, including vines, brambles, and tall reeds.

- At station 13/TT-27, on the west side of the Wells G&H wetland, the dataset is highly skewed due to two samples with high concentrations. EPA used an arsenic EPC that is equal to the maximum concentration (4210 mg/kg) because the calculated 95%UCL exceeded the maximum. EPA's use of the maximum concentration as the EPC indicates that the 13/TT-27 area has too few samples to be well characterized. The skewed dataset strongly suggests that this exposure area is not well delineated, and thus that this dataset may be inappropriate for use in risk management decisions. The average concentration of all samples is 840 mg/kg. Based on this dataset, if a person visited each sample location with equal frequency, then, on average, that individual would be exposed to an

average concentration that is much lower than 4210 mg/kg. Therefore, using an EPC equal to the maximum concentration of 4210 mg/kg likely overestimates risks for 13/TT-27.

EPA Response: According to EPA guidance, the arithmetic average concentration is not to be used as the reasonable maximum exposure point concentration (EPC). The reasonable maximum EPC in the report were calculated appropriately in accordance with EPA guidance. EPA acknowledges that the reasonable maximum EPC used at a small number of stations maybe uncertain due to one or a small number of elevated arsenic detects compared to the remainder of the data set. This uncertainty is specifically applicable to stations WH (sample SD-12-01-ME; 3230 mg/kg), CB-03 (sample CB-03-11; 1410 mg/kg), and 13/TT-27(samples SD-13-01-FW and SD-13-02-FW; 4210 mg/kg and 2480 mg/kg, respectively). During public presentations, this uncertainty was acknowledged, and the public was informed that the risk estimated for these stations was largely attributable to elevated arsenic levels in one or a small number of samples. This information will be added to the text of the risk assessment, with the locations of the highest arsenic levels identified. The highest concentatrions of arsenic found in sediments can be extracted from Figure 2-24. In addition, EPA has recently released version 3.0 of the ProUCL calculation software (version 2.1 was used for the draft report). ProUCL version 3.0 is being used in revisions to the draft report. Its use may result in a more accurate estimate of exposure point concentrations for these stations.

EPA's uncertainty analysis should be expanded to more clearly articulate how many of the assumptions are biased towards overestimating rather than underestimating potential health risks. The impact of these conservative assumptions on the uncertainty in the calculated risks should be explained. In addition, as noted below in Appendix B, EPA's cancer slope factor for arsenic is very conservative, especially as applied to U.S. populations experiencing relatively low levels of exposure, and thus will further tend to overestimate the cancer risk from exposure to arsenic in sediment.

EPA Response: The uncertainty section will be expanded to include additional notations as to those assumptions that tend to overestimate or underestimate risk. In addition, the discussion in the uncertainty section relative to the arsenic toxicity factor will be expanded.

Chapter 3 presents the results of Gradient's deterministic risk calculations to demonstrate the implications of alternate (and more realistic) exposure assumptions for the risk estimates at Stations WH, NT-1, NT-2, NT-3, 13/TT-27, and CB-03. The exposure frequencies were reduced to 6 days/year, to reflect the fact that the Wells G&H wetland and the Cranberry Bog are very undesirable areas for wading, because they are covered in dense vegetation, including vines and brambles, and are difficult to access. In addition, the soil ingestion rates were reduced to the more recent values in EPA's 1997 Exposure Factors Handbook. The use of more realistic, yet still conservative, exposure assumptions results in deterministic RME cancer risks that are at or below $2x10^{-5}$ at all six of these stations. Noncancer risks are also at or below 0.4 at all six stations. These risks do not exceed EPA's permissible risk limits.

EPA Response: Exposure frequencies used in Gradient's deterministic risk calculations are not sufficiently protective of reasonable maximum exposures that are occurring or may occur in the future at these stations. These areas are currently utilized by the community at a higher frequency than would be accounted for by an exposure frequency of 4 to 6 days/year. Future plans to develop these areas into more attractive and more highly utilized recreational spaces would only serve to increase the frequency with which individuals visit the site and contact impacted media. The deterministic calculations performed by Gradient and provided in Chapter 3 of these comments have not been reviewed for accuracy since the exposure assumptions (i.e., exposure frequencies and soil ingestion rates) used are not sufficiently protective of current or potential future reasonable maximum exposures

in these areas. In addition, the reference dose for arsenic used in these calculations is not appropriate for childhood exposures, even if occurring for less than 7 years in duration.

Chapter 3 also presents the results of Gradient's probabilistic (Monte Carlo) analyses. In order to assess the uncertainty associated with EPA's deterministic risk calculations, Gradient performed probabilistic risk calculations for the ingestion of arsenic in sediment, for current risk at CB-03, and future risk at stations WH, NT-1, NT-2, NT-3, and 13/TT-27. for both cancer and noncancer risks. Ingestion of arsenic in sediment is the major contribution to EPA's cancer and noncancer risks. The probabilistic risk calculations are presented to help put EPA's risks into perspective, and because USEPA Region I "considers Monte Carlo analysis to be an acceptable approach for analyzing uncertainty in the risk assessment" (USEPA, 1994).

The results of the probabilistic risk calculations indicate that all of the 95th percentile cancer risks are at or below $3x10^{-5}$. The 95th percentile risk is $1x10^{-5}$ at both WH and CB-03, $2x10^{-5}$ at NT-1, $6x10^{-6}$ at NT-2, $4x10^{-6}$ at NT-3, and $3x10^{-5}$ at 13/TT-27. The 95th percentile noncancer hazards range from 0.07 to 0.95 and are all less than EPA's acceptable hazard of 1.0. The 95th percentile risk means that there is a 95% probability that the risks to any one individual will be below this value.

The probabilistic risks are substantially lower than EPA's individual risk estimates for the ingestion of arsenic in sediment (Table 1). Although the probabilistic risks are only for the ingestion of arsenic in sediment, this pathway represents a major portion (about 75%) of EPA's total cancer risks for these stations. This analysis indicates that EPA's RME risks are very high-end values and hence are not representative of RME values. Use of a more plausible range of exposure inputs results in cancer risks falling within EPA's acceptable risk range of 10^{-6} to 10^{-4}, and noncancer hazards falling below 1.0.

EPA Response: The probabilistic calculations performed by Gradient and contained in Chapter 3 have not been reviewed for accuracy. Specific assumptions used in Gradient's probabilistic assessment are not sufficiently protective of current or potential future site conditions. Should it be determined that probabilistic information would be useful during the risk management process, an evaluation will be conducted at that time by EPA.

Appendix A presents the results of recent soil ingestion rate studies to demonstrate that the sediment ingestion rates used by EPA overestimate likely sediment ingestion rates. EPA used RME sediment ingestion rates of 200 mg/day for a child and 100 mg/day for an adult, based on 1994 EPA Region I Guidance. EPA's 1997 Exposure Factors Handbook recommends soil ingestion rates of 100 mg/day for a child and 50 mg/day for an adult (USEPA, 1997). Use of these sediment ingestion rates would decrease predicted risks by approximately a factor of two.

EPA Response: As stated previously, the child and adult soil ingestion values recommended in the 1997 Exposure Factors Handbook represent central estimate values and are appropriate for use in a central tendency evaluation. Since the Exposure Factors Handbook does not recommend upper percentile values for use with a reasonable maximum scenario, EPA Region I values, recommended for use in a reasonable maximum scenario, were selected for use. These values are consistent with ingestion rates recommended by MADEP and, as stated in the Exposure Factors Handbook, are within the range of ingestion estimates from published studies. The central tendency ingestion rates utilized are the same as those recommended in the Exposure Factors Handbook for use in a central tendency evaluation.

Appendix B presents a discussion of arsenic toxicity to illustrate the very conservative nature of the arsenic toxicity factor, especially as applied to US populations, and provides evidence that the use of

this factor will tend to overestimate the cancer risk from exposure to arsenic in sediment. Appendix B discusses U.S. epidemiological studies of arsenic carcinogenicity, demonstrating that the estimated arsenic exposures to sediment in the Aberjona River are well below the exposures experienced by U.S. populations where epidemiological studies have not found elevated cancer risks. In addition, the estimated arsenic exposures to sediment in the Aberjona River are well below the exposures found in studies of non-U.S. populations that show an increased risk of cancer due to exposure to high concentrations of arsenic in drinking water. Appendix B also discusses the implications of the non-linearity of the dose-response relationship for arsenic carcinogenicity, and the fact that exposure to arsenic in soil has not been shown to cause adverse health effects.

EPA Response: The discussion in the uncertainty section relative to the arsenic toxicity factor will be expanded.

2 Specific Comments on Text and Appendices

Gradient's specific comments on the risk assessment report are presented below by report section and page number.

3.1.2 Identification of Exposure Stations

p. 3-8, 1st ¶. The exposure assumptions at NT-1, NT-2, and NT-3 are implausible. The NT-3 exposure area is a proposed nature trail on the eastern side of the wetland, near Well H, without access into the wetland. NT-2 includes the proposed NT-3 nature trail area, *plus* a pier extending west into the wetland. NT-1 includes the proposed NT-3 nature trail area, *plus* an elevated walkway located farther west in the wetland. The future RME cancer risks at NT-3, NT-2, and NT-1 are calculated by EPA as 1E-04, 2E-04, and 5E-04, respectively. Area NT-3 has an acceptable cancer risk of 1E-04. The cancer risks increase to what EPA concludes to be unacceptable levels (greater than 1E-04) with the addition of the pier (NT-2) and the elevated walkway (NT-1) that extend farther west into the wetland. Both the pier and the elevated walkway (boardwalk) would need to be elevated a few feet off the ground in order not to be subject to flooding. Thus, in order for a child (1 to 6 years old) to be exposed to sediment, he or she would have to leave the boardwalk and engage in activity bringing him or her in contact with sediments on each visit to the boardwalk. This is an implausible assumption given the young age of the children, and the fact that children of this age would be under supervision. It is unlikely that children would be allowed to leave the boardwalk, especially on each visit, and particularly if the area adjacent to and below the boardwalk is filled with dense vegetation.

EPA Response: It is acknowledged that children and adults would be unlikely to leave a hypothetical boardwalk each time the study area is visited. However, without the boardwalk being fenced and the exact construction details not available, it would be remiss to assume a lesser degree of exposure. This evaluation is intended to evaluate future uses proposed by the City of Woburn as described in the report. If the City of Woburn elected not to construct a pier or boardwalk, or design a pier or boardwalk to minimize exposures to sediments, then EPA may need to re-evaluate the future risks associated with NT-2 or NT-3. If the City of Woburn decided to construct a boardwalk or pier into the wetlands and unacceptable risks remained under the NT-2 or NT-3 exposure scenarios, then cleanup alternatives would need to be considered for the area to reduce those risks.

For the exposure stations on the east side of the Wells G&H wetland, EPA used an exposure frequency of 78 days/year, based solely on professional judgment. However, an exposure frequency of 78 days/year (3 days/week for 6 months/year) is unrealistically high given the fact that this portion of the wetland is located immediately adjacent to the shooting range of the rod and gun club, the wetland is filled with reeds, the sediment is soft, and the area has mosquitoes during the summer months. These attributes would render the area unattractive as a wildlife viewing or recreational area. We also note that exposures to sediment do not occur unless the person leaves the path or boardwalk. EPA has blurred the distinction between how often someone visits the area to take a walk, and how often he or she might actually contact sediments by wading. Because the wetland lacks desirability as a play area, a person is unlikely to contact sediment each time he or she visits the area to take a walk. EPA's scenario implies that a child would ingest sediment during each of his or her 78 visits per year. This assumption is unrealistic, even for a reasonable maximum exposure scenario.

EPA Response: The Wells G&H wetland has been reported by residents as an area that is used daily by nearby residents for nature walks, and recently, has been utilized periodically as a paint ball range by community children. Activities reported and observed as occurring include fishing, catching frogs

and insects and playing games. These activities may all result in contact with the wetland media. The 78 day/year-exposure frequency for the Wells G&H wetland area is for future exposures. It is likely that children and adults would visit this area more frequently than 78 days per year. In fact, residents have stated to EPA that they currently go to this area nearly every day. The 78 days/year exposure frequency is intended to provide a reasonable maximum estimate of the number of days of sediment and surface water contact per year for future site use in the Wells G&H wetland area. The total number of visits per year, which may include visits without sediment and surface water contact, is acknowledged as likely to exceed 78 days per year.

3.2.2.5 Data Evaluation

p. 3-16. Data Evaluation. The Aberjona River floods periodically. EPA should explain the basis for its implied assumption that samples collected in 1995 are still representative of current conditions, *i.e.*, whether EPA has evaluated co-located samples to show that 1995 and 2001 samples have similar concentrations, or whether EPA has studied the temporal variation in arsenic sediment concentration over time.

EPA Response: The 2001 sampling was conducted to fill data gaps and considered new locations. Co-located sampling was conducted in 1997, when some of the 1995 sediment sampling locations were re-sampled. The results for those co-located samples were similar and indicate that concentrations are remaining relatively stable over time. Appendix A.2 (M&E Supplemental Data Compendium) contains a comparison of the co-located 1995 and 1997 data.

p. 3-17, 2nd ¶, 1st sentence. Cr(VI) was not detected in a sample with total chromium of 930 mg/kg, but was detected in a sample with total chromium of 13,400 mg/kg. On this basis, EPA assumes Cr(VI) is not present at sediment concentrations equal to or less than 930 mg/kg, and that Cr(VI) is present at 0.13% of the total chromium concentration at sediment concentrations greater than 930 mg/kg. This sentence should make it clear that the estimate of 0.13% of Cr(VI) is based on only one sample with a total chromium concentration of 13,400 mg/kg. This is a very conservative assumption, since the sample with Cr(VI) detected (13,400 mg/kg) has a concentration two orders of magnitude higher than the next highest concentration sample where Cr(VI) was not detected (930 mg/kg). Based on the observation that Cr(VI) was only detected in a sample with total chromium concentration of 13,400 mg/kg, EPA should apply the Cr(VI) assumption only to samples that have concentrations of total chromium greater than 10,000 mg/kg, as there is no justification for a broader application of this assumption.

EPA Response: The uncertainty section will be modified to explain that the 0.13% value is based on the results of a single detected concentration of Cr(VI) in those samples analyzed in 2002 via ion chromatography. Due to the lack of Cr(VI) ion chromatography data in samples with total chromium levels between 930 mg/kg and 13,400 mg/kg, 930 mg/kg was selected as the threshold above which Cr(VI) may be present. Cr(VI) data collected by an alternate colorimetric method were not used in this site-wide extrapolation due to interferences with the method that resulted in some data rejection. However, those colorimetric data that were not rejected were used on a station-by-station basis.

p. 3-17, 3rd ¶, 2nd sentence. EPA states that "stations NT-2, NT-3 and WG have *station-specific* results demonstrating that Cr(VI) was non-detect at the location of the *maximum* detected total chromium value". The maximum concentration of total chromium at all three of these stations was 2570 mg/kg, in sample SD-WG-10. However, the report does not present Cr(VI) results for a sample with a total chromium concentration of 2570 mg/kg. EPA also implies (in the first paragraph on page 3-17) that there are no Cr(VI) results for total chromium concentrations between 930 and 13,400 mg/kg. Moreover, Appendix C.4 does not contain Cr(VI) results for any samples with total chromium concentrations between 930 and 13,400 mg/kg. Therefore the basis for the statement regarding station-specific Cr(VI) results for samples

at stations NT-2, NT-3, and WG is unclear. If EPA has Cr(VI) data for samples with total Cr concentrations between 930 and 13,400 mg/kg, then these data should be presented.

EPA Response**: Cr(VI) was analyzed using a colorimetric method and by ion chromatograph. Since some of the colorimetric results were rejected, the ion chromatography results were deemed as the most reliable and used to develop site-specific assumptions concerning the relative presence of Cr(VI). However, those colorimetric Cr(VI) results that were not rejected (e.g.., at SD-WG-10) were used as station-specific results for those stations that included that data point (NT-2, NT-3 and WG). Therefore, station NT-2, NT-3 and WG are stated as having station-specific results that demonstrate non-detect levels of Cr(VI) above 930 mg/kg total chromium.

p. 3-17, last ¶. The assumption that all chromium in surface water, surface soil, and fish exists as Cr(VI) has no scientific basis and is unrealistically conservative. It is not reasonable to assume that chromium in surface water exists entirely as Cr(VI) when EPA's data show that most of the chromium in these sediments exists as Cr(III).

EPA Response: In the absence of medium-specific Cr(VI) results for surface water, surface soil and fish, total chromium results were assumed to be Cr(VI). This assumption is protective and reflects the lack of site-specific chromium speciation data for these media.

3.2.3 Identification of COPCs

p. 3-19. It should be noted that the use of residential soil PRGs as a COPC screening criterion for surface soil along the streambank is very conservative. The level of exposure in residential scenarios is well above what is contemplated for recreational exposures. Similarly, using drinking water PRGs as a COPC screening tool for evaluating surface water in the Aberjona River, which is not used as a source of drinking water, is overly conservative.

EPA Response: It will be noted in the uncertainty section that conservative screening values (i.e., PRGs) are used when selecting COPCs so as not to omit a compound that might contribute significantly to risk.

p. 3-19. The AWQC for arsenic should not be used as an ARAR. The AWQC was derived using a toxicity value for inorganic arsenic. The majority of arsenic in fish exists as arseno-sugars (*e.g.*, arsenobetaine, arsenocholine). The fraction of inorganic arsenic in freshwater fish has been reported to be less than 10% (Schoof *et al.*, 1999). The arseno-sugars are essentially non-toxic because they are excreted unmetabolized in a relatively short time. EPA is currently revising the AWQC for arsenic based on this information (*Fed. Reg.* Oct. 12, 2000). In addition, the arsenic AWQC is more than 500 times lower than the maximum contaminant level (MCL), the regulatory limit for arsenic in drinking water that is based on a lifetime of daily exposure.

EPA Response**: The BRA is not intended to identify ARARs. ARAR identification will ocurr in the Feasibility Study. The uncertainty section will discuss that the arsenic AWQC is currently under review by EPA. Regardless of the use of this value, arsenic would continue to be selected as a surface water COPC.

p. 3-20. EPA notes that the background fish tissue lead level is 0.34 mg/kg but then delays use of this value until the risk characterization. Lead should be eliminated as a COPC for fish tissue at this stage by comparison to background.

EPA Response: According to EPA Region I guidance, contaminants are not typically eliminated as COPCs based on a comparison to background. Therefore, lead has been retained as a fish COPC until the risk characterization section.

3.2.4 Determination of Exposure Point Concentrations

p. 3-22. Exposure Point Concentrations.

Gradient is unable to reproduce the EPC calculations for stations NT-1 and 13/TT-27 because the database provided by EPA[1] does not include some of the samples that EPA used in their exposure areas (according to Table C.1-1). At NT-1, seven samples are missing, and at 13/TT-27, one sample is missing.

EPA Response: All data utilized in the EPC calculations are provided in Appendix B.

EPA states (p.3-22, 2nd ¶) that "USEPA requires the use of the 95%UCL on the arithmetic mean concentration for the estimation of both the CT and RME risk", and notes that wherever possible, the 95%UCL has been used as the EPC. This discussion is misleading and should be modified for clarification and consistency with current guidance. EPA should cite their current guidance on calculating EPCs (USEPA, 2002).[2] The 2002 guidance recommends the use of the 99% Chebyshev UCL for certain datasets, and in fact EPA has used the 99% Chebyshev UCL as the EPC for certain datasets. Therefore discussing the use of only the 95% UCL in this discussion is incorrect. In addition, Table 3-3.2, which lists the statistic used for the EPC in each exposure area, is incorrect, because for stations where the EPC is the 99% Chebyshev UCL (for example, at WH), the table states that the 95%UCL was used. Table 3-3.2 should be corrected.

EPA Response: The 99% Chebyshev UCL was used as the EPC for certain datasets. This information is presented in the ProUCL documentation provided in Appendix B.

The EPC (and hence the risk) in the WH exposure area is heavily influenced by the samples that EPA chose to include in this exposure area. EPA selected the boundary of the WH exposure area, presumably based on professional judgment. However, EPA has not demonstrated that all of the sample locations they included in the WH exposure area are uniformly accessible. The arsenic EPC of 1900 mg/kg for the WH exposure area is heavily influenced by EPA's inclusion of one sample with a very high arsenic concentration (SD-12-01-ME). The WH samples (WH-01 to WH-10) included in the WH exposure area range from 4.7 to 424 mg/kg, and have an average arsenic concentration of 123 mg/kg. However, the last sample included in the WH exposure area, SD-12-01-ME, has a concentration of 3230 mg/kg, which is an order of magnitude higher in concentration than the next highest WH sample. This sample is the southernmost sample within this exposure area (Figure 1). Including this sample yields an EPC for station WH that is potentially biased high. If EPA did not include sample SD-12-01-ME in the WH exposure area, the EPC at WH would be 663 mg/kg[3], and risks at WH would decrease by a factor of 3. Thus, the inclusion of this one sample tends to overestimate the risk for the entire WH exposure area.

At station WH, the EPC of 1900 mg/kg is the <u>99%</u> Chebyshev minimum variance unbiased estimate (MVUE) UCL. Table 3-3.2 should note that the EPC for WH is the 99%UCL, not the 95%UCL, and EPA should provide the statistical rationale for using the 99% Chebyshev UCL as the EPC, as described

[1] Metcalf & Eddy (Wakefield, MA). 2002. "Analytical data for baseline risk assessment, Wells G&H Superfund Site, Aberjona River study, Operable Unit 3, Woburn, Massachusetts." February.

[2] USEPA, 2002. Office of Emergency and Remedial Response (Washington, DC). "Calculating upper confidence limits for exposure point concentrations at hazardous waste sites. Supplemental guidance to RAGS." OSWER Directive 9285.6-10. December. Downloaded from: http://www.epa.gov/superfund/programs/risk/ragsa/ucl.pdf.

[3] The EPC of 663 mg/kg was obtained from the ProUCL program, and is the 99% Chebyshev (MVUE) UCL.

in their 2002 guidance. EPA's use of the 99% Chebyshev UCL indicates that this dataset is highly skewed due to the inclusion of sample SD-12-01-ME. The skewed dataset strongly suggests that this exposure area is not well delineated, and thus that this dataset may be inappropriate for use in risk management decisions.

EPA Response: As stated above, all samples applied to the human health risk assessment were thoroughly investigated by the Agency and risk assessors and considered reasonably accessible. Appendix B documents the use of the 99% Chebyshev UCL for certain data sets. It is acknowledged that the EPC used at this station is uncertain due to one arsenic result (SD-12-01-ME; 3230 mg/kg) that was elevated in comparison to the remainder of the data set. This uncertainty will be added to the text of the risk assessment, with the locations of the highest arsenic levels identified. EPA is currently evaluating the use of ProUCL version 3.0 for revisions to the draft report. Its use may result in a more accurate estimate of exposure point concentrations for this station.

EPA has not provided sufficient information in their UCL guidance (USEPA, 2002) or ProUCL manual (USEPA, 2003) to assess the validity of their choice of the 99% Chebyshev UCL as better than other possible methods. EPA should provide its underlying analyses that led to the UCL recommendations so that experts in the community can review and refine if appropriate. For example, Saranko and Tolson (2003) (provided in Appendix C) show that the UCL of data sets with statistical characteristics similar to the WH dataset may be better estimated with alternative methods that give rise to lower UCL values. Their analysis suggests that EPA's method may have overestimated the EPC, and therefore the risk, for the WH dataset.

EPA Response: Use of the methods provided by the ProUCL software package and associated guidance provided consistent decision-making with respect to calculating UCLs for the various data sets. EPA is currently evaluating the use of ProUCL version 3.0 for revisions to the draft report. Its use may result in a more accurate estimate of exposure point concentrations.

At Station CB-03, on the western side of the Cranberry Bog, EPA used an exposure point concentration (EPC) that is equal to the maximum concentration (1410 mg/kg) detected in this exposure area, because the calculated 95%UCL exceeded the maximum. EPA's use of the maximum concentration as the EPC indicates that the CB-03 area has too few samples to be well characterized. The sediment concentrations in 11 of the 12 CB-03 samples are much lower, ranging from 9.1 to 510 mg/kg, and the average concentration of all 12 CB-03 samples is only 272 mg/kg. Based on this dataset, if a person visited each sample location with equal frequency, then on average, he or she would be exposed to an average concentration that is much lower than the EPC of 1410 mg/kg. Therefore, using an EPC equal to the maximum concentration of 1410 mg/kg likely overestimates the risks for CB-03.

At station 13/TT-27, on the west side of the Wells G&H wetland, EPA used an EPC that is equal to the maximum concentration (4210 mg/kg) detected in this exposure area, because the calculated 95%UCL exceeded the maximum. EPA's use of the maximum concentration as the EPC indicates that the 13/TT-27 area has too few samples to be well characterized. Seven of the nine samples used to characterize this area have arsenic concentrations ranging from 12 to 356 mg/kg, but the last two samples have concentrations of 2480 and 4210 mg/kg, respectively. The average concentration of all samples is 840 mg/kg. Based on this dataset, if a person visited each sample location with equal frequency, then, on average, he or she would be exposed to an average concentration that is much lower than 4210 mg/kg. Therefore, using an EPC equal to the maximum concentration of 4210 mg/kg in all likelihood overestimates the risks for 13/TT-27.

EPA Response: According to EPA guidance, the arithmetic average concentration is not to be used as the reasonable maximum exposure point concentration (EPC). It is acknowledged that the reasonable

maximum EPCs used at a small number of stations are uncertain due to one or a small number of elevated arsenic detects compared to the remainder of the data set. This uncertainty is specifically applicable to stations WH, CB-03 and 13/TT-27. During public presentations, this uncertainty was acknowledged, and the public was informed that the risk estimated for these stations was largely attributable to elevated arsenic levels in one or a small number of samples. This information will be added to the text of the risk assessment, with the locations of the highest arsenic levels identified. As previously stated, EPA will be using ProUCL version 3.0 for revisions to the draft report. Its use may result in a more accurate estimate of exposure point concentrations for these stations.

p. 3-22. Two stations were evaluated that had only one sample to represent the exposure area, stations AM and TT-30. It is not clear why these areas were evaluated as separate exposure areas with only one sample. The risks from these areas are highly uncertain.

EPA Response: It is acknowledged that the estimated risks for these stations are uncertain. However, these samples were not in the vicinity of other samples applicable to the human health risk assessment.

p. 3-22, 3rd ¶. The following statement is incorrect and should be corrected: "In cases where the arithmetic mean value exceeded the maximum detected value, the maximum detected value was used as the EPC for both the RME and CT cases". It is not mathematically possible for the mean to exceed the maximum detected value (because the mean is an average of the maximum and at least one lower value). The sentence would be correct if "95%UCL" were substituted for "arithmetic mean value".

EPA Response: It is possible for the arithmetic mean to exceed the maximum detected concentration. If the maximum detected concentration is an estimated value (i.e., close to the detection limit) and the non-detect values were slightly elevated due to sample dilution or interferences. In this case, it is possible that use of half the slightly elevated detection limit would increase the mean value to slightly greater than the maximum detected value. No change to the text is required.

3.3 Exposure Assessment

p. 3-26, 1st ¶. The age of the child receptor is 1 to 6 years of age (p. 3-26). It is highly implausible that a child this young would have exposure to sediment with the frequency noted by EPA for the various scenarios, due to the fact that the wetlands are undesirable areas for wading, and are difficult to access by a small child due to the presence of dense vegetation both in and around the wetland. It is also implausible that a child would be exposed to sediment over his face, forearms, hands, lower legs, and feet on each and every exposure event, as EPA has assumed (p. 3-34).

EPA Response: This assumption is reasonable given the proximity of residential and future recreational properties. The evaluation estimated risks associated with childhood and adult exposures for a combined duration of 30 years, as prescribed by EPA guidance. For this evaluation, it is assumed that 6 of those years are during childhood and 24 years are during adulthood. Since childhood exposures may in fact occur for longer than 6 years, a young child (age 1 to 6) was selected for evaluation to capture the reasonable maximum childhood risk that may occur during a 6 year childhood exposure duration. Since exposures are assumed to occur during the warmest months of the year, body surface area equivalent to the face, forearms, hands, lower legs and feet may reasonably be exposed. This assumes that individuals, during the warmest six months of the year, wear short-sleeve shirts, shorts and sandals.

p. 3-28, 2nd ¶. It could be better described that NT-3 is a subset of both NT-1 and NT-2, so that it is clear that these exposure areas overlap.

EPA Response: The Executive Summary and Section 5 clearly describe the relationship of NT-1, NT-2, and NT-3 as follows: "Stations NT-1, NT-2, and NT-3 were also evaluated under a future land use scenario due to potential development plans of the City of Woburn within the Wells G&H wetland that may include the construction of a nature trail (station NT-3) with a possible boardwalk (station NT-1) or pier (station NT-2) extending out into the wetland". Further clarification can be found in Appendix C.1 which shows a listing of sediment samples comprising each of these stations. The overlap is clearly shown with this information.

p. 3-28. The frequency with which an individual might go wading and contact sediment is much less than the frequency with which a receptor might *visit* a given exposure area, because the Wells G&H wetland and the Cranberry Bog are undesirable areas for wading. Both the Wells G&H wetland and the Cranberry Bog have very low desirability for wading because to access the wetland, one must walk through dense vegetation including vines and brambles, and the wetland itself is densely filled with reeds, the sediment is soft, and the area is filled with mosquitoes during the summer months. Although a person might walk along the path on the west side of the Cranberry Bog a few times per week, that individual might never contact sediment. Due to the lack of desirability of the Wells G&H wetland for wading, it is highly unlikely that a child who walks along a boardwalk in the future Nature Trail area would leave the boardwalk and contact sediment with a frequency of 78 days/year. Moreover, it should be noted that a recent article in the Woburn Daily Times Chronicle (8/26/03) indicated that it is possible that no nature trail will be built in this area. EPA should provide a basis for their assumptions and should support their exposure frequencies by providing information regarding observations of adults or children wading in sediment during any of their site visits.

EPA Response: All the samples applied to the human health risk assessment were thoroughly investigated by the Agency and risk assessors and considered reasonably accessible. The Wells G&H wetland and Cranberry Bog are areas that are utilized by the surrounding neighborhoods and the community as a whole. The Wells G&H wetland has been reported by residents as an area that is used daily by nearby residents for nature walks, and recently, has been periodically utilized as a paint ball range by community children. The Cranberry Bog wetland is used as a play area by local children. Activities reported and observed as occurring include fishing, catching frogs and insects and playing games (such as capture the flag or hide-and-go-seek). A bridge has been built to connect the eastern and western sides of the wetland to allow greater access by individuals utilizing this area recreationally. The community performs cleanup of these areas on a regular basis, which includes trash removal in the interior wetland areas. These activities may all result in contact with the wetland media. The 78 day/year-exposure frequency for the Wells G&H wetland area is for future exposures. It is likely that children and adults would visit this area more frequently than 78 days per year. In fact, residents have stated to EPA that they currently go to this area nearly every day. The 78 days/year-exposure frequency is intended to provide a reasonable maximum estimate of the number of days of sediment and surface water contact per year for future site use in the Wells G&H wetland area. The total number of visits per year, which may include visits without sediment and surface water contact, is acknowledged as likely to exceed 78 days per year.

p. 3-30 2nd ¶. The RME sediment ingestion rate of 100 mg/day for an adult and 200 mg/day for a child is particularly conservative. These values are based on 1994 Region I Guidance. However, EPA's 1997 Exposure Factors Handbook recommends soil ingestion rates of 100 mg/day for a child and 50 mg/day for an adult (USEPA, 1997). Use of these soil ingestion rates would decrease risks by a factor of two.

EPA Response: The child and adult soil ingestion values recommended in the 1997 Exposure Factors Handbook represent reasonable central estimate values and are appropriate for use in a central tendency evaluation. The Exposure Factors Handbook does not recommend upper percentile values for use with a reasonable maximum scenario. Therefore, EPA Region I values, recommended for use

in a reasonable maximum scenario, were selected for use. These values are consistent with ingestion rates recommended by MADEP and, as stated in the Exposure Factors Handbook, are within the range of ingestion estimates from published studies. The central tendency ingestion rates utilized are the same as those recommended in the Exposure Factor-s Handbook for use in a central tendency evaluation.

p. 3-30 3rd ¶. The adult exposure frequency ranges from 26 to 104 days/year, depending on the station. EPA states that due to the presence of shallow surface waters, wading is likely to be the primary recreational activity at stations along the river (p. 3-25). However, the frequency with which a receptor might go *wading* and contact and ingest sediment is much less than the frequency with which a receptor might *visit* an exposure area like the Cranberry Bog, to walk their dog, for example. We believe that the exposure frequencies used by EPA are too high, because they reflect a high-end estimate of the number of potential *visits* per year, rather than the potential number of *wading and ingestion events* per year. The Wells G&H wetland and the Cranberry Bog are unattractive areas for wading because access to the sediment is through dense vegetation including vines and brambles, the wetlands are filled with tall reeds, the sediment is soft, and these areas have mosquitoes during the summer months. At the Cranberry Bog, a 6-ft tall adult was waist-deep in brush to access the sediment; and once in the sediment, he stood in 10-ft tall reeds. Thus it is unreasonable to assume that a 1-7 year old child would contact sediment in the bog on a regular basis. It is also unreasonable to assume that wading and sediment contact activity would occur with a frequency as high as 4 days/week for 6 months/year at the Cranberry Bog, or 3 days/week for 6 months/year at the proposed future nature trail areas (NT-1, NT-2, NT-3).

EPA Response: As stated previously, the Wells G&H wetland and Cranberry Bog are areas that are utilized by the surrounding neighborhoods and the community as a whole. The Wells G&H wetland may become more highly utilized in the future should the City of Woburn decide to develop this area as recreational space. Activities reported or observed as occurring (e.g., fishing, game playing, and frog catching) result in contact with the wetland media. Sampling locations determined as inaccessible to humans (e.g., in areas of soft sediment, dense vegetation, water greater than 2 feet) were not quantitatively evaluated. The exposure frequencies utilized in the human health risk assessment are sufficiently protective of current and potential future land use without being unrealistically conservative. The number of visits per year, including those without sediment and surface water contact, is acknowledged as likely exceeding the current and future exposure frequencies used in the risk calculations.

3.3.2.2 Exposure Parameters

p. 3-32. EPA used an arsenic dermal absorption fraction of 3%, the default value recommended by EPA (USEPA, 2001a). This value is based on a study by Wester *et al*. (1993), where estimates of arsenic absorption ranged from 3.2 to 4.5 percent *in vivo* in monkeys. Various factors affect the efficiency of dermal absorption, and thus there is considerable uncertainty associated with this value. However, the 3% value is likely to overestimate arsenic absorption and thus overestimate risks for the following reasons:

- Wester *et al*. used a soluble form of arsenic (sodium arsenate) mixed with soil. However, the forms of arsenic found in sediment are likely to be relatively insoluble, since the arsenic has been present for decades, and the sediment is in contact with surface water.
- Wester *et al*. added sodium arsenate to moist soil, and applied the mixture to the skin; thus the arsenic was not "aged." However, in the environment, metals tend to transform to less soluble forms in soil over time, and can also become sequestered in the pores of soil particles (Loehr, 1996).

- Wester *et al*. applied soil to the abdominal skin of the animals for 24 hours, whereas a child receptor along the river might only be exposed to sediment *via* wading for a short period of time. Specifically, "…studies with 24-hour (or longer) exposure periods are likely to overestimate the degree of dermal absorption that would occur under typical human exposure conditions" (NEPI, 2000). The absorption of any material is time-dependent. To the extent that an individual washes his skin more often than once every 24 hours, the uptake will be reduced. Washing may remove any soil residues adhering to the skin before absorption can occur to the same extent as in the animal study.
- In the Wester study, no urinary arsenic measurements were collected within the first 24 hours; therefore, it is not possible to estimate the amount of arsenic absorbed in periods less than 24 hours. After 7 days, a total of 3.2% of the arsenic was absorbed from the soil high dose. After one day (*i.e.*, in the first 24 hours), a total of 1.2% of the arsenic was absorbed from the soil high dose. Thus, about 40% (1.2% ÷ 3.2%) of the total absorption from soil occurred in the first 24 hours. A child playing in sediment would be exposed to arsenic in sediment for less than 24 hours. Thus it is reasonable to assume that the child's dermal absorption of arsenic from sediment, even assuming an extremely conservative exposure period on the order of 2 hours, would be no more than 1.2%. For this reason, use of a dermal absorption value of 3% is conservative and would overestimate the amount absorbed and thus overestimate risk *via* the dermal contact pathway.

EPA should point out in the uncertainty section that use of a dermal absorption value of 3% overestimates the amount absorbed, possibly by a factor of two or more, and thus overestimates risk *via* the dermal contact pathway.

EPA Response: The uncertainty section will be modified to include information related to the dermal absorption of arsenic, specifically that the dermal risk may be overestimated for this compound. Without site-specific information, it is impossible to quantify the degree of overestimation.

p. 3-32. EPA's assumption is that 50% of the fish consumed is obtained from the study area. EPA should support this assumption by providing data on the productivity of this river, types of food fish in the river, and whether the fish populations can support this rate of consumption. Support for the consumption rate is cited from EPA's Exposure Factors Handbook which states that approximately half of the total fish consumed in fishing households is obtained from recreational activities (USEPA, 1997). The implication is that 50% of the fish consumed comes from recreational activities, and that 100% of the recreational fishing occurs in the study area. The assumption that 100% of the recreational fishing for 24 years occurs in the same river is highly implausible. In reality, individuals are likely to fish in different locations over the span of 24 years as conditions in this and other fishing locations change over time.

EPA Response: The fish ingestion rates utilized (5 g/day and 13 g/day) are applicable to adults engaging in recreational fishing in the New England area. The additional information from the Exposure Factors Handbook provides data with which to adjust the adult ingestion rates so as to be appropriate for a younger age group (Table 10-61). This table also provides information on the relative ingestion of store-bought and recreationally-caught fish. However, since the ingestion rates utilized are for recreationally-caught fish, the information on store-bought vs. recreationally-caught fish ingestion rate was not used. Instead, the 50% value was selected to account for the fact that it is unlikely that 100% of the recreational fish consumed by a receptor would be caught in the same river.

3.4 Toxicity Assessment

p. 3-37. EPA's adjustments to the surface water RfD for manganese to account for dietary intake of manganese are overly conservative. Manganese presents a unique problem in that the level required for physiologic functioning is only slightly lower than the level where neurological effects are seen. Therefore, IRIS recommends taking into consideration dietary contributions of manganese when "using the reference dose to determine acceptable concentrations of manganese in water and soils" and suggests using a modifying factor of 3 for drinking water (IRIS, 1996). The IRIS modifying factor of 3 for drinking water also considers neonatal exposures. The IRIS RfD, without modification, is 0.14 mg/kg-day.

USEPA Region I guidance differs from the IRIS guidance. For drinking water exposures, USEPA Region I guidance advises adjusting the IRIS RfD to account for dietary intake (a 2-fold-reduction) and to account for neonatal exposures (a 3-fold reduction) (USEPA, 1996). This 6-fold reduction of the IRIS RfD results in a Region I RfD for drinking water of 0.024 mg/kg-day.

In this risk assessment, EPA has adjusted the manganese RfD for surface water according to the Region I guidance for drinking water. This is overly conservative, because surface water from the Aberjona river is not used as a drinking water source. Furthermore, neonatal exposures are not expected under the recreational exposure scenarios that EPA evaluated. For surface water, the IRIS recommendation of a 3-fold reduction of the RfD is more appropriate and still takes into account dietary intake and neonate exposures.

EPA Response: For the revised Wells G&H OU-3 Risk Assessment and the risk assessment to be conducted for the Aberjona River north of Route 128, the manganese RfD for the surface water pathway will be changed from 0.024 mg/kg-day to 0.07 mg/kg-day, which will reflect the removal of the 3-fold adjustment to the IRIS RfD.

p. 3-38. It is unreasonable to assume that chromium in surface water exists entirely as Cr(VI) when EPA's data show that most of the chromium in these sediments (where the chromium in surface water originates) exists as Cr(III).

EPA Response: Chromium may be present in surface water as a result of overland transport. The assumption that chromium in surface water exists as Cr(IV) accounts for the lack of medium-specific speciation data.

3.4.4 Toxicity Information for Arsenic in Sediment

p. 3-40. 1st ¶, last sentence. Oral bioavailability information is provided in Appendix C.8, but Table C.8-1 should be referenced to allow the reader to easily find the information.

EPA Response: As stated, the oral bioavailability values can be found on Tables 3-5.1 and 3-6.1.

p. 3-41. EPA describes the two bioavailability values derived from the swine study as a range of best estimate bioavailability values. EPA should state that these values represent the mean bioavailability values for two different sediment types.

EPA Response: The text of the uncertainty section will be modified to clarify this point.

p. 3-41. For the equation, the second RfD term should have the subscript "IRIS", not "IRIA".

EPA Response: It is acknowledged that this subscript should correctly read "IRIS".

3.4.5 Toxicity of Lead

p. 3-42, 1st ¶, 6th sentence. Blood lead levels are reported in units of micrograms per deciliter (µg/dL). Change 10 mg/dL [milligrams per deciliter] to 10 µg/dL. This sentence does not make sense in this context. The model was used to calculate a blood lead level, not a soil lead concentration.

EPA Response: The unit will be corrected. It is acknowledged that the model is used to determine whether or not exposures to a soil lead concentration will result in the exceedance of a childhood blood lead level goal.

p. 3-42, 2nd ¶, 3rd sentence. Change 10 mg/dL to 10 µg/dL. This sentence does not make sense in this context. The model was used to calculate a blood lead level, not a soil lead concentration.

EPA Response: The unit will be corrected. It is acknowledged that the model is used to determine whether or not exposures to a soil lead concentration will result in the exceedance of a 95th percentile fetal blood lead level goal.

p. 3-42, 4th and 5th sentence. Change mg/dL to µg/dL.

EPA Response: The unit will be corrected.

3.5.2.2 Description of ILCR Estimates

p. 3-48. It should be noted that the risk and hazard index estimates would decrease if a lower, more reasonable exposure frequency were used, particularly for stations in the Wells G&H wetland, which is an undesirable area for wading. Chapter 3 of this report presents revised risk calculations that show the effect of using a lower and more realistic exposure frequency.

EPA Response: The exposure frequencies are adequately protective and represent reasonable maximum and central tendency estimates, based on known information on current land use and anticipated future land use.

p. 3-50, 5th and 6th sentence. Change mg/dL to µg/dL.

EPA Response: The unit will be corrected.

3.5.3 Description of Uncertainties

p. 3-51. The uncertainty analysis states that uncertainty exists for certain parameters, but does not note the steps that were taken to address the uncertainties in the risk assessment. In a conservative risk assessment such as this, many of the assumptions are biased towards overestimating potential health risks. The impact of these conservative assumptions on the uncertainty in the calculated risks should be explained.

EPA Response: The uncertainty section will be expanded to include additional notations as to those assumptions that tend to overestimate or underestimate risk.

p. 3-52, 2nd ¶. EPA states: "Conversely, the biodegradation of chemicals to more toxic chemicals was also not considered." The discussion of the biodegradation of chemicals should be clarified. There are two types of biodegradation that could be the subject matter here. Metabolism or biodegradation within the human body (and potential conversion of chemicals to more toxic metabolites) is accounted for in the studies that support the RfDs and CSFs. Biodegradation in the environment, prior to human exposure, is not accounted for in the toxicity values. However, most environmental processes transform chemicals towards less reactive, less toxic forms (*e.g.*, oxidation of double bonds, dechlorination, binding in complexes, *etc*.). Thus, although biodegradation of COPCs in the environment is not factored into the risk assessment, it is not likely to result in an underestimate of potential health risks.

EPA Response: This section will be clarified to state that environmental biodegredation was not considered. Since some compounds can be converted to more toxic forms as a result of environmental conditions (for example, inorganic mercury into organic forms of mercury), this uncertainty may result in either an underestimation or overestimation of risk.

p. 3-52, 3rd ¶, last sentence. EPA states that "…it is not expected that actual risks will be significantly greater than estimated risks". In fact, due to the extremely conservative screening approach employed (comparing maximum detected concentrations to screening values based on residential exposures), the added contribution of chemicals that were eliminated as COPCs would be negligible. The word "significantly" should be deleted.

EPA Response: This section discusses the impact of eliminating chemicals from further quantitative evaluation (i.e., COPC selection). Since the elimination of some chemicals from further quantitative evaluation will result in an underestimate of risk, the word "significantly" should remain to provide information to the reader that the underestimation is not expected to be very great.

p. 3-53. Section 3.5.3.3, Toxicological Data. This section should provide greater detail on the uncertainty and conservatism in the toxicity factor for arsenic, because arsenic is the major risk driver at this site. Appendix B to this report presents a discussion of arsenic toxicity.

EPA Response: The discussion in the uncertainty section relative to the arsenic toxicity factor will be expanded.

p. 3-53., 2nd sentence. This sentence: "For the study area, there is a probability of overestimating health risks or hazards for a number of reasons…" does not appear to belong in the section on "Toxicological Data". This sentence should be moved to the first paragraph on p. 3-51.

EPA Response: The sentence will be moved as suggested.

p. 3-53. 2nd ¶. EPA states that "one of the major contributors to uncertainty is the accuracy of the toxicity values used." EPA gives several assumptions used in the dose-response model for carcinogens, and states that "to the extent that any of these assumptions are incorrect, the extrapolated risks may be over- or under-estimates." However, EPA should note that, in the derivation of toxicity values, conservative assumptions are made to account for these uncertainties, and thus the values tend to be biased towards overestimating risk. For example, humans are considered to be as sensitive as the most sensitive test species. In the case of arsenic, the major risk driver in this- assessment, the toxicity factor is, as discussed in Appendix B, very conservative as applied to U.S. populations.

EPA Response: This section of the uncertainty section will be expanded as suggested.

p. 3-53, 3rd ¶. The toxicity factors are conservative and contain uncertainty factors. Appendix B to this report discusses toxicological uncertainties for arsenic.

EPA Response: The discussion in the uncertainty section relative to the arsenic toxicity factor will be expanded.

p. 3-54, 3rd ¶. The sixth sentence should be revised to: "The assumption that RME receptors obtain 100% of their self-caught dietary fish intake from the Aberjona River was also conservative."

EPA Response: The sixth sentence will be removed since it does not accurately reflect the approach utilized in the HHRA.

p. 3-54. The EPC uncertainty section should make the following points:

The EPC (and hence the risk) in the WH exposure area is heavily influenced by the samples that EPA chose to include in this exposure area. EPA selected the boundary of the WH exposure area, presumably based on professional judgment. However, EPA has not demonstrated that all of the sample locations they included in the WH exposure area are uniformly accessible. The arsenic EPC of 1900 mg/kg for the WH exposure area is heavily influenced by EPA's inclusion of one sample with a very high arsenic concentration (SD-12-01-ME). The WH samples (WH-01 to WH-10) included in the WH exposure area range from 4.7 to 424 mg/kg, and have an average arsenic concentration of 114 mg/kg. However, the last sample included in the WH exposure area, SD-12-01-ME, has a concentration of 3230 mg/kg, which is an order of magnitude higher in concentration than the next highest WH sample. This sample is the southernmost sample within this exposure area (Figure 1). Including this sample yields an EPC for station WH that is potentially biased high. If EPA did not include sample SD-12-01-ME in the WH exposure area, the EPC at WH would be 663 mg/kg[4], and risks at WH would decrease by a factor of 3. Thus, the inclusion of this one sample tends to overestimate the risk for the entire WH exposure area.

At station WH, the EPC of 1900 mg/kg is the 99% Chebyshev minimum variance unbiased estimate (MVUE) UCL. Table 3-3.2 should note that the EPC for WH is the 99%UCL, not the 95%UCL, and EPA should provide the statistical rationale for using the 99% Chebyshev UCL as the EPC, as described in their 2002 guidance. EPA's use of the 99% Chebyshev UCL indicates that this dataset is highly skewed due to the inclusion of sample SD-12-01-ME. The skewed dataset strongly suggests that this exposure area is not well delineated, and thus that this dataset may be inappropriate for use in risk management decisions.

EPA has not provided sufficient information in their UCL guidance (USEPA, 2002) or ProUCL manual (USEPA, 2003) to assess the validity of their choice of the 99% Chebyshev UCL as better than other possible methods. EPA should provide its underlying analyses that led to the UCL recommendations so that experts in the community can review and refine if appropriate. For example, Saranko and Tolson (2003) (provided in Appendix C) show that the UCL of data sets with statistical characteristics similar to the WH dataset may be better estimated with alternative methods that give rise to lower UCL values. Their analysis suggests that EPA's method may have overestimated the EPC, and therefore the risk, for the WH dataset.

At Station CB-03, on the western side of the Cranberry Bog, EPA used an exposure point concentration (EPC) that is equal to the maximum concentration (1410 mg/kg) detected in this exposure area, because the calculated 95%UCL exceeded the maximum. EPA's use of the maximum concentration as the EPC indicates that the CB-03 area has too few samples to be well characterized. The sediment concentrations

[4] The EPC of 663 mg/kg was obtained from the ProUCL program, and is the 99% Chebyshev (MVUE) UCL.

in 11 of the 12 CB-03 samples are much lower, ranging from 9.1 to 510 mg/kg, and the average concentration of all 12 CB-03 samples is only 272 mg/kg. Based on this dataset, if a person visited each sample location with equal frequency, then on average, he or she would be exposed to an average concentration that is much lower than the EPC of 1410 mg/kg. Therefore, using an EPC equal to the maximum concentration of 1410 mg/kg in all likelihood overestimates the risks for CB-03.

At station 13/TT-27, on the west side of the Wells G&H wetland, EPA used an EPC that is equal to the maximum concentration (4210 mg/kg) detected in this exposure area, because the calculated 95%UCL exceeded the maximum. EPA's use of the maximum concentration as the EPC indicates that the 13/TT-27 area has too few samples to be well characterized. Seven of the nine samples used to characterize this area have arsenic concentrations ranging from 12 to 356 mg/kg, but the last two samples have concentrations of 2480 and 4210 mg/kg, respectively. The average concentration of all samples is 840 mg/kg. Based on this dataset, if a person visited each sample location with equal frequency, then, on average, he or she would be exposed to an average concentration that is much lower than 4210 mg/kg. Therefore, using an EPC equal to the maximum concentration of 4210 mg/kg likely overestimates the risks for 13/TT-27.

EPA Response: The discussion of uncertainties associated with the EPCs will be expanded as appropriate. As previously discussed, it is acknowledged that the arsenic EPC used at a small number of stations are uncertain due to one or a small number of arsenic results that were elevated in comparison to the remainder of the data set. This uncertainty will be added to the risk assessment, with the locations of the highest arsenic levels identified.

5.1.6 Baseline Human Health Risk Assessment

p. 5-9, 2nd ¶. The EPC is the 95% upper confidence limit <u>on the mean concentration</u>.

EPA Response: The clarification will be added.

p. 5-10, last sentence. Change "are:" to "are arsenic and benzo(a)pyrene."

EPA Response: The text will remain unchanged so as to provide information on the locations where arsenic and benzo(a)pyrene are driving risk.

APPENDIX C-3 Human Health Reference Calculations

Table C.3-2.1. The AWQC for arsenic should not be considered for COPC screening. The AWQC was derived using a toxicity value for inorganic arsenic. However, the majority of arsenic in fish exists as arseno-sugars (*e.g.*, arsenobetaine, arsenocholine). The fraction of inorganic arsenic in freshwater fish has been reported to be less than 10% (Schoof *et al.*, 1999). The arseno-sugars are essentially non-toxic because they are excreted unmetabolized in a relatively short time. EPA is currently revising the AWQC for arsenic based on this information (*Fed. Reg.* Oct. 12, 2000).

EPA Response: As stated above, the BRA is not intended to identify ARARs. ARAR identification will ocurr in the Feasibility Study. The uncertainty section will discuss that the arsenic AWQC is currently under review by EPA.

Tables C.3-3.1, C.3-3.2, C.3-3.3. Arsenic, lead and mercury concentrations in wetland surface water are below their respective MCLs, meaning this water meets drinking water standards.

EPA Response: These compounds have been selected as surface water COPCs based on a comparison of maximum detected concentrations in all background surface water samples combined to risk-based preliminary remediation goals and AWQCs. Average or 95% UCL concentrations may not exceed these screening criteria. However, the compounds selected as COPCs are carried forward for quantitative risk evaluation.

Table C.3-5. Regarding the primary target organ column, bis-2-ethylhexyl-phthalate is also a reproductive toxin in animals (only the liver is mentioned) and inorganic mercury is better known as a nephrotoxin than an immunotoxin. The primary target organ should not be listed as "NOAEL" for chromium (VI) and vanadium. The kidney is the primary target organ for chromium *via* oral exposure. The target organ for vanadium by oral exposure could be listed as "not known".

EPA Response: The target organ for bis(2-ethylhexyl)phthalate will remain as liver, since treated offspring also displayed liver effects. Since the RfD for inorganic mercury is based on autoimmune effects, the target organ for this compound will also remain unchanged. After additional research, the target organs for chromium (VI) and vanadium will be listed as GI system and kidney, respectively. This information will be used in the revised Wells G&H OU-3 Risk Assessment and the risk assessment for the Aberjona River north of Route 128.

Table C.3-6. It should be stated in the notes that cadmium and chromium (VI) are recognized as carcinogens by the inhalation route of exposure but do not appear to be oral or dermal route carcinogens (IRIS, 2003).

EPA Response: Since Table C.3-6 lists information related to the toxicity of compounds via the oral route of exposure, it is not necessary to add information on the inhalation route of exposure.

APPENDIX C-5 Derivation of Allowable Daily Intake

General. Several calculations of allowable daily intake result in improbable values; either soil concentrations greater than 1 million mg/kg (*i.e.*, more than 100%) or fish tissue concentrations that are biologically implausible (*e.g.*, a fish composed of 10% magnesium). EPA should not use solutions that are not possible in real life. One million mg/kg should be used as the maximum soil concentration. A nominal cutoff value (*e.g.*, 1% or 10,000 mg/kg) should be used as the value in edible fish tissue when very high risk-based values are calculated.

EPA Response: Comment noted. These calculations have been presented to demonstrate that essential nutrients may be removed from further quantitative risk evaluation. The calculations will be reviewed further and changed, if necessary.

p. C.5-1. The FDA Daily Recommended Value (DRV) for sodium is 2,400 mg/day. The soil value of 1,000,000 mg/kg equates to a block of pure salt.

EPA Response: See response to general comment above.

p. C.5-2. The FDA Recommended Daily Intake (RDI) for calcium is 1,000 mg/day. EPA should indicate why a 10-fold reduction was not applied as was the case for sodium (presumably because excessive sodium intake is more of a health hazard than excessive calcium intake). The soil value of 4,000,000 mg/kg is greater than 100%. The fish value of 50,000 mg/kg would require that the edible fish tissue (*i.e.*, excluding bones and viscera) be 5 percent calcium, which is not possible.

EPA Response: See response to general comment above.

p. C.5-3. The FDA RDI for magnesium is 400 mg/day. The soil value of 8,050,000 mg/kg is greater than 100%. The fish value of 100,630 mg/kg would require that 10% of the edible fish tissue be pure magnesium, which is not possible.

EPA Response: See response to general comment above.

p. C.5-4. The FDA DRV for potassium is 3500 mg/day. The soil value of 1,000,000 mg/kg is equal to 100% potassium, which is not possible.

EPA Response: See response to general comment above.

APPENDIX C-8 Toxicity Profiles for COPCs

General.

- Although the inhalation route of exposure is not being evaluated in this risk assessment, this section contains information on the toxicity of compounds *via* the inhalation route. This should be eliminated as confusing to the reader.
- The discussion and citation of RfDs and CSFs is inconsistent between chemicals, *i.e.*, for some chemicals these values are provided and for others they are not.
- Because inhalation exposures are not being evaluated, discussion of RfCs should be eliminated.
- Bis-2-ethylhexyl-phthalate is a COPC in the risk calculation tables, but there is no discussion of this compound in Appendix C-8.

EPA Response: Information related to the inhalation route of exposure will be retained in Appendix C.8. This information is important in characterizing the overall toxicity of a COPC. The toxicity profiles will be updated and appended to the revised Wells G&H OU-3 Risk Assessment. Discussion and citations of RfDs and CSFs will be removed from the toxicity profiles since the toxicity values as well as information relative to them are provided in Tables 3-5.1 and 3-6.1. This information does not need to be repeated in the toxicity profiles. At that time, a toxicity profile will be added for bis(2-ethylhexyl)phthalate.

p. C.8-2. The term q1* is outdated terminology. The term CSF (cancer slope factor) is currently in use.

EPA Response: The toxicity profiles will be updated and appended to the revised Wells G&H OU-3 Risk Assessment.

p. C.8-5. IRIS lists the animal dose as 20 but the human equivalent concentration (HEC) as 14. These terms should be clarified.

EPA Response: This information, since it relates to the toxicity values and information provided in Tables 3-5.1 and 3-6.1, will be removed when the toxicity profiles are updated.

p. C.8-10. It should be explained that there is no RfD for the carcinogenic PAHs because either RfDs are given for individual compounds, or there is an explanation for why the RfD is lacking. Change "factor" to "factors".

EPA Response: This information will be added to the uncertainty section of the report since it contributes to the underestimation of risk.

p. C.8-24. EPA should provide the absorption of PCBs through the skin and GI tract in a more quantitative manner. EPA should note that chloracne (like non-chemical acne) is only disfiguring if it is severe.

EPA Response: No additional discussion of absorption is required. The dermal absorption of PCBs is quantitatively discussed in Section 3.3.2.2 (Exposure Parameters). Since the oral absorption efficiency of PCBs exceeds 50%, no further quantification is necessary (see Section 3.4.3 Adjustment of Toxicity Factors).

p. C.8-29. The statement in the toxicity profile that states that dermal absorption of arsenic is "not significant" contradicts the results in this risk assessment, in which dermal exposure to arsenic contributes 20-30% of the risk. In addition, arsenic is embryotoxic, fetotoxic and teratogenic only at doses and in some cases *via* routes which are inconsistent with plausible human exposures. See DeSesso in Teratology 2001 64(3):170-3.

EPA Response: This information will be added to the discussion on arsenic toxicity, to be incorporated into the uncertainty section.

p. C.8-31. The acutely toxic dose noted for barium should be put in units of mg (800 mg not 0.8 g) to make it more easily comparable to the other doses and the RfD which are expressed in mg.

EPA Response: Comment noted.

p. C.8-32. The RfC for barium is not relevant to this assessment as inhalation route exposures are not being evaluated.

EPA Response: As noted above, information related to the inhalation route of exposure will be retained in the profiles.

p. C.8-34. According to IRIS, the chronic oral RfD for chromium (VI) is 3E-3 mg/kg-day not 5E-3 mg/kg-day as stated in the text. According to IRIS, the chronic oral RfD for chromium (III) is 1.5 mg/kg-day not 1 mg/kg-day as stated in the text. The correct IRIS RfD for chromium (VI) was used in the calculations so this correction only affects the text, not the risk estimates.

EPA Response: As noted above, when the toxicity profiles are updated, discussion and citations of RfDs and CSFs will be removed. The toxicity values as well as information relative to them are provided in Tables 3-5.1 and 3-6.1.

p. C.8-34. EPA should state explicitly that chromium (VI) has not been shown to be a carcinogen by the oral route of exposure. While it is true, as stated, that ingested chromium VI is listed by USEPA as not classifiable as to carcinogenicity, the available data indicate a lack of tumorigenicity after oral chromium (VI) exposure. For example, a recent expert review panel report commissioned by the State of California indicated that chromium (VI) was not likely to be carcinogenic by the oral route of exposure (California Chromate Toxicity Review Committee, 2001). See also Proctor *et al.* (2002), and Zhang and Li (1997).

EPA Response: The language from IRIS will continue to be used in the profile ("Carcinogenicity by the oral route of exposure can not be determined and is classified as Group D").

p. C.8-36. The discussion of copper hydroxyquinoline should be deleted because an organic copper compound like copper hydroxyquinoline is not relevant to an environmental copper exposure.

EPA Response: The discussion of copper hydroxyquinoline will remain in the profile. Little data are available to assess the carcinogenic potency of copper compounds. The data from the organic copper compound is stated as uncertain with unknown relevance. However, it represents the body of information that was examined in order to arrive at "inadequate" classification.

p. C.8-37. No RfD is identified for copper in this section although a value of 4E-2 mg/kg-day is used in the risk calculations. There is currently no RfD for copper listed on IRIS, although an MCL exists which is often used to derive an RfD. EPA should document the basis for the 4E-2 mg/kg-day value in the text.

EPA Response: The provisional RfD, provided by NCEA, is back-calculated from the MCLG of 1.3 mg/L.

p. C.8-39. The RfD for cyanide (2E-2 mg/kg-day) should not be included in this section, as cyanide is not a COPC for this risk assessment.

EPA Response: The cyanide toxicity profile will be removed when the toxicity profiles are updated.

p. C.8-42. Although EPA has classified lead and lead compounds as a Group B2 probable human carcinogen, EPA has stated that the carcinogenic potency of lead appears weak and that risk management decisions based on lead's neurodevelopmental effects should be adequate to address possible carcinogenic effects. The statement should be made that it is EPA policy to evaluate lead for neurodevelopmental effects and not carcinogenicity. As written, it implies that carcinogenicity is not evaluated simply because a value is not available.

EPA Response: When the toxicity profiles are updated, this point will be clarified as noted.

p. C.8-49. The discussion of the toxicity of nickel carbonyl should be deleted. This compound has a toxicity quite different from inorganic nickel, is an occupational chemical, and is not found at the site. The same applies to nickel subsulfide.

EPA Response: Since these nickel compounds give an indication of the overall potential toxicity of nickel, this information will be retained in the toxicity profile.

p. C.8-51. The first two sentences of the selenium discussion are out of place and add no significant information to the discussion. They should be deleted and the section should start with the next paragraph.

EPA Response: The profile will be modified when the toxicity profiles are updated.

p. C.8-58. Some explanatory text should accompany Table C.8-1 because toxicity, not oral bioavailability, is the primary topic of the preceding 57 pages. The reason why other compounds (*e.g.*, PCBs, lead, organic mercury, *etc.*) are not listed should also be noted. Finally, the special case of the site-specific oral bioavailability of arsenic in sediment should be discussed.

EPA Response: The purpose of this table is presented in Section 3.4.3 and on Tables 3-5.1 and 3-6.1.

APPENDIX C-9 Relative Bioavailability of Arsenic in Sediments from the Aberjona River

p. 3. EPA should provide more information on where the sediment samples were collected. For example, the location in the streambed and the depth of the overlying water column should be provided.

EPA Response: This information will be provided in the Comprehensive Remedial Investigation Report being prepared by TetraTech NUS in support of the Aberjona River Study Area north and south of Route 128. All samples were collected along the edge of the stream or wetland, in areas considered accessible to humans (e.g., below less than 2 feet of standing water). The following correlates the bioavailability sampling locations to historical sampling locations:

Bioavailability Sample	***Historical Location***
1	***HB01-08***
2	***HB02-04***
3	***HB03-08***
4	***SD12-01-ME***
5	***WG-07***
6	***WS-08***
7	***CB03-06***
8	***CB03-11***
9	***CB03-09***
10	***SD07-10-FW***
11	***SD07-04-FW***
12	***SD07-05-ME***

p. 17. This section should note whether any data were excluded from the analysis.

EPA Response: Test Material (TM) 3 was excluded from the in vivo portion of the study because the arsenic levels were judged not to be high enough to yield measurable results in swine. However, TM3 was used in the in vitro portion of the study.

Figure 4-2. The data suggest two groups of results at high arsenic doses, one following linear dose-elimination pattern and one following a sublinear pattern. EPA should state whether the data following the apparent sublinear pattern represent a subgroup of animals, or if this is simply random variability in the data.

EPA Response: Figure 4-2 presents results for all animals receiving TM1, followed over time from 6 to 11 days after the beginning of dosing. Random variability in the data is shown.

Figure 5-1. The RBA values in this figure appear to be approximately 54% and 43%. What do these values represent? Their arsenic concentrations seem to match sediment samples TM1 and TM2. However the RBA estimates provided for TM1 and TM2 on page 17 are 37% and 51%. EPA should add error bars to this figure so that the apparent dose-effect on RBA can be more clearly evaluated by the reader.

EPA Response: The values plotted for RBA represent the upperbound of the 90% confidence interval. Since this figure is simply provided to show the inverse correlation between RBA and sediment concentration, no further clarification is necessary.

Table B-3. The footnotes indicate that some pigs ate only part of their dose. An estimate of the amount of the dose consumed is noted. However, if the soil is not homogeneous within the doughball but rather located in the center, EPA should explain whether it is possible to accurately estimate the amount of soil not consumed from the amount of dough not eaten.

EPA Response: Since the amount of dough not consumed tended to be minimal (typically between 5% and 10%), it is unlikely that this type of estimation will have a significant impact on the determination of RBA.

3 Revised Risk Calculations

3.1 Revised Deterministic Risk Calculations

EPA's risk analysis overestimates risks due to a number of overly conservative exposure assumptions, including high-end estimates for exposure frequency and soil ingestion rate. EPA's assumptions for exposure frequencies, which are based on professional judgment, are especially troubling. For example, it is highly implausible that any individual, starting at age 1, would wade in the maximum concentration sediment at CB-03 for 4 days/week, 6 months/year, and 30 years. This section presents recalculated cancer and noncancer risks for WH, NT-1, NT-2, CB-03, and 13/TT-27, to show the impact of more plausible (yet still conservative) estimates for exposure frequency and soil ingestion rate. The changes to exposure frequency and ingestion rate are still conservative, but yield a more realistic estimation of risk.

Exposure frequencies were modified in the following manner:

- For current and future risks from exposure to sediment at the west side of the Cranberry Bog (CB-03), we assumed a sediment exposure frequency of 1 day/month for 6 months/year (6 days/year). The cranberry bog has little desirability as a wading area, because it is densely filled with reeds, it is accessible only by walking through dense vegetation that includes vines and brambles, it has mosquitoes present during the summer, and it shows little evidence that humans use this area on a frequent basis.
- For current risks from exposure to sediment at WH, we used an exposure frequency of 4 days/year. Like the Cranberry Bog, the Wells G&H wetland has little desirability as a wading area, because it is densely filled with reeds, it is surrounded by dense vegetation including vines and brambles, and it has mosquitoes present during the summer. For current exposures at WH, we used a lower exposure frequency than for the Cranberry Bog, because this wetland is currently even harder to access than the Cranberry Bog.
- For future risks from exposure to sediment at the stations in the Wells G&H wetland (WH, NT-1, NT-2, NT-3, and 13/TT-27), we used an exposure frequency of 1 day/month for 6 months/year (6 days/year), the same as that used for the Cranberry Bog. Future redevelopment may make accessibility to this wetland approximately equal to that of the Cranberry Bog, and this wetland is considered as undesirable for wading as the Cranberry Bog.

The soil ingestion rate was decreased to 100 mg/day for a child and 50 mg/day for an adult, using the recommended soil ingestion rates in EPA's Exposure Factors Handbook (USEPA, 1997). All other parameters were kept the same, including the EPCs, the 50% fraction from site, and the exposure duration.

Table 1 presents revised risks to illustrate the impact of two modest changes in exposure parameters. All revised risks are within EPA's acceptable levels for both cancer and noncancer risks. The current RME cancer risks at WH and CB-03 decrease to $8x10^{-6}$ and $9x10^{-6}$, respectively. The current RME noncancer risks at WH and CB-03 both decrease to 0.2. The future RME cancer risks at WH, NT-1, NT-2, and NT-3 decrease to between $4x10^{-6}$ and $2x10^{-5}$. The future cancer risk decreases to $9x10^{-6}$ at CB-03, and to $3x10^{-5}$ at 13/TT-27. The future RME noncancer risks decrease to 0.3 at WH and NT-1, 0.1 at NT-2 and NT-3, 0.2 at CB-03, and 0.4 at 13/TT-27. All risks are within EPA's acceptable exposure limits. Note that

although we used an exposure frequency of 4 days/year for current risk, and 6 days/year for future risk, at stations in the Wells G&H wetland (WH, NT-1, NT-2, NT-3, and 13/TT-27), the exposure frequency could be as high as 15 days/year and risks would still fall within EPA's acceptable risk limits. The exposure frequency at CB-03 in the Cranberry Bog could be as high as 35 days/year and risks would still fall within EPA's acceptable risk limits.

Only two exposure parameters were modified for the revised risks presented in Table 1. However, other parameters could be modified that would reduce risks even further, such as use of a lower dermal absorption for arsenic, or use of a subchronic RfD for arsenic. Dermal contact accounts for 20-30% of the total risk for both cancer and non-cancer and is thus a significant contribution to risk. EPA used a dermal absorption of 3%. Using the results of the Wester study (see Section 3.3.2.2) it is reasonable to assume that a child's dermal absorption of arsenic from sediment, even assuming an extremely conservative exposure period on the order of 2 hours, would be no more than 1.2%. Thus the dermal absorption value of 3% is conservative and tends to overestimate the amount absorbed and overestimate risk *via* the dermal contact pathway. We note, in addition, that there is no literature to indicate that dermal contact with arsenic in sediment or soil causes cancer or any other health effects.

A subchronic RfD is appropriate for evaluation of exposures that are less than 10% of a lifetime. Therefore, it is appropriate to use a subchronic RfD to evaluate noncancer risks for the 1-6 year old child. USEPA Region 8 has derived an oral RfD for arsenic of 0.015 mg/kg-day that addresses both acute and subchronic exposures (USEPA, Region 8, 2001). This value is 50 times higher than the chronic RfD that EPA used for arsenic (0.0003 mg/kg-day). According to Region 8, the subchronic RfD is appropriate to quantify non-cancer health risks from subchronic exposures to inorganic arsenic lasting 15 days to 7 years (USEPA, Region 8, 2001). If the Region 8 subchronic arsenic RfD is used, the noncancer risks would be about 50 times lower than those presented in Table 1, since arsenic contributes more than 99% of the noncancer risks for these stations.

EPA Response: Exposure frequencies used in Gradient's deterministic risk calculations are not sufficiently protective of reasonable maximum exposures that are occurring or may occur in the future at these stations. These areas are currently utilized by the community at a higher frequency than would be accounted for by an exposure frequency of 4 to 6 days/year. Future plans to develop these areas into more attractive and more highly utilized recreational spaces would only serve to increase the frequency with which individuals visit the site and contact impacted media. The deterministic calculations performed by Gradient and provided in Chapter 3 of these comments have not been reviewed for accuracy since the exposure assumptions (i.e., exposure frequencies and soil ingestion rates) used are not sufficiently protective of current or potential future reasonable maximum exposures in these areas. In addition, the reference dose for arsenic used in these calculations is not appropriate for childhood exposures, even if occurring for less than 7 years in duration.

Table 1
EPA and Revised Risk Calculations

Current Cancer Risk
Exposure Factors Revised: Exposure Frequency, Soil Ingestion Rate

	EPA Current RME Risks		Revised Current RME Risks	
Station	**Exp Freq (d/yr)**	**Cancer Risk**	**Exp Freq (d/yr)**	**Cancer Risk**
WH	26	1E-04	4	8E-06
CB-03	104	**3E-04**	6	9E-06

Current Noncancer Risk
Exposure Factors Revised: Exposure Frequency, Soil Ingestion Rate

	EPA Current RME Risks		Revised Current RME Risks	
Station	**Exp Freq (d/yr)**	**Noncancer Risk**	**Exp Freq (d/yr)**	**Noncancer Risk**
WH	26	**2**	4	0.2
CB-03	104	**6**	6	0.2

Notes:
1. Values in bold exceed 1E-04 for cancer risks or 1 for noncancer risks.
2. Revised soil ingestion rates taken from USEPA Exposure Factors Handbook (1997), Table 4-25: Child: 100 mg/day, Adult: 50 mg/day.

(Continued)

Table 1
EPA and Revised Risk Calculations (cont'd)

Future Cancer Risk
Exposure Factors Revised: Exposure Frequency, Soil Ingestion Rate

	EPA Future RME Risks		Revised Future RME Risks	
Station	**Exp Freq (d/yr)**	**Cancer Risk**	**Exp Freq (d/yr)**	**Cancer Risk**
WH	78	**4E-04**	6	2E-05
NT-1	78	**5E-04**	6	2E-05
NT-2	78	**2E-04**	6	8E-06
NT-3	78	1E-04	6	4E-06
CB-03	104	**3E-04**	6	9E-06
13/TT-27	78	**8E-04**	6	3E-05

Future Noncancer Risk
Exposure Factors Revised: Exposure Frequency, Soil Ingestion Rate

	EPA Future RME Risks		Revised Future RME Risks	
Station	**Exp Freq (d/yr)**	**Noncancer Risk**	**Exp Freq (d/yr)**	**Noncancer Risk**
WH	78	**7**	6	0.3
NT-1	78	**8**	6	0.3
NT-2	78	**3**	6	0.1
NT-3	78	**2**	6	0.1
CB-03	104	**6**	6	0.2
13/TT-27	78	**10**	6	0.4

Notes:
1. Values in bold exceed 1E-04 for cancer risks or 1 for noncancer risks.
2. Revised soil ingestion rates taken from USEPA Exposure Factors Handbook (1997), Table 4-25: Child: 100 mg/day, Adult: 50 mg/day.

3.2 Probabilistic Risk Calculations

In order to assess the uncertainty associated with EPA's deterministic risk calculations, Gradient performed probabilistic risk calculations for the ingestion of arsenic in sediment at stations WH, NT-1, NT-2, NT-3, 13/TT-27, and CB-03. The probabilistic risk calculations are presented to help put EPA's risks into perspective, and because USEPA Region I "considers Monte Carlo analysis to be an acceptable approach for analyzing uncertainty in the risk assessment" (USEPA, 1994).

Probabilistic risk calculations use distributions for the input parameters instead of point estimates, to express the fact that a given exposure parameter may have a range of plausible values for different individuals. We used distribution inputs for five exposure parameters: exposure frequency, sediment ingestion rate, body weight, bioavailability, and fraction from site. For the purpose of this calculation, we used EPA's point estimates for the exposure point concentration (EPC), exposure duration, averaging time, and cancer slope factor. EPA guidance (USEPA, 2001b) states that the EPC should be a point estimate rather than an input distribution, and distributions are not available for the cancer slope factor.

The input distributions used for each exposure parameter are described in Table 2. We used a uniform distribution for bioavailability because we have a range for this parameter, but it is difficult, at this time, to identify any particular value as more likely than any other. Inputs for other distributions are based on literature values or professional judgment.

Table 3 presents the results of the probabilistic risk calculations. All of the 90th percentile cancer risks are at or below $2x10^{-5}$, and all of the 95th percentile cancer risks are at or below $3x10^{-5}$. The 95th percentile risk is $1x10^{-5}$ at both WH and CB-03, $2x10^{-5}$ at NT-1, $6x10^{-6}$ at NT-2, $4x10^{-6}$ at NT-3, and $3x10^{-5}$ at 13/TT-27. The 95th percentile risk is used here as an estimate of the RME, because EPA's Guidance for Probabilistic Risk Assessment states that "In human health PRA, a recommended starting point for risk management decisions regarding the RME is the 95th percentile of the risk distribution." (EPA, 2002; p. 7-4). The 95th percentile risk means that there is a 95% probability that the risk to any one individual will be below this value. The probabilistic noncancer hazard quotients are presented in Table 3. The 95th percentile noncancer hazards range from 0.07 to 0.95 and are all less than EPA's acceptable hazard of 1.0.

The probabilistic risks are substantially lower than EPA's individual risk estimates for the ingestion of arsenic in sediment (Table 1). Although the probabilistic risks are only for the ingestion of arsenic in sediment, this pathway represents a major portion (about 75%) of EPA's total cancer risks for these stations. This analysis indicates that EPA's RME risk, derived by using point estimates for all inputs, is a very high end value and hence is not representative of an RME value. Use of a more plausible range of exposure inputs results in risks falling within EPA's acceptable risk range of 10^{-6} to 10^{-4}. The probabilistic risks corroborate the revised deterministic risks (Table 1), in that both sets of risks do not exceed EPA's acceptable risk levels when more realistic inputs are used.

A sensitivity analysis was also performed as part of the probabilistic risk calculations. The results of the sensitivity analysis indicate that the calculated cancer risk is most sensitive to the Child exposure frequency, the Child fraction from site, and the Child sediment ingestion rate. These three parameters together account for 69% of the variability in the calculated cancer risk. This means that variation in the values used for these parameters has a large influence on the calculated risk. Thus, the sensitivity analysis highlights the fact that use of accurate and reasonable values for these parameters is critical to the overall confidence in the predicted risks.

EPA Response: The probabilistic calculations performed by Gradient and contained in Chapter 3 have not been reviewed for accuracy. Specific assumptions used in Gradient's probabilistic assessment are not sufficiently protective of current or potential future site conditions. Should it be determined that probabilistic information would be useful during the risk management process, an evaluation will be conducted at that time by EPA.

Table 2
Distributions for Exposure Parameters

Parameter	Assumed Distribution	Distribution Parameters: WH (Current)	Distribution Parameters: CB-03 (Current and Future)	Distribution Parameters: WH, NT-1, NT-2, NT-3, 13/TT-27 (Future)	Source
Exposure frequency	Lognormal	Range = 1-20 50th% = 4 95th% = 12	Range = 1-20 50th% = 6 95th% = 12	Range = 1-20 50th% = 6 95th% = 12	Professional judgment based on site visits.
Sediment ingestion rate	Lognormal	Child: Range = 1-300 50th% = 45 95th% = 124 Adult: Range = 1-300 50th% = 23 95th% = 100	Child: Range = 1-300 50th% = 45 95th% = 124 Adult: Range = 1-300 50th% = 23 95th% = 100	Child: Range = 1-300 50th% = 45 95th% = 124 Adult: Range = 1-300 50th% = 23 95th% = 100	Literature values, see Appendix A. Used half the child value for 50th percentile, and EPA value for 95th percentile.
Body weight	Normal	Child: Range = 11-19 Mean = 15 Stdev = 2 Adult: Range = 34-216 Mean = 70 Std Dev = 4	Child: Range = 11-19 Mean = 15 Stdev = 2 Adult: Range = 34-216 Mean = 70 Std Dev = 4	Child: Range = 11-19 Mean = 15 Stdev = 2 Adult: Range = 34-216 Mean = 70 Std Dev = 4	EPA Exposure Factors Handbook
Bioavailability of Arsenic in Sediment	Uniform	Min = 37% Max = 51%	Min = 37% Max = 51%	Min = 37% Max = 51%	EPA swine study
Fraction from site	Triangular	Min = 0% Max = 100% Most likely = 50%	Min = 0% Max = 100% Most likely = 50%	Min = 0% Max = 100% Most likely = 50%	Professional judgment.

Table 3
Probabilistic Cancer Risks
Current Risks from Ingestion of Arsenic in Sediment

Station	Arsenic EPC (mg/kg)	Probabilistic Cancer Risk 90th Percentile	Probabilistic Cancer Risk 95th Percentile	EPA Point Estimate Current RME Risk from Ingestion of Arsenic in Sediment
WH	1900	8.0E-06	1.1E-05	8.6E-05
CB-03	1400	7.6E-06	1.0E-05	2.6E-04

Probabilistic Noncancer Hazards
Current Risks from Ingestion of Arsenic in Sediment

Station	Arsenic EPC (mg/kg)	Probabilistic Noncancer Hazard 90th Percentile	Probabilistic Noncancer Hazard 95th Percentile	EPA Point Estimate Current RME Hazard from Ingestion of Arsenic in Sediment
WH	1900	0.15	0.21	1.5
CB-03	1400	0.14	0.19	4.5

(Continued)

Table 3 (continued)
Probabilistic Cancer Risks
Future Risks from Ingestion of Arsenic in Sediment

Station	Arsenic EPC (mg/kg)	Probabilistic Cancer Risk 90th Percentile	Probabilistic Cancer Risk 95th Percentile	EPA Point Estimate Future RME Risk from Ingestion of Arsenic in Sediment
WH	1900	1.0E-05	1.3E-05	2.6E-04
NT-1	2500	1.4E-05	1.8E-05	3.3E-04
NT-2	820	4.5E-06	5.9E-06	1.1E-04
NT-3	500	2.7E-06	3.5E-06	6.7E-05
13/TT-27	4200	2.3E-05	2.9E-05	5.7E-04
CB-03	1400	7.6E-06	1.0E-05	2.6E-04

Probabilistic Noncancer Hazards
Future Risks from Ingestion of Arsenic in Sediment

Station	Arsenic EPC (mg/kg)	Probabilistic Noncancer Hazard 90th Percentile	Probabilistic Noncancer Hazard 95th Percentile	EPA Point Estimate Future RME Hazard from Ingestion of Arsenic in Sediment
WH	1900	0.19	0.25	4.6
NT-1	2500	0.24	0.32	6.0
NT-2	820	0.08	0.11	2.0
NT-3	500	0.05	0.07	1.2
13/TT-27	4200	0.41	0.95	10
CB-03	1400	0.14	0.19	4.5

Appendix A

Recent Studies of Soil Ingestion Rate

Appendix A Recent Studies of Soil Ingestion Rate

EPA's use of RME sediment ingestion rates of 200 mg/day for a child and 100 mg/day for an adult is overly conservative. These values are based on 1994 Region I Guidance. EPA's 1997 Exposure Factors Handbook recommends soil ingestion rates of 100 mg/day for a child and 50 mg/day for an adult (USEPA, 1997). These values were used in our deterministic risk calculations presented in Section 3.1. In addition, more recent studies indicate that the average and high-end soil ingestion rates are lower than the 1994 values used by EPA. Recent studies of soil ingestion rates are discussed below. The results of these studies indicate that child soil ingestion rates would be better described by a mean rate of 45 mg/day, and a 95th percentile rate of 124 mg/day. This distribution was used in our probabilistic risk calculations described in Section 3.2.

Stanek and Calabrese (1995a) performed a re-analysis of a previous soil ingestion study of 64 children (ages 1-4) in Amherst, Massachusetts (Calabrese *et al*., 1989). The Amherst study is one of the most comprehensive and detailed studies of children's incidental soil ingestion to date (Calabrese *et al*., 1989). In this study, incidental soil ingestion rates were estimated using a mass balance approach. In the re-analysis, the Amherst data were used to develop distributions of potential daily soil ingestion rates, including estimates for various percentiles of the study population. Using this approach, the authors estimated a mean soil ingestion rate for the 50th percentile child (ages 1-4 years) of 45 mg/day (Stanek and Calabrese, 1995a). This re-analysis differs from earlier interpretations of the Amherst study (including evaluations conducted by the study researchers) and reflects a more robust approach that takes into account a greater degree of the information reflected in the study data.

Stanek and Calabrese (1995b) re-analyzed a combined data set (n=168) based on the Amherst study mentioned above, and another soil ingestion study by Davis *et al*. (1990) that involved 104 children (ages 2-7) in the state of Washington. Based on their re-analysis of the combined dataset, the authors estimated a mean soil ingestion rate for the 50th percentile child of 37 mg/day.

Stanek and Calabrese (2000) performed a soil ingestion study of 64 children (ages 1-4 years) living on a Superfund site in Anaconda, Montana. Stanek and Calabrese derived a seven-day average soil ingestion rate for the 50th percentile child of 17 mg/day. (The comparable value based on the 1989 Amherst population was 45 mg/day.) The seven-day average soil ingestion rate for the 95th percentile child was 141 mg/day (compared to 208 mg/day for the Amherst population.) Stanek and Calabrese (2000) also estimate average soil ingestion rates over longer time periods, based on the seven-day study period. They estimate that the 95th percentile child will have a 365 day average soil ingestion rate of 106 mg/day for the Anaconda population and 124 mg/day for the Amherst population. These estimates are based on an analysis of uncertainty in the daily soil ingestion estimates, using standard statistical techniques.

EPA Response: The child and adult soil ingestion values recommended in the 1997 Exposure Factors Handbook represent central estimate values and are appropriate for use in a central tendency evaluation. The Exposure Factors Handbook does not recommend upper percentile values for use with a reasonable maximum scenario. Therefore, EPA Region I values, recommended for use in a reasonable maximum scenario, were selected for use. These values are consistent with ingestion rates recommended by MADEP and, as stated in the Exposure Factors Handbook, are within the range of ingestion estimates from published studies. The central tendency ingestion rates utilized are the same as those recommended in the Exposure Factors Handbook for use in a central tendency evaluation.

Appendix B

Arsenic Toxicity

Appendix B Arsenic Toxicity

The current arsenic Cancer Slope Factor (CSF) for arsenic of 1.5 $(mg/kg\text{-}day)^{-1}$ is based on skin cancer observed in a study of over 40,000 people in Taiwan who were exposed for a significant portion of their lifetime to high concentrations of arsenic in groundwater used for drinking water (Chen *et al*., 1985; Tseng *et al*., 1968). The CSF derived from this study is generally believed to be conservative – see for example, Morales *et al*., 2000; SEGH, 2002; Brown *et al*. 2000; and Buchet and Lison, 2000. This section discusses the toxicity of arsenic, providing evidence of the conservatism in the current USEPA CSF for arsenic. Although we do not necessarily suggest that EPA use an alternative value for the CSF, this Appendix provides a perspective on the conservatism in the calculated risks. Several studies conducted in the U.S. have shown that people exposed to arsenic in drinking water, at doses higher than those estimated in this risk assessment, do not have an increased risk of cancer. In addition, the estimated doses of arsenic for individuals exposed to this site are much lower than those in studies of overseas populations that do show evidence of an increased risk of cancer from exposure to arsenic.

B.1 U.S. Epidemiological Studies of Arsenic Carcinogenicity

B.1.1 Overview of U.S. Epidemiological Studies of Arsenic Exposure

Several well-designed epidemiological studies have been conducted in U.S. populations with highly elevated arsenic exposures. The U.S. epidemiological studies consistently show a lack of association between arsenic exposure and cancer outcomes. Table B-1 summarizes findings from the best available epidemiological studies of U.S. populations with elevated arsenic exposures, including two with high childhood exposures. These studies are summarized below:

- The Lewis *et al*. (1999) study, conducted by USEPA scientists, was designed to investigate the health effects of chronic consumption of arsenic-contaminated drinking water in a cohort of 4,058 residents of Millard County, Utah. For the seven communities included in the study, average drinking water concentrations ranged from 18 to 191 µg/L, and maximum detected concentrations ranging as high as 620 µg/L. Together with information on the residence history of the cohort members, the median drinking water concentrations were used to establish three arsenic exposure indices: low (<1,000 ppb-years), medium (1,000-4,999 ppb-years), and high (≥5,000 ppb-years).

- Despite highly elevated exposures to arsenic in drinking water, Lewis *et al*. (1999) reported the lack of a relationship between bladder and lung cancer and exposure to drinking water arsenic in the Utah cohort. A small, but statistically significant increase in prostate cancer was noted, but it was not dose dependent, and thus does not confirm a relationship between arsenic and prostate cancer.

- Based on their findings, the authors concluded "Whereas the studies in Taiwan and Argentina reported high exposures to drinking water arsenic, this study population was exposed to much lower levels, perhaps indicating that bladder cancer occurs in response to higher arsenic."

- A case-control study in Utah failed to find a relationship between bladder cancer and arsenic exposure from drinking water. The drinking water concentrations of arsenic in this study averaged 5μg/L (the total range was 0.5-160 μg/L). While this case-control study suggested that smoking might potentiate the effects of arsenic-induced bladder cancer, this observation was not consistent with respect to latency period (Bates *et al.*, 1995).

- A large ecological study, conducted by Morton *et al*. (1976), examined skin cancer incidence in a large study population of 190,871 exposed to arsenic drinking water concentrations averaging 16.5 μg/L and 4.8 μg/L in rural and urban regions respectively. No relationship between skin cancer and arsenic was found. Based on results, the authors stated that "it seems safe to conclude that our data showed no evidence of water arsenic influence on skin cancer incidence in Lane County over this 14-year period."

- In Churchill County, Nevada, Moore *et al*. (2002) investigated the relationship between childhood cancer incidence and arsenic exposure in drinking water from 1979 to 1989. Over 327,000 Nevada children were grouped into low, medium, and high exposure categories (*i.e.*, >10 μg/L, 10-25 μg/L and 35-90 μg/L, respectively). No statistically significant association between arsenic and any type of childhood cancer was found in any of the exposure groups.

- Tollestrup *et al*. (2002) used a cohort of over 3,000 children (aged 2 to 14) who had lived in close vicinity to the ASARCO Ruston copper smelter between the years 1910 and 1932 to examine to association between arsenic exposure and cause of death, which occurred 30 to 80 years after exposure. The authors used the number of years lived in a one-mile radius (*i.e.*, designed categories of 0- < 1.0 year, 1.0-3.9 years, 4.0 –9.9 years, and >10 years) of the smelter stack as a surrogate for arsenic exposure. The study found no evidence of increased bladder or lung cancer mortality rates, even in the three highest arsenic exposure categories.

- Lamm and coworkers (2002) conducted an extensive analysis of the relationship between arsenic in drinking water and cancer incidence using data from 133 US counties and over 75 million person-years of observations. Bladder cancer mortality data were collected for the years 1950 to 1979 along with Unites States Geological Survey (USGS)-derived data on arsenic levels in US groundwater supplies. Bladder cancer standard mortality ratios (SMRs) from individual counties dependent on groundwater as a drinking source, having median levels ranging from 3-60 μg/L, were compared to county-specific arsenic groundwater concentrations. Linear regression analysis of these data indicated that the slope estimate of this relationship was indistinguishable from zero, *i.e.* there was no evidence of a dose response relationship between arsenic intake and bladder cancer.

In summary, despite some highly elevated arsenic exposures (higher than those for the Aberjona River), these studies do not show evidence of increased excess bladder, lung, or skin cancer risk in the United States. These studies indicate that ingestion of arsenic in drinking water, at the levels found in the U.S., do not cause cancer. It should be noted that what are considered to be elevated arsenic exposures among U.S. populations are still substantially lower than those of the Taiwanese and South American populations where large excess lifetime bladder, lung, and skin cancer risks have been observed. Consequently, these U.S. epidemiological studies are suggestive of a possible threshold for arsenic carcinogenicity. Findings from these studies thus indicate that the use of a cancer slope factor (CSF)

based on studies of cancer occurrence (bladder, lung, and skin) in highly exposed Taiwanese populations may result in overestimates of arsenic-related cancer risk in the United States.

B.1.2 Interpretation of U.S. Studies

Prevalence of Skin Cancer In Populations With Elevated Arsenic Exposures

As noted above, there is a lack of observed skin cancer cases in U.S. epidemiological studies of populations with elevated arsenic exposures. Valberg *et al*. (1998) examined whether this observation was more likely due to an absence of risk in U.S. populations or random variability from a predicted risk. This was done using a likelihood ratio approach that evaluated which of two hypotheses was the more likely explanation for the lack of observed skin cancer cases in the studies of U.S. populations. This analysis showed that no effect of arsenic on skin cancer prevalence was about 2.2 times more likely than an effect of arsenic exposure on skin cancer prevalence as predicted by EPA's current arsenic cancer potency factor of 1.5 $(mg/kg/day)^{-1}$. This study thus indicates that using a cancer potency factor based on a study of elevated arsenic exposures in the Taiwanese population may result in overestimates of skin cancer prevalence in the U.S. population.

Power of U.S. Epidemiological Studies To Detect Arsenic-Related Health Risks

A recent sample size calculation published in *Environmental Health Perspectives* supports the point that epidemiological studies of U.S. populations, such as the Lewis *et al*. (1999) study of Millard County, Utah, have sufficient power to detect the postulated arsenic-health risks if the risks are indeed as high as those estimated for Taiwanese populations (Frost *et al*., 2002). Specifically, Frost *et al*. (2002) estimated the sample size required to test the arsenic risk predicted by Morales *et al*. (2000) for the United States.[5] In order to detect these large predicted excess risks, Frost *et al*. concluded that a sample size of approximately 1,400 would be needed for an arsenic drinking water exposure level of 100 µg/L. This sample size requirement was more than satisfied by the Lewis *et al*. (1999) study of a cohort of 4,058 individuals in Millard County, Utah, described in Section B.1.1. Frost *et al*. concluded that their findings were inconsistent with the "postulated excess risk for lung and bladder cancers", and did not "support the concerns that epidemiologic studies in the United States are not sufficiently powerful to detect the postulated arsenic-related health risks."

B.2 Non-U.S. Epidemiological Studies of Arsenic Carcinogenicity

Several studies conducted outside the United States have established arsenic as a skin, bladder, and lung carcinogen in humans. However, many of these studies have found an increased risk of cancer only at relatively high doses of arsenic, *i.e.*, arsenic concentrations in drinking water greater than 100 µg/L (for review see Brown and Ross, 2002). Several key studies are summarized below:

- The relative risk for urinary cancer and transitional cell carcinoma in a northeastern Taiwanese study population (based on a National Taiwan comparison group) was statistically significant only at arsenic concentrations in drinking water greater than 100 µg/L (Chiou *et al*., 2001).

[5] The Morales *et al*. (2000) re-analysis of internal cancer risks in the arsenic-endemic region of southwestern Taiwan was used by U.S. EPA to calculate cancer risks at various MCL options in revising the arsenic drinking water regulations.

- Morales *et al*. (2000) re-analyzed the original data from Southwestern Taiwan. Using a the Taiwanese population as a comparison group, a recalculation of the relative risks for lung and bladder cancer showed a statistically significant dose-response relationship only at arsenic concentrations in drinking water that were greater than 400 µg/L.
- Lamm (2003) also re-analyzed data from southwestern Taiwan, considering differences in arsenic exposure from artesian (pressurized deep water) *vs.* non-artesian (shallow water) wells. Lamm concluded that bladder cancer incidence was independent of arsenic levels in villages that did not rely on the artesian wells as a water source. In contrast, when a village relied exclusively on water from artesian wells, a relationship was found. This indicates that contaminants in artesian wells (*i.e.*, humic acids, fluorescent substances, and fungal toxins), other than arsenic, may have contributed to increased bladder cancer risk.
- Guo and Tseng (2000) re-collected and re-analyzed data from Southwestern Taiwan. The study examined both bladder cancer incidence and death in the arsenic-contaminated region. While the study demonstrated a relationship between arsenic concentration and bladder cancer (incidence and death), this relationship was observed only at drinking water arsenic concentrations greater than 640 µg/L.
- In a cross-sectional study from Inner Mongolia (Tucker *et al.*, 2001; as cited in NRC, 2001), skin cancer was observed only in individuals exposed to peak concentrations of 150 µg/L or greater.

B.3 Non-linearity of Dose-response Relationship for Arsenic Carcinogenicity

The use of a cancer slope factor to quantify cancer risks associated with arsenic ingestion includes the default assumption that the dose-response relationship is linear at low doses. This assumption implies that even a very low dose of arsenic confers some excess cancer risk, and that, as the dose increases, risk increases in a directly proportional fashion. Careful examination of the biological principles that govern arsenic toxicity indicate that this assumption is incorrect for arsenic and that the true dose-response relationship is likely to be sub-linear or non-linear. Thus, from a toxicological perspective, low doses of arsenic would be relatively less harmful than higher doses, and may, in fact, be associated with zero risk.

A key fact that supports non-linearity for the arsenic dose-response relationship is associated with the way in which arsenic alters gene expression (Rudel *et al*., 1996; Kitchin *et al*., 2001). Specifically, arsenic does not interact directly with DNA to produce point mutations, but instead may modify gene transcription through one or more indirect mechanisms, including chromosome alterations, changes in DNA-methylation patterns, and perturbation of key regulatory enzymes.

A description of possible mechanisms of arsenic-induced carcinogenesis is provided below. These mechanisms are not mutually exclusive and all are consistent with a non-linear dose-response relationship.

- Arsenic has conclusively been shown to induce chromosome damage without interacting with DNA in cell culture systems as well as in animals. (Noda *et al*., 2002; Wang *et al*. 1994; Vega *et al*., 1995; NRC 1999).

- Arsenic affects DNA methylation status, which can affect the transcriptional regulation of genes critical to cell growth and cell death (Zhao *et al.*, 1997; Mass and Wang, 1997).
- Arsenic may inhibit aspects of DNA repair including inhibition of p53 (Mass and Wang, 1997) and components of the nucleotide excision repair system (Hu *et al*., 1998; Andrew *et al*., 2003)
- Arsenic may modulate cell signaling pathways responsible the regulation of cell proliferation. Specifically, exposure to arsenic can activate the c-Src dependent Epidermal Growth Factor Receptor (EFGR) and the mitogen-activated protein kinase (MAPK) cell signaling pathways (Simeonova and Luster, 2002; Bode and Dong, 2001)
- Metabolism of arsenic to its trivalent methylated metabolites (MMA^{III} and DMA^{III}) can generate reactive free oxygen radicals that can cause DNA damage (Kitchin *et al*., 2003; Mass *et al*., 2001).
- Treatment of human cells with micromolar concentrations of arsenic can induce protective cellular mechanisms such as the enhanced transcription of glutathione-related genes and induction of heat shock proteins (Del Razo *et al*., 2001; Schuliga *et al*., 2002).
- Luster (2003) suggests that arsenic acts through multiple mechanisms and suggests that the dose-response for arsenic is likely to be non-linear in the low dose region.

Based on available data, including the above proposed modes of action, arsenic does not appear to be an initiating carcinogen (*i.e.*, the type of carcinogen for which a linear dose-response relationship is plausible).

Despite the strong evidence that arsenic does not exert its toxicity in a linear fashion, both the EPA and the NRC have used linear models to estimate human risks at low arsenic exposures. This decision was made based on a 1996 EPA guidance document which states that, in the absence of definitive mode of action, a linear default assumption will be utilized. Thus, the decision to reject a non-linear or threshold model for arsenic carcinogenesis was a decision based on policy and not the most biologically plausible model. Because the EPA cancer slope factor in IRIS is based on a linear dose-response relationship, and the true dose-response is likely to be non-linear, use of the cancer slope factor is likely to overestimate cancer risks at exposure levels lower than those experienced in the Taiwanese study upon which the CSF is based.

Evidence of arsenic's non-linearity is further supported by evidence from epidemiological studies. As discussed previously, U.S.-based studies indicate that elevated of levels of arsenic in drinking water are not associated with increased bladder and lung cancer risk. In addition, studies from Taiwan and Inner Mongolia demonstrate that arsenic does not pose a significant cancer risk until drinking water levels are greater than 100 µg/L. Collectively, these studies indicate that increased risk of cancer is not associated with low doses of arsenic.

B.4 Evaluation of Exposure to Arsenic in Soil

By comparison with food and water, incidental ingestion of arsenic from contaminated soil or sediment does not contribute significantly to total arsenic intake and resulting arsenic body burden. The modest impact of arsenic on body burden is evidenced by studies that show low increases in urinary arsenic levels after soil exposure. Although elevated urinary arsenic levels were reported to be associated

with very high soil arsenic levels near copper smelters (Baker *et al*., 1977; Binder *et al*., 1987), studies of populations of children residing in communities with concentrations of arsenic in soil at or below 200 mg/kg indicate very little, if any, effect of arsenic in soil on body burden of arsenic, as reflected in urine arsenic levels (Valberg *et al.*, 1997; Hewitt *et al.*, 1995). In addition, the Anaconda, MT study demonstrated that urinary arsenic levels were unaffected by soil arsenic levels as high as 500 mg/kg (Hwang *et al.*, 1997). This observation is likely due to the small impact of soil arsenic relative to the impact of background levels of arsenic in food and water. Although there is no literature specifically on arsenic exposures to sediment, we would expect that exposure to sediment would be similar to that in soil, and that it would have a similarly small impact.

Studies of arsenic contamination in the area adjacent to the former ASARCO copper smelter in Ruston, Washington indicate that childhood exposures to arsenic in soil and air do not result in increased rates of bladder or lung cancer during adulthood. The study followed a cohort of children residing in the area during smelter operation during 1907-1932. The authors used the number of years lived within a one-mile radius of the smelter stack as a surrogate for total ambient arsenic exposure *via* soil and air. Exposure was evaluated as a function of duration of residence (categories of 0-<1.0 year, 1.0-3.9 years, 4.0-9.9 years, and >10 years). Arsenic soil concentrations ranged from 100 to 1600 mg/kg when measured in 1974 (Harter *et al.* 1993), and thus were at least that high during the exposure period of 1907-1932. The study found no evidence of increased bladder or lung cancer mortality rates, even in the three highest arsenic exposure categories (Tollestrup *et al*., 2002; Harter *et al*., 1993; Frost, 2003). While the cohort in this study was exposed to arsenic *via* both soil and air, another study conducted at this site in the mid 1980's demonstrated that exposure to arsenic *via* incidental ingestion of soil had a strong correlation to urinary arsenic levels indicating that soil exposure is an important determinant of total arsenic dose in children (Polissar *et al*., 1990).

Adverse health effects from exposure to arsenic in soil are not addressed in any of the above studies. ATSDR's Toxicity Profile for Arsenic (ATSDR, 2000) does recognize arsenic-contaminated soil as a potential source of adverse health effects. However, ATSDR acknowledges that arsenic-bound soil has low bioavailability, through both the oral and dermal route, that will limit toxicity. Additionally, the profile does not present any studies in which exposure to arsenic-contaminated soil resulted in adverse health effects.

B.5 Conclusions

Several U.S.-based studies provide evidence that even relatively high levels of arsenic in drinking water do not result in increased cancer risk. By estimating water consumption in these exposed populations, we can calculate total arsenic intake and compare these values to estimated exposures to arsenic in sediment along in the Aberjona River. It is also useful to compare site-specific exposures of arsenic to levels ingested at the MCL for arsenic in drinking water. In all cases, we find that exposures to arsenic in sediment along the Aberjona River are well below levels at which no cancer increase was observed in U.S. studies, and are also less than permissible exposures to arsenic in drinking water at the MCL of 10 μg/L.

EPA has estimated site-related lifetime daily average arsenic intakes up to 0.3 μg/kg-day for a child, and 0.13 μg/kg-day for an adult, (for future RME exposures at NT-1). In contrast, estimated arsenic intakes as high as 5.7 μg/kg-day have been experienced by U.S. populations without evidence of increased cancer risks (see Table B-1). Specifically, for the Lewis *et al.* (1999) study, which is among the largest and best-conducted of the epidemiological studies of U.S. populations with elevated arsenic

exposures, average intakes of arsenic in drinking water ranged from 0.26 to 2.7 μg/kg-day (based on average drinking water consumption of 1L/day (Jacobs *et al*. 2000)). Over 1,200 members of the Millard County, Utah, cohort resided in the two communities with the highest intake level (average 2.5 μg/kg-day), many for their entire lifetimes. Despite these elevated intakes, no elevated death rates from bladder or lung cancers were observed for those who died through November 1996 (2,203 cohort members), and death rates were not elevated among the cohort members with the highest levels of drinking water arsenic. The observed bladder and cancer mortality risks in the Lewis *et al*. study are lower than the baseline health risks predicted for the general population of Utah, even with arsenic drinking water concentrations that on average were as high as 191 μg/L, and at times exceeded 600 μg/L.

In the non-U.S. studies cited in Section B.2, populations had exposure to arsenic in drinking water at concentrations of 100 μg/L or greater. In order to calculate arsenic intakes, certain assumptions must be made about the exposed populations. For example, using estimates of water consumption patterns in Taiwanese males developed by the National Research Council (NRC, 1999; NRC, 2001), calculated arsenic intakes at 100 μg/L are 5.5 μg/kg-day. This assumes an average Taiwanese male weighs 55 kg and drinks 3L/day of contaminated water. Moreover, if one assumes, based on the work of Lamm and Kruse (2003) and the re-analysis of the Taiwan data by Morales *et al*. (2000), that cancer is not increased until levels of 400 μg/L, then the estimated carcinogenic intake in Taiwan would be 22 μg/kg-day. In contrast, site-related exposures are considerably less than the drinking water exposures in these studies. For example, EPA's estimated arsenic intakes for an adult at CB-03 are 0.12 μg/kg-day, which is 45 times lower than doses received at 100 μg/L in the Taiwanese studies. Thus, modest intakes of arsenic from exposure to sediment along the river are unlikely to present a significant toxicological concern.

Estimated arsenic exposures along the Aberjona River are less than arsenic exposures permitted in drinking water at the MCL of 10 μg/L, which is a level designed to be health protective (USEPA, 2001a). As an example, EPA's RME estimates of arsenic intake at CB-03 are 0.27 μg/kg-day for children and 0.12 μg/kg-day for adults. By comparison, exposure to arsenic in drinking water at the current MCL of 10 μg/L would yield an estimated intake of 0.7 μg/kg-day for a 15 kg child and 0.3 μg/kg-day for a 70 kg adult, based on drinking water intakes of 1L/day for children and 2L for adults. Thus, site-related arsenic exposures are less than those considered by EPA to be health protective in drinking water.

EPA Response: The discussion in the uncertainty section relative to the arsenic toxicity factor will be expanded.

Table B-1
Summary of Epidemiological Studies of Cancer Risks in U.S. Populations with Elevated Arsenic Exposures

Study Type	Study Location	Study Population(s)	As Drinking Water Levels (µg/L)	Average Daily As Intakes (µg/kg-day)	Key Findings on Cancer Health Effects	Reference
Lifetime/Adult Exposures						
Retrospective Cohort	Millard County, UT	4,058 Adults	Averages ranging from 18 to 191	0.26 to 2.7 (based on average water levels, 1 L/day ingestion rate, and 70 kg body weight)	No elevated death rates from bladder or lung cancers have been observed for those who died through November 1996, and death rates show no association with exposure level. For bladder and lung cancers together, the authors observed 39 deaths when 63.5 were expected ($p<0.05$).	Lewis *et al.*, 1999
Retrospective Cohort	Nationwide (133 US counties)	75 million person-years of observations	Median concentrations ranging from 3 to 60 µg/L	SMR: 0.73 (0.41 to 1.27) for bladder cancer at highest exposure level	After reviewing groundwater arsenic levels in 133 counties in the US dependent on groundwater as a drinking source, the authors found no relationship between arsenic exposure and bladder cancer mortality.	Lamm *et al*. 2002
Meta-analysis	Utilized studies of Fallon, NV (Vig *et al*., 1984), Fairbanks, AK (Harrington *et al*., 1978), and Millard County, UT (Southwick *et al*., 1983)	105 for Fallon, 79 for Fairbanks, and 145 for Millard County	100 for Fallon, 76-401 for Fairbanks, and 208 for Millard County	1.4 for Fallon, 1.1-5.7 for Fairbanks, and 2.9 for Millard County (based on average water levels, 1 L/day ingestion rate, and 70 kg body weight)	No skin cancers were found in the exposed populations in each study location. This study further examined whether an absence of risk in U.S. populations or random variability from a predicted risk was the more likely explanation for the study findings. Likelihood ratio analysis showed that no effect of arsenic on skin cancer prevalence is about 2.2 times more likely than an effect of arsenic exposure on skin cancer prevalence as predicted by EPA's current arsenic cancer potency factor of 1.5 $(mg/kg/day)^{-1}$.	Valberg *et al*., 1998
Case-control	88 towns in Utah	117 cases, 266 population-based controls	Range of 0.5 to 160, with a mean of 5 (81 out of 88 towns <10 µg/L; 1 town >50 µg/L)	0.00l to 2.3 (based on range of water levels, 1 L/day ingestion rate, and 70 kg body weight)	No association found between bladder cancer risk and arsenic exposure for two exposure metrics-total cumulative exposure (<19 up to >53 mg) and intake concentration. Analyses indicated increased bladder cancer risks for smokers, although authors could not rule out possible bias in data.	Bates *et al.*, 1995

Study Type	Study Location	Study Population(s)	As Drinking Water Levels (µg/L)	Average Daily As Intakes (µg/kg-day)	Key Findings on Cancer Health Effects	Reference
Ecological	Lane County, Oregon	190,871 total study population	Averages of 16.5 and 4.8 in all rural and urban regions, respectively, with a maximum recorded conc. of 33	Averages of 0.23 and 0.07 for rural and urban regions, respectively (based on average water levels, 1L/day ingestion rate, and 70 kg body weight)	Did not detect any excess risk of skin cancer associated with arsenic exposures up to 33 µg/L (note 19,063 people were exposed at this maximum concentration). Among the 3,237 skin-cancer cases identified in the study, only three had evidence of arsenic keratosis. "	Morton *et al.*, 1976
Childhood Exposures						
Ecologic Study	Entire State of Nevada, including Churchill County and Fallon, Nevada.,	327,947 children between 0-19 years of age	0-7.8 in low-exposure group, 10-24.6 in medium-exposure group, 35.9-91.5 in high-exposure group	0.57 to 1.4 in high-exposure group (based on average 0.6 L/day ingestion rate, and 38 kg body weight)	No statistically significant association between arsenic and any type of childhood cancer was found in any of the exposure groups.	Moore *et al.*, 2002
Retrospective Cohort	Ruston, Washington in vicinity of American Smelting and Refining Company (ASARCO) copper smelter	3,132 children residing near smelter between 1907-1932	Not reported in study (note that ambient air exposures are considered to be the primary exposure source)	Not known during 1907-1932 exposure period, although elevated urine As levels observed in 1970s following improvements in smelter processes	Despite elevated childhood As exposures, no elevated incidence of bladder or lung cancer mortality observed in 1,075 deceased members of cohort as of 12/31/90.	Tollestrup *et al.*, 2002

B.6 References

Agency for Toxic Substances and Disease Registry (ATSDR). 1997. "Toxicological Profile for Vinyl Chloride Update (Update)." Sciences International, Inc. Prepared for US Public Health Service, Agency for Toxic Substances and Disease Registry (ATSDR). September.

Agency for Toxic Substances and Disease Registry (ATSDR). 2000. "Toxicological Profile for Arsenic (Update)." Syracuse Research Corp. National Technical Information Service (NTIS), Springfield, VA. Prepared for US Public Health Service, Agency for Toxic Substances and Disease Registry (ATSDR). NTIS PB2000-108021. 446p. September.

Andrew, A.S., M.R. Karagas, and J.W. Hamilton. 2003. Decreased DNA repair gene expression among individuals exposed to arsenic in United States drinking water. *Int. J. Cancer* 104(3):263-268.

Baker, Jr., E.L., C.G. Hayes, P.J. Landrigan, J.L. Handke, R.T. Leger, W.J. Housworth, and J.M. Harrington. 1977. A nationwide survey of heavy metal absorption in children living near primary copper, lead, and zinc smelters. *Am. J. Epidemiol.* 106(4):261-273.

Bates M.N., A.H. Smith, and K.P. Cantor. 1995. Case-control study of bladder cancer and arsenic in drinking water. *Am. J. Epidemiol.* 141:523-530.

Binder, S., D. Forney, W. Kaye, and D. Paschal. 1987. Arsenic exposure in children living near a former copper smelter. *Bull. Environ. Contam. Toxicol.* 39:114(8).

Bode, A.M. and Z. Dong. 2002. The paradox of arsenic: Molecular mechanisms of cell transformation and chemotherapeutic effects. *Crit. Rev. Oncol. Hematol*. 42(1):5-24.

Brown, K.G., T.L. Kuo, H.R. Guo, L.M. Ryan, and C.O. Abernathy. 2000. Sensitivity analysis of U.S. EPA's estimates of skin cancer risk from inorganic arsenic in drinking water. *Hum. Ecol. Risk Assess*. 6:1055-1074.

Brown, K.G. and G.L. Ross. 2002. Arsenic, drinking water, and health: A position paper of the American Council on Science and Health. *Regul. Toxicol. Pharmacol.* 36(2):162-174.

Buchet, J.P. and D. Lison. 2000. Clues and uncertainties in the risk assessment of arsenic in drinking water. *Food Chem. Toxicol*. 38 (Suppl. 1):S81-85.

Calabrese, E.J., R. Barnes, E.J. Stanek, H. Pastides, C.E. Gilbert, P. Veneman, X. Wang, A. Lasztity, and P.T. Kostecki. 1989. How much soil do young children ingest: An epidemiologic study. *Regul. Toxicol. Pharmacol*. 10:123-137.

California Chromate Toxicity Review Committee. 2001. "Scientific Review of Toxicological and Human Health Issues Related to the Development of a Public Health Goal For Chromium(VI)." Downloaded from: http://www.dhs.cahwnet.gov/ps/ddwem/chemicals/Chromium6/reviewpanelreport.pdf. August 31.

Chen, C.J., Y.C. Chuang, T.M. Lin, and H.Y. Wu. 1985. Malignant neoplasms among residents of a blackfoot disease-endemic area in Taiwan: High-arsenic artesian well water and cancers. *Cancer Res.* 45:5895-5899.

Chiou, H.Y., S.T. Chiou, Y.H. Hsu, Y.L. Chou, C.H. Tseng, M.L. Wei, and C.J. Chen. 2001. Incidence of transitional cell carcinoma and arsenic in drinking water: A follow-up study of 8102 residents in an arseniasis-endemic area in Northeastern Taiwan. *Am. J. Epidemiol.* 153(5):411-418.

Davis, S., P. Waller, R. Buschbom, J. Ballou, and P. White. 1990. Quantitative estimates of soil ingestion in normal children between the ages of 2 and 7 years: Population-based estimates using aluminum, silicon, and titanium as soil tracer elements. *Arch. Environ. Health* 45(2):112-122.

Del Razo, L.M., B. Quintanilla-Vega, E. Brambila-Colombres, E.S. Calderón-Aranda, M. Manno, and A. Albores. 2001. Stress proteins induced by arsenic. *Toxicol. Appl. Pharmacol.* 177:132-148.

DeSesso, J.M. 2001. Teratogen update: Inorganic arsenic. *Teratology* 64(3):170-173.

Federal Register. 2000. Notice of Intent To Develop Ambient Water Quality Criteria for Protection of Human Health--Arsenic, Methylmercury, and Carbofuran; Notice of Data Availability; Request for Data and Information. 65(198). October 12. Downloaded from http://www.gpoaccess.gov/fr/advanced.html

Ferreccio, C., C. Gonzalez, V. Milosavjlevic, G. Marshall, A.M. Sancha, and A.H. Smith. 2000. Lung cancer and arsenic concentrations in drinking water in Chile. *Epidemiology* 11(6):673-679.

Frost, F. 2003. "An Epidemiological Perspective on Arsenic Exposure and Risk Assessment." Presented at Lovelace Respiratory Research Institute, Albuquerque, NM. 8 pp.

Frost, F., G. Craun, and K.G. Brown. 2002. Detection of excess arsenic-related cancer risks. *Environ. Health Perspect.* 110(1):A12-A13.

Germolec, D.R., J. Spalding, G.A. Boorman, J.L. Wilmer, T. Yoshida, P.P. Simeonova, A. Bruccoleri, F. Kayama, K. Gaido, R. Tennant, F. Burleson, W. Dong, R.W. Lang, and M.I. Luster. 1997. Arsenic can mediate skin neoplasia by chronic stimulation of keratinocyte-derived growth factors. *Mutat. Res.* 396(3):209-218.

Guo, H.R. and Y.C. Tseng. 2000. Arsenic in drinking water and bladder cancer: Comparison between studies based on cancer registry and death certificates. *Environ. Geochem. Health* 22(2):83-91.

Harter, L., F. Frost, K. Tollestrup, and F. Westrum. 1993. "Mortality Study of Children Residing Near ASARCO Copper Smelter in Ruston, Washington (Draft)." Washington Dept. of Health. December 3.

Hewitt, D.J., G.C. Millner, A.C. Nye, M. Webb, and R.G. Huss. 1995. Evaluation of residential exposure to arsenic in soil near a Superfund site. *Hum. Ecol. Risk Assess.* 1(3):323-335.

Hu, Y., L. Su, and E.T. Snow. 1998. Arsenic toxicity is enzyme specific and its affects on ligation are not caused by the direct inhibition of DNA repair enzymes. *Mutat. Res.* 408(3):203-218.

Hwang, Y.H., R.L. Bornschein, J. Grote, W. Menrath, and S. Roda. 1997. Environmental arsenic exposure of children around a former copper smelter site. *Environ. Res.* 72:72-81.

Integrated Risk Information System (IRIS). 1996. Manganese. U.S. Environmental Protection Agency. Downloaded from http://www.epa.gov/iris/subst/0373.htm.

Jacobs, H.L., J.T. Du, H.D. Kahn and K.A. Stralka. 2000. "Estimated Per Capita Water Ingestion in the United States." Prepared for US EPA, Office of Water, Washington, DC. EPA 815-R-00-008. April. Downloaded from http://www.epa.gov/waterscience/drinking/percapita/text.pdf on 8/7/01.

Kitchin, K.T. 2001. Recent advances in arsenic carcinogenesis: Modes of action, animal model systems, and methylated arsenic metabolites. *Toxicol. Appl. Pharmacol.* 172:249-261.

Kitchin, K.T. and S. Ahmad. 2003. Oxidative stress as a possible mode of action for arsenic carcinogenesis. *Toxicol. Lett.* 137(1-2):3-13.

Lamm, SH; Engel, A; Wilson, R; Feinleib, M. 2002. "Examination of the NRC Bladder Cancer Risk from Arsenic in Drinking Water Estimate, Using US Data with 75 Million Person-Years of Observation." Abstract presented at Fifth International Conference on Arsenic Exposure and Health Effect, Society of Environmental Geochemistry and Health. July 12-18, San Diego, CA.

Lamm, S.H. and M. Kruse. 2003. "Comments in Response the CPSC Report and Hearings on the Risk of Human Cancer from Arsenic Exposure." Consultants in Epidemiology & Occupational Health, Inc., Washington, DC. Submitted to US Consumer Product Safety Commission (CPSC), CCA Docket, 26 pp., March 28.

Lee, T., N. Tanaka, P.W. Lamb, T.M. Gilmer, and J.C. Barrett. 1988. Induction of gene amplification by arsenic. *Science* 241:79-81.

Lewis, D.R., J.W. Southwick, R. Ouellet-Hellstrom, J. Rench, and R.L. Calderon. 1999. Drinking water arsenic in Utah: A cohort mortality study. *Environ. Health Perspect.* 107(5):359-365.

Loehr, R.C. 1996. "The Environmental Impact of Soil Contamination: Bioavailability, Risk Assessment, and Policy Implications." Prepared for National Environmental Policy Institute, Bioavailability Policy Project, Reason Foundation, Policy Study No. 211, 21 pp. August.

Luster, M.I. 2003. "Proposed Mechanisms for Arsenic Carcinogenicity: Implications for the Shape of the Dose-Response Curve." Abstract presented at 2003 Society of Toxicology Conference.

Mass, M.J. and L. Wang. 1977. Arsenic alters cytosine methylation patterns of the promoter of the tumor suppressor gene p53 in human lung cells: A model for a mechanism of carcinogenesis. *Mutat. Res.* 396(3):263-277.

Mass, M.J., A. Tennant, B.C. Roop, W.R. Cullen, M. Styblo, D.J. Thomas, and A.D. Kligerman. 2001. Methylated trivalent arsenic species are genotoxic. *Chem. Res. Toxicol.* 14:355-361.

Moore, L.E., M. Lu, and A.H. Smith. 2002. Childhood cancer incidence and arsenic exposure in drinking water in Nevada. *Arch. Environ. Health* 57:201-206.

Morales, K.H., L. Ryan, T-L. Kuo, M-M. Wu, and C-J. Chen. 2000. Risk of internal cancers from arsenic in drinking water. *Environ. Health Perspectives* 108(7):655-661.

Morton, W., G. Starr, D. Pohl, J. Stoner, S. Wagner, and P. Weswig. 1976. Skin cancer and water arsenic in Lane County, Oregon. *Cancer* 37:2523-2532.

National Environmental Policy Institute (NEPI). 2000. "Assessing the Bioavailability of Metals in Soil for Use in Human Health Risk Assessments." Bioavailability Policy Project Phase II, Metals Task Force Report. Summer. Downloaded from http://www.nepi.org/pubs/metals-bio%20final.pdf on June 27, 2001.

National Research Council (NRC). 1999. "Arsenic in Drinking Water." Subcommittee on Arsenic in Drinking Water. National Academy Press, Washington, DC.

National Research Council (NRC). 2001. "Arsenic in Drinking Water: 2001 Update." National Academy Press, Washington, DC.

Noda, Y., T. Suzuki, A. Kohara, A. Hasegawa, T. Yotsuyanagi, M. Hayashi, T. Sofuni, K. Yamanaka, and S. Okada. 2002. *In vivo* genotoxicity evaluation of dimethylarsinic acid in Muta(TM)Mouse. *Mutat. Res*. 513(1-2):205-212.

Polissar, L., K. Lowry-Coble, D.A. Kalman, J.P. Hughes, G. van Belle, D.S. Covert, T.M. Burbacher, D. Bolgiano, and N.K. Motte. 1990. Pathways of Human Exposure to Arsenic in a Community Surrounding a Copper Smelter. *Environmental Research* 53:29-47.

Proctor, D.M., J.M. Otani, B.L. Finley, D.J. Paustenbach, J.A. Bland, N. Speizer, and E.V. Sargent. 2002. Is hexavalent chromium carcinogenic *via* ingestion? A weight-of-evidence review. *J. Toxicol. Environ. Health A* 65:701-746.

Rudel, R., T.M. Slayton, B.D. Beck. 1996. Implications of arsenic genotoxicity for dose-response of carcinogenic effects. *Regul. Toxicol. Pharmacol.* 23: 87-105.

Schoof, R.A., L.J. Yost, J. Eickhoff, E.A. Crecelius, D.W. Cragin, D.M. Meacher, and D.B. Menzel. 1999. A market basket survey of inorganic arsenic in food. *Food Chem. Toxicol.* 37: 839-846.

Schuliga M., S. Chouchane, and E.T. Snow. 2002. Upregulation of glutathione-related genes and enzyme activities in cultured human cells by sublethal concentrations of inorganic arsenic. *Toxicol. Sci.* 70(2):183-192.

Simeonova P.P. and M.I. Luster. 2002. Arsenic carcinogenicity: relevance of c-Src activation. *Mol. Cell Biochem.* 234-235(1-2):277-82.

Society for Environmental Geochemistry and Health. 2002. *SEGH Fifth International Conference on Arsenic Exposure and Health Effects, Book of Abstracts, San Diego, CA*, July 14-18, 251 pp.

Stanek, E.J. and E.J. Calabrese. 1995a. Soil ingestion estimates for use in site evaluations based on the best tracer method. *Human Ecol. Risk Assess*. 1(2):133-156.

Stanek, E.J. and E.J. Calabrese. 1995b. Daily estimates of soil ingestion in children. *Environ. Health Perspect.* 103(3):276-285.

Stanek, E.J. and E.J. Calabrese. 2000. Daily soil ingestion estimates for children at a Superfund site. *Risk Analysis* 20(5):627-635.

Stokinger, H.E. 1955. Standards for safeguarding the health of the industrial worker. *Public Health Rep*. 70(1):1(11).

Tollestrup, K., F.J. Frost, L.C. Harter, and G. McMillan. 2002. Mortality in children residing near the ASARCO copper smelter in Ruston, Washington. *Am. J. Epidemiol.* 155(11): SER Abstract #154. Society for Epidemiologic Research. Abstracts of the 35th Annual Meeting, Palm Desert, CA, June 18-21.

Tucker, S.B., S.H. Lamm, F.X. Li, and R. Wilson. 2001. "Relationship Between Consumption of Arsenic-Contaminated Well-Water and Skin Disorder in Huhhot, Inner Mongolia." 33 pp. July 15.

United States Environmental Protection Agency (USEPA). 1992. "Dermal Exposure Assessment: Principles and Applications." Office of Health and Environmental Assessment, Washington DC. EPA/600/8-91/011B. January.

U.S. Environmental Protection Agency (USEPA). 1994. "EPA Region 1 Waste Management Division: Risk Updates (Number 2)." 24 pp. August.

U.S. Environmental Protection Agency (USEPA). 1996. "EPA Region 1 New England: Risk Updates (Number 4)." 10 pp. November.

U.S. Environmental Protection Agency (USEPA). 1997. "Exposure Factors Handbook. Volume I: General Factors and Volume III: Activity Factors." Office of Research and Development, Washington, D.C. EPA/600/P-95/002Fa, EPA/600/P-95/002Fc. August.

U.S. Environmental Protection Agency (USEPA). 2000. "Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health (2000) (Final Report)." Prepared for US EPA, Office of Water US EPA, Office of Science and Technology, Washington, DC. EPA-822-B-00-004. October.

U.S. Environmental Protection Agency (USEPA). 2001a. Arsenic in drinking water. Final Rule. *Federal Register* 66(14)7000-7010. January 22.

U.S. Environmental Protection Agency (USEPA). 2001b. "Risk Assessment Guidance for Superfund (RAGS). Volume III: Part A, Process for Conducting Probabilistic Risk Assessment." Office of Emergency and Remedial Response, Washington, DC. EPA 540-R-02-002; Publication 9285.7-45; PB2002-963302. 385 pp. December.

U.S. Environmental Protection Agency (USEPA). 2002. Office of Emergency and Remedial Response (Washington, DC). "Calculating upper confidence limits for exposure point concentrations at hazardous waste sites. Supplemental guidance to RAGS." OSWER Directive 9285.6-10. December. Downloaded from: http://www.epa.gov/superfund/programs/risk/ragsa/ucl.pdf.

U.S. Environmental Protection Agency (USEPA). 2003. Office of Research and Development, National Exposure Research Laboratory (Las Vegas, NV). "ProUCL statistical software and user's guide (Version 2.1)." February.

U.S. Environmental Protection Agency Region 8 (Denver, CO); Benson, R. 2001. "Derivation of acute and subchronic oral reference doses for inorganic arsenic." February.

Valberg, P.A., B.D. Beck, P.D. Boardman, and J.T. Cohen. 1998. Likelihood ratio analysis of skin cancer prevalence associated with arsenic in drinking water in the USA. *Environ. Geochem. and Health* 20:61-66.

Valberg, P.A., B.D. Beck, T.S. Bowers, J.L. Keating, P.D. Bergstrom, and P.D. Boardman. 1997. Issues in setting health-based cleanup levels for arsenic in soil. *Regul. Toxicol. Pharmacol.* 26:219-229.

Vega, L., M.E. Gonsebatt, P. Ostrosky-Wegman. 1995. Aneugenic effect of sodium arsenite on human lymphocytes in vitro: An individual susceptibility effect detected. *Mutat. Res.* 334:365-373.

Wang, T.S. and H. Huang. 1994. Active oxygen species are involved in the induction of micronuclei by arsenite in XRS-5 cells. *Mutagenesis* 9(3):253-257.

Wester, R.C., H.I. Maibach, L. Sedik, J. Melendres, and M. Wade. 1993. *In vivo* and *in vitro* percutaneous absorption and skin decontamination of arsenic from water and soil. *Fundam. Appl. Toxicol.* 20:336-340.

Zhang, J. and S. Li. 1997. Cancer mortality in a Chinese population exposed to hexavalent chromium in water. *J. Occup. Environ. Med.* 39(4):315-319.

Zhao, C.Q., M.R. Young, B.A. Diwan, T.P. Coogan, and M.P. Waalkes. 1997. Association of arsenic-induced malignant transformation with DNA hypomethylation and aberrant gene expression. *Proc. Natl. Acad. Sci.* 94:10907-10912.

Appendix C

Evaluation of the EPA's UCL Recommendations for Skewed Data Sets

Evaluation of the EPA's UCL Recommendations for Skewed Data Sets

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Exposure point concentrations (EPCs) used in risk assessments should reflect the average contaminant concentrations encountered by a receptor at a site. This parameter is typically represented by the upper 95% confidence limit on the mean (95% UCL). The 95% UCL of the concentration mean is a measure of the precision to which the average concentration can be measured. Statistically, the 95% UCL estimates the 95th percentile of the sampling distribution of the sample average. That is, if one were to create 100 sets of measurements each set selected at random from the same population having a known mean, then 95 of the computed UCL values would be expected to be above the true mean and 5 would be expected to be below the true mean. Any method for calculating the 95% UCL should have this property; while at the same time, it is preferable to use methods that do not substantially overestimate the true mean.

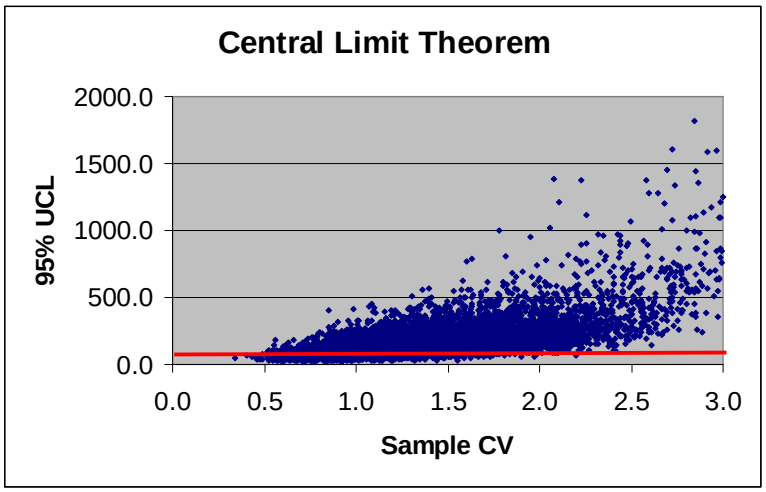
Numerous statistical methods are available for the calculation of 95% UCLs, however, they often yield disparate results. The U.S. Environmental Protection Agency (EPA) has recently provided guidance and companion software (EPA, 2002) for the calculation of EPCs at contaminated sites. These efforts extend previous EPA guidance (1992) by incorporating a variety of statistical methods and are generally considered an improvement of the earlier guidance. However, based on the results of an analysis we presented at the 2003 annual meeting of the Society of Toxicology (Mills et al., 2003), the EPA UCL recommendations may either underestimate or overestimate the true mean depending on site-specific data characteristics.

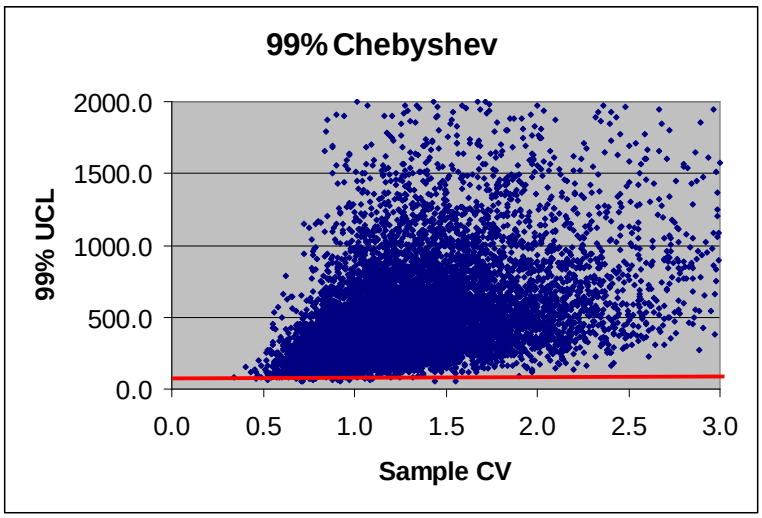
Our original analysis has significant implications for the UCLs selected by EPA to represent the EPCs in the human health risk assessment for the Wells G&H Superfund Site Operable Unit 3 - Aberjona River Study. An additional analysis was conducted to specifically evaluate the UCL selected by EPA to represent the EPC for the WH arsenic data set, a small, relatively skewed sample population with a sample size (n) of 12 and a Coefficient of Variation (CV) of 2.4. To evaluate the EPA methodology with this data set, we expanded our analysis on the performance of UCL methods to increase the sample observations in the 2.2 to 2.6 CV range, similar to the WH arsenic data set.

Although the true population parameters are never known for chemical concentrations at a site, a reasonable inference is that sample data with CV 2.4 were drawn from a population with CV 2.4. To evaluate this type of case, 10,000 synthetic lognormal data sets (n=12) were generated using Crystal Ball (Mean 100; Std 240; CV 2.4). For each sample data set, UCLs were calculated with the 95% CLT, 95% Bootstrap, 95% Chebyshev (MVUE), and the 99% Chebyshev (MVUE) methods. Table 1 below provides a summary of the UCL results for all the sample data sets. The Min and Max are the lowest and highest 95% UCL observed out of the data sets. Mean and Median UCLs are also shown. The 'Coverage' refers to the percent of the UCLs from the sample data sets that were larger than the true population mean of 100. For example, for 68% of the samples, the Central Limit Theorem (CLT) produced 95% UCLs greater than 100. By definition, a 95% UCL method providing nominal coverage would have a coverage of 95%.

Table 1. UCL results from 10,000 samples drawn from Ln(100, 240)					
	Min	**Max**	**Median**	**Mean**	**Coverage**
95% CLT	22.5	2383.1	131.9	169.7	68%
95% Bootstrap	21.6	2379.5	129.4	166.9	67%
95% H-Stat	29.9	93672.5	413.2	951.5	95%
95% Cheby (MVUE)	29.7	2871.8	236.3	294.2	93%
99% Cheby (MVUE)	51.1	5676.4	430.7	544.8	99%

These results suggest that the 95% H-statistic or the 99% Chebyshev (MVUE) would be the UCL method of choice since these two methods are the only ones to deliver at least the desired 95% coverage. We believe that EPA used a similar approach in the development of their UCL recommendations in ProUCL. If this is the case, EPA has neglected an important detail. The range of UCL results produced by each method is highly dependent on the sample CV. To illustrate this point, scatter plots of the UCL results (y-axis) versus the sample CV (x-axis) for the 95% CLT and 99% Chebyshev methods are shown in the two graphs below.





As the CVs of the sample data sets increase, the 99% Chebyshev (MVUE) method significantly over predicts the true mean with increasing frequency. The same is true of the CLT, but the extent of the "overage" is much more limited.

Given this observed relationship, we evaluated the coverage of the methods for the data sets with CVs in the range of 2.2 to 2.6, bracketing the WH arsenic data set (CV=2.4). Within the 10,000 sample data sets, 491 were identified with CVs in the range of 2.2 to 2.6. The performance of the CLT and Chebyshev (MVUE) UCL methods for this portion of the sample data is shown in Table 2 below.

Table 2. UCL results for 491 of 10000 samples drawn from Ln(100, 240) with sample CV of 2.2 to 2.6

	Min	Max	Median	Mean	Coverage
95% CLT	71.4	1374.8	329.3	375.9	99.4%
95% Cheby (MVUE)	72.3	2657.7	367.6	451.3	99.2%
99% Cheby (MVUE)	131.7	5176.4	683.6	856.8	100%

In this case, the coverage properties of these three methods are all adequate, in that all three provide at least 95% coverage. However, the 95% CLT produces the lowest mean and median UCLs indicating that the frequency of overestimation of the true mean (i.e., overage) is reduced. If the sample CV is a reasonable estimate of the population CV, this analysis indicates that the 95% CLT estimate provides 99% coverage and is a more appropriate (yet still conservative) estimate of the 95% UCL. When applied to the arsenic data set from the WH station, the 95% CLT yields a substantially lower UCL estimate (806 mg/kg) than the estimate based on the 99% Chebyshev (MVUE) method applied by EPA (1910 mg/kg).

An important uncertainty associated with this analysis is that the true population distribution that gives rise to site sampling data is never known. If the population is considerably more highly skewed than the sample would indicate, the coverage properties of these methods might be less than optimal. In fact, as the CV of the underlying population rises, eventually even the 99% Chebyshev method will fail to provide nominal coverage. Thus, if the population is considerably more skewed than the sample would indicate, then there is a higher probability that the UCL will under predict the true population mean. Alternatively, if the sample population was biased so as to produce sample data with more variability than the underlying population, then the UCL often greatly exceeds the true population mean.

The methodology used by EPA to develop recommendations for their ProUCL program is not available for review. It is unclear how the EPA distinguished between sample and population parameters in the development of the ProUCL recommendations. Use of sample parameters to estimate population parameters and underlying distribution types are particularly problematic when dealing with small sample sizes and highly skewed data sets. There is usually only weak evidence that the underlying population even follows a specified distribution. Formal Goodness-of-

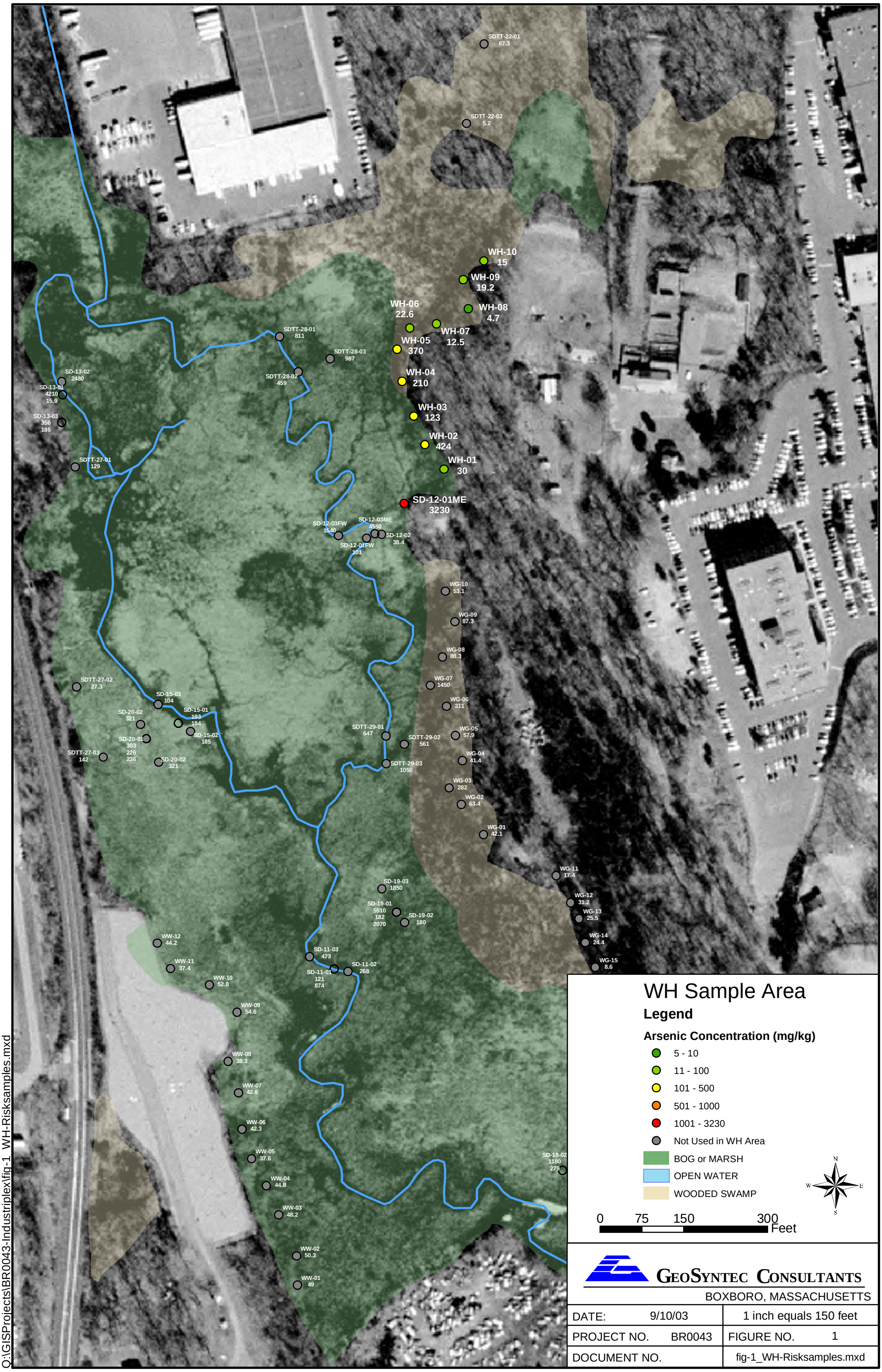
Fit tests only provide for the exclusion of a specified distribution. In addition, point source contamination areas that fit highly skewed lognormal distributions would have very significant hot spots. For example, at sites with a population CV of greater than 5, we would expect to see more than four orders of magnitude difference between the lowest and highest sample concentrations, with the data set being heavily weighted at the low end. Distributions of this sort are certainly possible, but are probably the result of a mixture of populations resulting from different sources or activities rather than a true multiplicative (dilution) process as would be the assumption for a lognormal distribution. The use of lognormal theory to develop statistical confidence intervals for such nonparametric samples is highly suspect. In such cases, the only practical method of evaluating UCL performance is through simulation. This is the approach we used in the analysis presented at the 2003 annual meeting of the Society of Toxicology (cited above). We believe that recommendations developed using this type of approach are superior to those provided by EPA's ProUCL program.

References

Mills CE, CJ Saranko, JK Tolson, SM Roberts, and KM Portier. (2003). Comparison of techniques for calculating 95% upper confidence limits (95% UCLs) on the mean. *Toxicol. Sci.* 72(S-1): 395.

USEPA (1992). *Calculating the Concentration Term: Supplemental Guidance to RAGS.* Publication EPA 9285.7-081, May 1992

USEPA (2002). *Calculating upper confidence limits for exposure point concentrations at hazardous waste sites. Supplemental guidance to RAGS,* Office of Emergency and Remedial Response, OSWER Directive 9285.6-10. December, 2002. Download at http://www.epa.gov/superfund/programs/risk/ragsa/ucl.pdf.



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