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Jerome C. Muys, Jr.
Direct Phone: (202) 373-6547
Direct Fax: (202) 424-7647
jerome.muys@bingham.com
Our File No.: 4125000055

October 19, 2006

Bingham McCutchen LLP
Suite 300
3000 K Street NW
Washington, DC
20007-5116
202.424.7500
202.424.7647 fax

bingham.com

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VIA OVERNIGHT MAIL

Ms. Anna Krasko
Remedial Project Manager
U.S. Environmental Protection Agency, Region 1
1 Congress Street, Suite 1100 (HBO)
Boston, MA 02114

Re: Centredale Manor Restoration Project Superfund Site, North Providence, Rhode Island

Dear Ms. Krasko:

On behalf of Emhart Industries, Inc. ("Emhart"), we are submitting for inclusion in the administrative record for the above-referenced site comments on the Interim-Final Baseline Human Health Risk Assessment (Aug. 2004; revised Nov. 2005) ("BHHRA"), Interim-Final Baseline Ecological Risk Assessment (Sept. 2004) ("BERA"), Interim-Final Remedial Investigation Report (Jun. 2005) ("RI Report"), and Interim-Final Preliminary Remediation Goals Report ("PRG Report") for the Centredale Manor Restoration Project Superfund Site ("Site"). Emhart respectfully requests that EPA withdraw these decision documents and correct the errors set forth therein, as discussed in this letter and in the enclosed comments.

I. Background

Atlantic Chemical Company began operations at a portion of the Site in the early 1940s. In the early 1950s, Atlantic Chemical changed its name to Metro-Atlantic, Inc. ("Metro-Atlantic"). Metro-Atlantic reportedly manufactured textile chemicals, sulfonated tallow for wool, urea-formaldehyde resin, and a metal stripper for use in the jewelry trade. For brief periods of time in the 1960s, Metro-Atlantic also reportedly manufactured trifluralin for Eli Lilly & Company and hexachlorophene for Sterling Winthrop. All of Metro-Atlantic's activities at the Site ceased upon the company's consolidation with Crown Chemical in November of 1968.

New England Container Co. ("NECC") operated an incinerator-based drum reconditioning facility on a portion of the Site from approximately 1952 until 1971. NECC received barrels from Metro Atlantic, other companies in New England, and barrel dealers. NECC reconditioned approximately 50,000 steel drums per year at the Site by

cleaning them with caustic soda solution, and/or furnacing and shotblasting with steel shot, and painting them. As part of the reconditioning process, NECC sent barrels, which normally contained chemical residues, through an oven by placing them upside down on a conveyor.

In the late-1970s and early 1980s, two apartment buildings were constructed on the Site. These buildings provide housing for the elderly.

In 1999 and 2000, the United States Environmental Protection Agency ("EPA") notified Emhart, Brook Village Associates Limited Partnership ("BVA"), Centerdale Manor Associates Limited Partnership ("CMA"), and NECC of their potential liability under CERCLA, 42 U.S.C. § 9601 *et seq.*, for environmental contamination at the Site. Since that time, Emhart, BVA, CMA and NECC have been issued, and have complied with, two Unilateral Administrative Orders and one Administrative Order on Consent, issued pursuant to CERCLA § 106, 42 U.S.C. § 9606, and other authorities, requiring them to conduct removal activities at the Site.

In May 2005, EPA lodged two proposed Consent Decrees, with BVA and CMA, with the United States District Court for the District of Rhode Island. The proposed consent decrees would release BVA and CMA from all governmental claims for response costs and natural resource damages that have been or will be incurred at the Site, and would provide contribution protection not only to the defendants, but also to their current and former general and limited partners, and their officers and directors, among others.

II. Conceptual Site Model

All the decision documents are based on an inaccurate Conceptual Site Model ("CSM"). The CSM assumes that hexachlorophene was produced at the Site by reacting materials such as 2,4,5-trichlorphenol ("TCP") and 2,4,5-trichloroaninsole. *See, e.g.*, Stephen Emsbo-Mattingly, Senior Scientist, NewFields, *Environmental Forensics Review of Persistent Chlorinated Organics, Centredale Manor Restoration Project, North Providence, Rhode Island*, dated Jun. 20, 2005 (App. E to RI Report), at 2. The CSM further assumes that the hexachlorophene manufacturing process generated dioxins, furans, and 1,2,4,5,7,8-hexachloro(9h)xanthene ("HCX"). *See id.* Finally, the CSM assumes that high variability in the manufacturing process caused the ratio of HCX to TCDD to also be variable, and, therefore, precludes the use of such ratios to identify hexachlorophene byproducts. *See id.* Therefore, HCX has been used as a "marker" for hexachlorophene byproducts. *See id.* All of the decision documents rely on the CSM. The CSM, however, is inaccurate and does not reflect actual conditions at the Site.

The hexachlorophene manufacturing process at the Site is assumed to be similar to that reported by Archer and Crone (2000). *See, e.g.*, RI Report, at 1-3, 4-1, 4-11, App. E. Archer and Crone analyzed data from the process used by the Northeastern Pharmaceutical and Chemical Company ("NEPACCO") in Missouri. *See* J. Ronald Hass, Ph. D., *Evaluation and Opinions on the Conceptual Site Model Contained in U.S. EPA's Interim-Final Remedial Investigation Report and Human Health and Ecological Risk*

Assessment Reports, dated October 19, 2006, at 1 (enclosed as Exhibit A). As explained in Dr. Hass's report, the NEPACCO process was fundamentally different from the process used by Metro-Atlantic, as described by Thomas Cleary in his patents and deposition testimony. *See* Exhibit A, at 2-7. Accordingly, the process used by Metro-Atlantic would not have created the conditions found at the Site. *See* Exhibit A, at 7-13. Rather, materials from NECC's barrel reconditioning process are the more likely source of the contamination found at the Site. *See* Exhibit A, at 16-20.

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Mr. Cleary's patents and deposition testimony were provided to EPA in February 2003. The failure to modify the CSM to reflect this information contravenes EPA guidance, which provides that "[t]he initial conceptual model should be modified to document additional source pathway and contaminant information that is collected throughout the site investigation." Office of Solid Waste and Emergency Response, U.S. Environmental Protection Agency, *Contaminated Sediment Remediation Guidance for Hazardous Waste Sites*, OSWER 9355.0-85 (Dec. 2005), at 2-7; *see also* Office of Emergency and Remedial Response, U.S. Environmental Protection Agency, *Guidance for Conducting Remedial Investigations and Feasibility Studies Under CERCLA*, OSWER Directive 9355.3-01 (Oct. 1988), at 2-3 ("It is important to note that the scope of the project and, to some extent the specific project plans, are developed iteratively (i.e., as new information is acquired or new decisions are made, data requirements are reevaluated and, if appropriate, project plans are modified).").

The use of an inaccurate CSM is a significant error that should, by itself, cause EPA to withdraw the decision documents relying on the CSM. As set forth below, however, there are additional major errors in EPA's decision documents.

III. Interim-Final Remedial Investigation (Jun. 30, 2005)

In addition to the RI Report's reliance on a flawed and inaccurate CSM, we have identified several additional significant problems with EPA's decision documents. For example, the RI Report does not address the impacts of a number of potential release and transport mechanisms, including the 1972 fire at the NECC plant, the significant reworking of site soils during construction of the Brook Village and Centredale Manor apartment buildings in 1977 and 1982, respectively, and the remediation supervised by the Rhode Island Department of Environmental Management in 1982. These activities occurred and/or were conducted prior to collection of the samples during the remedial investigation. The report should discuss the impact of these events, specifically the potential that hazardous substances were released and/or redistributed by these activities.

Further, the RI Report concludes that the Woonasquatucket River recharges the aquifer throughout the study area except in the immediate vicinity of a small groundwater mound located beneath the Brook Village parking lot. It is hypothesized that this "local groundwater high may be due to groundwater perched above a low-permeability silt lens or man-made structure." RI Report, at 3-5. This is also the area where the U.S. Geological Survey ("USGS") found indication of volatile organic compounds ("VOCs") discharging to the River in a vapor-to-water diffusion study conducted in the fall of 1999

and the area where VOCs and dioxin have been found at elevated concentrations in groundwater. The RI Report does not adequately address the collection of these data. If the groundwater mound is a reflection of perched water, then the groundwater would not be discharging to the River as indicated by the USGS study. As this area is being considered for remedial action, an alternate hypothesis needs to be developed and tested before proceeding.

Finally, the results reported for hexachloroxanthene in samples taken at the Site do not meet normal quality standards. *See* Exhibit A, at 8-13. These results, therefore, are invalid and should be disregarded.

IV. Interim-Final Baseline Human Health Risk Assessment (Nov. 2005)

AMEC Earth and Environmental, Inc. ("AMEC") has reviewed and prepared technical comments on EPA's risk assessment and preliminary remediation goal reports. AMEC's review of the BHHRA has uncovered a number of concerns, including the use and application of incorrect and invalid assumptions and approaches that have resulted in an overestimation of potential risk to human health. AMEC's *Comments on the Interim-Final Baseline Human Health Risk Assessment Report*, dated October 19, 2006, are enclosed as Exhibit B.

V. Interim-Final Baseline Ecological Risk Assessment (Sept. 2004)

According to EPA guidance, in areas where site-related impacts might be indistinguishable from non-site-related impacts, remediation to reduce ecological risks might not be necessary. U.S. Environmental Protection Agency, Solid Waste and Emergency Response, *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments, Interim Final*, OSWER 9285.7-25 (Jun. 1997), at 1-3. In this case, the portion of the Woonasquatucket River below Smithfield has been contaminated by sewage and industrial waste for over 100 years. BERA, at 2-1. The reach of the river where the Site is located is classified as an "impaired water" under Clean Water Act § 303(d) due to pathogens, cadmium, copper, lead, PCBs, mercury, dioxins, depressed dissolved oxygen and elevated nutrient levels. *Id.* Moreover, the Smithfield Wastewater Treatment Plant ("WWTP"), which is located upstream of the Site, has been issued a Rhode Island Pollutant Discharge Elimination System discharge permit to control the release of nutrients. *Id.* Discharges from the WWTP have resulted in a two- to three-fold increase in concentrations of total nitrogen and phosphorus in the Woonasquatucket River as far as Allendale Dam. *Id.* Discharges from the WWTP also increase levels of copper, lead, mercury, nickel, silver, and zinc in the River. *Id.* The BERA, however, does not address how site-related impacts can be distinguished from these non-site-related impacts.

AMEC's review of the BERA has uncovered a number of additional concerns, including some fundamental errors that necessitate withdrawal of the BERA and the preliminary remediation goals that are based on the BERA. In particular, the Executive Summary and Conclusions of the BERA state that demersal and pelagic fish populations

"may be at substantial risk of harm due to exposure to Site-related contaminants in surface water, sediment, and tissue residues." *See* BERA, at ES-14, 11-1 to 11-2. The technical portions of the BERA, however, indicate that EPA did *not* find a significant risk of harm to these receptors. *See* BERA, at 5-14, 6-4. AMEC's *Comments on the Interim-Final Baseline Ecological Risk Assessment Report*, dated October 19, 2006, are enclosed as Exhibit C.

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VI. Interim-Final Preliminary Remediation Goals Report (Nov. 2005)

AMEC's review of the PRG Report has uncovered a number of concerns, including some fundamental errors that necessitate withdrawal of the PRG Report. As discussed above, EPA mischaracterized the potential risk to demersal and pelagic fish populations in the Executive Summary and Conclusions of the BERA. This error was then carried through to the PRG Report resulting in the unnecessary calculation of PRGs for these receptors. *See* PRG Report, Part II, at 1-1 to 1-2. AMEC's *Comments on the Interim-Final Preliminary Remediation Goals Report*, dated October 19, 2006, are enclosed as Exhibit D.

* * *

Please feel free to contact us if you have any questions concerning the foregoing. We appreciate the opportunity to submit these comments.

Sincerely,

Jerome C. Muys, Jr. /LFB

Jerome C. Muys, Jr.

Enclosures

cc: Eve Vaudo, Esq.
Ms. Linda H. Biagioni
Jeffrey M. Karp, Esq.
Laura Ford Brust, Esq.

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Centredale Manor Restoration Project Superfund Site
North Providence, Rhode Island

Evaluation and Opinions on the Conceptual Site Model Contained in U.S. EPA's Interim-Final Remedial Investigation Report and Human Health and Ecological Risk Assessment Reports

J. Ronald Hass, Ph. D.
October 19, 2006

Executive Summary

The United States Environmental Protection Agency (U.S. EPA) Region 1 and the United States Army Corp of Engineers (USACE) have conducted a Remedial Investigation at the Centredale Manor Restoration Project (CMRP) Superfund Site located in North Providence, Rhode Island and portions of the Woonasquatucket River adjacent to or downstream from the Site to Dyerville Pond. The results of these efforts are contained in the Interim-Final Remedial Investigation Report (RI), issued June 30, 2005, Interim-Final Baseline Human Health Risk Assessment Report (BHHRA), issued August 2004 and revised November 2005, and Interim-Final Baseline Ecological Risk Assessment Report (BERA), issued September 2004.

In the RI, BHHRA, and BERA, U.S. EPA alleges that the CMRP Site is contaminated with 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) accompanied by hexachloroxanthene (HCX). On this basis, U.S. EPA concludes that the alleged contamination by 2,3,7,8-TCDD is a consequence of waste discharge from hexachlorophene manufacture directly into the Woonasquatucket River in the mid-1960s.

In this report it is demonstrated that these conclusions are in error. A detailed examination of the available evidence reveals the following points:

1. The hexachlorophene production process employed at the CMRP Site was incapable of producing 2,3,7,8-TCDD or HCX.

2. Any 2,3,7,8-TCDD contaminated wastes from this process would have been the result of contaminated feedstock.

3. Feedstock purification occurred prior to hexachlorophene synthesis. No HCX could conceivably accompany any waste that might have been produced during the removal of any 2,3,7,8-TCDD that might have been in the feedstock.

4. The chemical properties of the CMRP samples analyzed for 2,3,7,8-TCDD preclude the possibility that they originated from waste produced by the hexachlorophene manufacturing process at the CMRP Site.

5. The analytical methodology employed for the analysis of the CMRP samples does not provide a positive identification of HCX.

6. The analytical methodology employed for the analysis of the CMRP samples does not provide valid quantification of the material alleged to be HCX.

7. A drum recycling business that engaged in activities widely known for their potential to form 2,3,7,8-TCDD also operated on the CMRP Site.

8. The waste disposal practices of the drum recycling business operating on the CMRP Site would have resulted in 2,3,7,8-TCDD being deposited in the source area locations where contamination is reported in the RI.

9. Erosion of the 2,3,7,8-TCDD disposed by the drum recycling business is a simple and sufficient explanation for the analytical results for samples from the Woonasquatucket River.

These opinions are based upon the RI, references cited within the RI, the Administrative Record for the CMRP Superfund Site, sworn deposition testimony of individuals with first hand knowledge of the events, my personal knowledge of chemistry, and the peer-reviewed scientific literature.

Centredale Manor Restoration Project Superfund Site
North Providence, Rhode Island

Evaluation and Opinions on the Conceptual Site Model Contained in U.S. EPA's Interim-Final Remedial Investigation Report and Human Health and Ecological Risk Assessment Reports

J. Ronald Hass, Ph. D.
October 19, 2006

This document contains my evaluation and opinions regarding the Conceptual Site Model (CSM) set forth in the Interim-Final Remedial Investigation Report (RI), issued June 30, 2005, Interim-Final Baseline Human Health Risk Assessment Report (BHHRA), issued August 2004 and revised November 2005, and Interim-Final Baseline Ecological Risk Assessment Report (BERA), issued September 2004, for the Centredale Manor Restoration Project (CMRP) Superfund Site in North Providence, Rhode Island.

The CSM for the CMRP Site assumes the following:

> The historical manufacture of hexachlorophene was reported within the Source Area. It was produced by reacting of raw materials such as 2,4,5-trichlorophenol and 2,4,5-trichloroaninsole and used as an additive for antibacterial soap and cosmetics (Archer and Crone, 2000). In addition to the co-generation of dioxins and furans, the manufacture of hexachlorophene produced 1,2,4,5,7,8-Hexachloro(9h)xanthene (HCX) in widely varying amounts depending on the reaction conditions of the manufacturing process (Archer and Crone, 2000). The high variability of the manufacturing process prevented the use of source ratios, such as HCX:2,3,7,8-TCDD, for the reliable identification of hexachlorophene byproducts. Consequently, previous investigators recommended the use of detected levels of HCX to identify residues of the hexachlorophene manufacturing process (Archer and Crone, 2000).[1]

It is my opinion that the parts of the Conceptual Site Model presented in the RI relating to possible sources of 2,3,7,8-TCDD contamination are not consistent with, or supported by, the available facts.

A. <u>The hexachlorophene production process employed at the CMRP Site was incapable of producing 2,3,7,8-TCDD or HCX.</u>

Neither NewFields nor U.S. EPA cites any reference to support the assertion that the manufacture of hexachlorophene results in the formation of PCDD. The publication cited claims that 2,3,7,8-TCDD (<u>one</u> of the congeners) is formed during hexachlorophene manufacture. As discussed below, this conclusion is inaccurate with respect to the manufacturing process that reportedly occurred at the CMRP Site.

On pages 4-11 to 4-12 of the RI, U.S. EPA assumes a connection between hexachlorophene manufacture and HCX occurring along with 2,3,7,8-TCDD in a waste site. The only reference cited as a basis for that assumption is Archer and Crone.[2] That reference presents no data for samples whose origin was identified other than the Northeastern Pharmaceutical and Chemical Company (NEPACCO).

Archer and Crone cite Viswanathan and Kleopfer[3] as their basis for assuming HCX is a byproduct of hexachlorophene manufacture. Viswanathan and Kleopfer state that "[p]resumably HCX is formed through the loss of H_2O (Figure 1)."[4] No data are presented or literature cited to support that presumption. All data presented in Viswanathan and Kleopfer's papers originate with NEPACCO waste. U.S. EPA has not cited, and I have not found, any reported study that is independent of the NEPACCO operation that provides data supporting HCX as a valid marker for hexachlorophene manufacture. Nor do the published data from the NEPACCO facility demonstrate that HCX is a useful marker for any hexachlorophene process other than theirs.

In fact, the scientific basis for HCX being a marker compound for hexachlorophene is dubious. The earliest reference I have found is a citation to "Work to be published" as footnote 3 in Goethe and Wachtmeister.[5] The subject of the unpublished work was described as a search for neutral impurities such as HCX in hexachlorophene. A search of Chemical Abstracts for "hexachloroxanthene" did not find the subsequent publication or any other on that subject.

Based upon my experience as a research scientist and my observations of academic scientists, it is my opinion that it is highly unlikely the authors were successful in the search for HCX in hexachlorophene, then simply decided not to publish the work. Had they been successful in finding HCX in substantial concentrations in hexachlorophene, given that the justification for publishing the first paper[5] was to have an analytical standard for the study in reference 3 of their publication, they likely would have found a journal to publish these results. Therefore, it is more likely than not the authors were unable to find HCX in any hexachlorophene samples they analyzed.

Viswanathan and Kleopfer[3,4] assert that HCX is a byproduct of hexachlorophene manufacture without benefit of any supporting mechanistic data or, apparently, consideration of the data published by Goethe and Wachtmeister.[5] Any informed discussion of the possible formation or the utility of HCX as a marker compound for hexachlorophene manufacture should have included an accommodation of the results published by Goethe and Wachtmeister.[5] Archer and Crone[2] reference both publications[3,5] without commenting on the conflicts or referencing any subsequent publication providing experimental evidence to support the claim that HCX is an impurity in hexachlorophene. My research revealed no evidence that they recognized the differences in conditions used by Goethe and Wachtmeister and those conditions used to prepare hexachlorophene.

Nonetheless, assuming for argument's sake that HCX is a marker compound for the production of hexachlorophene, the conclusions drawn in these publications are applicable only to the extent that the production processes used by Metro-Atlantic, Inc. (MA) and NEPACCO result in similar production of PCDD and HCX.

1. <u>The manufacturing process used at the CMRP Site is fundamentally different from the NEPACCO manufacturing process.</u>

The documented process used at MA for the production of hexachlorophene is that described in Thomas Cleary's patents[6,7] and testimony.[8] MA reportedly manufactured hexachlorophene for a short time starting in approximately 1964.[9,10,11]

Mr. Cleary patented a unique process for synthesizing hexachlorophene.[6] The typical procedure at the time involved adding exactly two moles of 2,4,5-trichlorophenol (2,4,5-TCP) to exactly one mole of formaldehyde or equivalent, and then heating, under pressure, in the presence of strong acid. Mr. Cleary developed a stepwise procedure that he claims to be more

easily controlled and less dependent upon the accuracy of the reaction mixture.[6] That process involved the condensation of formaldehyde with 2,4,5-TCP in strongly acidic conditions using temperatures at or below the boiling point of perchloroethylene (PERC). That is, a temperature ≤ 121 °C.

Mr. Cleary testified that he licensed this technology to MA and performed demonstrations to assist in starting up the process.[12] The diagram below was developed using his description of the process.[8]

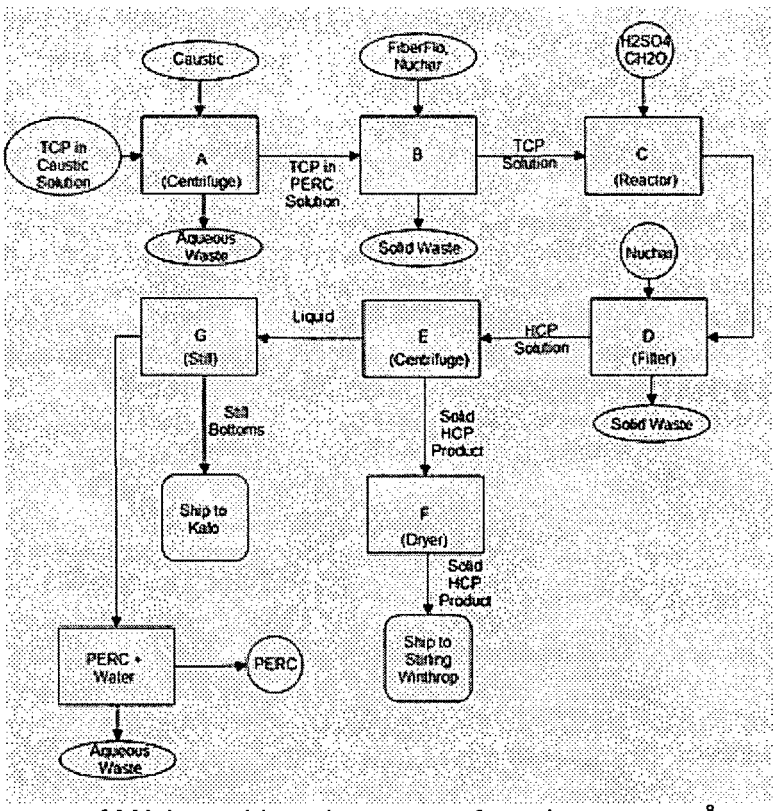

Figure 1: Flow diagram of MA hexachlorophene manufacturing process.[8]

NEPACCO produced 2,4,5-TCP and hexachlorophene between early 1970 and the end of 1971 in a facility that had previously been used to make 2,4,5-TCP and 2,4,5-trichlorophenoxyacetic acid.[13,14] Figure 2 presents a comparison of the relevant points between the Metro-Atlantic and NEPACCO processes to manufacture hexachlorophene.

	Feedstock Prep	Synthesis	Purification
Metro-Atlantic	Purchased TCP, Activated carbon TCDD trapped	Strong acid Step wise condensation	Activated carbon Acid to neutral pH
NEPACCO	Manufactured TCP Purification by distillation TCDD in still bottoms	Strong acid Concerted condensation	Strong basic or basic extraction Hexachloroxanthene byproduct

Figure 2. Comparison of Metro-Atlantic and NEPACCO hexachlorophene production processes.

For purposes of this report, I will describe the hexachlorophene production process to be composed of the following three phases: feedstock preparation, hexachlorophene synthesis, and hexachlorophene purification.

a. Feedstock Preparation

Mr. Cleary testified that one of the critical parameters for obtaining high quality hexachlorophene is the purity of the 2,4,5-TCP.[15] At his deposition, he presented a document that he testified is the bill of materials used by MA for purifying the 2,4,5-TCP (Steps A & B).[16] One of the ingredients on the list is Nuchar, an activated carbon.[17] Mr. Cleary testified that the TCP used by MA was simply the reactor contents from Diamond Alkali, which was a brownish caustic solution with no solids. The purification procedure implicit with the supplied bill of materials and Mr. Cleary's testimony is a classical recrystallization followed by dissolution of the TCP in PERC, with that solution being purified with activated charcoal. The 2,3,7,8-TCDD in the original solution would have followed the TCP through the recrystallization procedure until the activated charcoal was added. Essentially all the 2,3,7,8-TCDD would have been removed by the activated charcoal.

2,3,7,8-TCDD along with the other PCDDs/PCDFs has very limited water solubility and a high affinity for carbon. As examples, activated charcoal is the U.S. EPA's "Best Available Technology" recommendation for the removal of these compounds from drinking or wastewater.[18] The International Occupational Safety and Health Information Centre recommends drinking a slurry of activated charcoal as the first aid treatment in the event of ingestion of 2,3,7,8-TCDD.[19] Whiteside records the successful use of activated carbon by the Air Force as means to remove 2,3,7,8-TCDD from Agent Orange.[20]

The octanol:water partitioning coefficient (k_{ow}) is the ratio of the subject compound's concentration in octanol to its concentration in water when the system is at equilibrium. Since the ratio can be large, it is customary to discuss the logarithm of the ratio or log k_{ow}.

The larger the value of log k_{ow}, the more likely the compound is to leave an aqueous environment. The log k_{ow} for 2,3,7,8-TCDD is 6.8.[18] This means that the concentration of 2,3,7,8-TCDD in water saturated octanol will be approximately 6,300,000 times higher than its concentration in the octanol saturated water.

Activated charcoal has a higher affinity for 2,3,7,8-TCDD than does octanol, so the partitioning coefficient between an aqueous phase and charcoal will be even larger. The increased sorptive capacities of pyrogenic derived carbons (such as soot or charcoal) are estimated to be 100 to 1000 fold greater than soil organic matter.[21] Thus, we can estimate the distribution coefficient for 2,3,7,8-TCDD and charcoal, such as used in the MA process, to be on the order of 10^8 or 10^9.

What this means for the MA hexachlorophene process is that the 2,3,7,8-TCDD concentration in the 2,4,5-TCP feedstock would be reduced by more than 1/630,000,000 during the initial purification (Steps A and B). Making a worst case assumption that the 2,4,5-TCP has an initial concentration of 50 ppm (0.000050 $g_{TCDD}/g_{Solution}$), this means the purified 2,4,5-TCP after the PERC filtration step would have a 2,3,7,8-TCDD concentration much less than 0.5 parts per trillion (0.0000000000005 $g_{TCDD}/g_{Solution}$). The 2,3,7,8-TCDD and any PCDD/PCDF originally in the 2,4,5-TCP delivered to the MA facility would have been adsorbed on the activated charcoal and thus removed from the 2,4,5-TCP.

I have no reason to believe that the management of MA were aware of the presence of 2,3,7,8-TCDD when they used 2,4,5-TCP in the production of hexachlorophene or to know that forty years later the purification procedures they employed would still be recommended as the "Best Available Technology" for removal of 2,3,7,8-TCDD from aqueous media. The point is that the procedures they employed, whatever the reason, were effective at removing the 2,3,7,8-TCDD from the production material.

The feedstock preparation phase at NEPACCO formed copious quantities of 2,3,7,8-TCDD, which would have been in their waste stream. In contrast, any 2,3,7,8-TCDD that might have been present as a trace impurity in the 2,4,5-TCP purchased by Metro-Atlantic would have been tightly bound to activated carbon.

b. Hexachlorophene Synthesis

Mr. Cleary testified that the next step (Step C, hexachlorophene synthesis) was performed using strong acid.[22] The reported conditions for the formation of 2,3,7,8-TCDD from 2,4,5-TCP in aqueous media all require basic conditions. [a,23,24]

My research has revealed no publication that provides data to demonstrate that 2,4,5-TCP reacts in condensed media (i.e., a solution) to form TCDD under acidic conditions. There is substantial patent literature which contains claims about dioxin contamination reduced below the limit of detection in the production of chlorophenols when acidic conditions are employed and/or a Lewis acid is present.[25,26]

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[a] The initial purification of the 2,4,5-TCP was conducted under basic (or caustic) conditions so that the compound was present as the trichlorophenate anion (2,4,5-TCPhenate), and thus would be highly soluble in water. The initial step in the purification was to add an excess of caustic. The solution was then most likely cooled, causing 2,4,5-TCPhenate to crystallize. Any other insoluble solids, including the 2,3,7,8-TCDD that might have been present, would be collected with the 2,4,5-TCPhenate.

After washing the crystals with caustic, the 2,4,5-TCPhenate was most likely dissolved in dilute caustic, possibly the material listed as caustic (wash) in the ZEP recipe. There is no indication of any solids being present or the material being filtered at this point.

The 2,4,5-TCP was next acidified with sulfuric acid. This would cause the 2,4,5-TCPhenate to precipitate as 2,4,5-TCP in the trichlorophenol form. The 2,3,7,8-TCDD would accompany the 2,4,5-TCP. The crystals were washed with sulfuric acid.

The 2,4,5-TCP was next dissolved in PERC. The 2,3,7,8-TCDD would readily dissolve in this solvent. The PERC solution was treated with activated charcoal and filtered. At this point the 2,3,7,8-TCDD would bind tightly to the activated charcoal and be separated from the 2,4,5-TCP when the solids were removed.

In this manner, any 2,3,7,8-TCDD contaminated waste resulting from the MA hexachlorophene manufacture would be associated with activated charcoal. Thus, the presence or absence of activated charcoal with 2,3,7,8-TCDD provides a convenient marker to establish whether the 2,3,7,8-TCDD originated with MA.

The purification steps were performed at a temperature far below those encountered in the preparation of 2,4,5-TCP. The pH is within the range in which 2,3,7,8-TCDD can be formed. The reaction rate depends upon temperature and would be so low at the temperatures used for the purification that 2,3,7,8-TCDD formation would have been negligible. Any TCDD that did form would have been adsorbed to the 2,4,5-TCPhenate until it was trapped by the carbon and thus would not be present in the waste water.

Publications describing the formation of dioxins from chlorophenols require strongly basic conditions and prolonged elevated temperatures.[27,28,29]

My research revealed no credible report of 2,3,7,8-TCDD being formed in an acidic solution. Mr. Cleary testified that no 2,3,7,8-TCDD or any other dioxin would be formed in the MA hexachlorophene process.[30] Thus, no additional 2,3,7,8-TCDD would be added to the reaction mixture during the synthesis of hexachlorophene.

Based upon the chemical data published by Goethe and Wachtmeister[5] and the processes described in Cleary's patent and the patents issued to the officers of NEPACCO,[6,7,31,32,33] HCX could not have been produced by either company's hexachlorophene synthesis process.

Thus, no 2,3,7,8-TCDD or HCX could have been formed in the process used by NEPACCO or by MA for hexachlorophene synthesis. However, as discussed above, 2,3,7,8-TCDD was generated by NEPACCO during the feedstock preparation step.

c. Hexachlorophene Purification

The hot reaction mixture was filtered with activated charcoal (Step D). As discussed for Step A above, had there been any residual 2,3,7,8-TCDD or PCDD/PCDF, its concentration would have been reduced by approximately a billion-fold or more through adsorption on the activated charcoal.

The reaction mixture was next cooled and centrifuged (Step E). The purified, crystalline product was dried and shipped. No 2,3,7,8-TCDD or PCDD/PCDF would have entered the CMRP Site through these actions.

The supernatant solution or "mother liquor" from the recrystallization was co-distilled using water (Step G) to recover the PERC that would have resulted in a biphasic liquid. The PERC would have been drained from the receiving container. Had there been any 2,3,7,8-TCDD or PCDD/PCDF present in the "mother liquor," it would have remained in the still as "still bottoms" that were sold.

The purification process described in the initial NEPACCO patents uses highly basic conditions. The initial NEPACCO patent refers to the heating of hexachlorophene with an aqueous mixture containing 5% sodium hydroxide and 2% sodium carbonate to give a solution with pH of about 13.5 (strongly basic conditions) as part of the purification method.[33] The same purification method is described in their patents for the preparation of hexachlorophene filed in June 1967, with completed specification in August 1970, and November 1969, with completed specification in March 1973.[31,32] This purification procedure is similar to the reaction conditions reported by Goethe and Wachtmeister for the synthesis of HCX from hexachlorophene and is likely the purification procedure initially in use at the NEPACCO facility.

The principals of NEPACCO were issued an additional patent on the purification of hexachlorophene. The completed specification for that patent was submitted in June 1971.[33] They reiterate the use of basic conditions for hexachlorophene purification in their "prior art" discussion.

The new patent states a procedure that forms an ammonium salt of hexachlorophene.[33] Their new purification process still involves basic conditions and likely resulted in HCX production.

Considerable discussion has occurred concerning the high levels of TCDD found in the NEPACCO "still bottoms." My research of the relevant patents,[31,32,33] Record of Decision,[14] and other literature[2,3] revealed nothing to indicate whether the NEPACCO process transferred the 2,4,5-TCP to a separate reactor for the hexachlorophene production or simply continued in the same vessel for the next step in the process. The record does indicate the "still bottoms" were removed after 2 to 4 batches of product were made.[14] This detail should have a profound effect on the relative concentration of impurities strictly from 2,4,5-TCP production and those strictly from hexachlorophene production/purification in any particular portion of the NEPACCO waste.

2. <u>The sampling results do not conform to the hexachlorophene manufacturing process used at the CMRP Site.</u>

Hexachlorophene manufacture is reported to be accompanied by 2,3,7,8-TCDD and HCX as "marker" compounds.[2,3,4,34] Both of these compounds are alleged to be found at the CMRP Site.[35] Accordingly, one might conclude that the hexachlorophene manufacture by MA contributed to the 2,3,7,8-TCDD contamination found at the CMRP Site. However, an examination of the record evidence and the scientific literature reveals that neither 2,3,7,8-TCDD nor HCX would be produced by the hexachlorophene production procedures employed by MA.

The NEPACCO hexachlorophene plant in Missouri was operational in the early 1970s. The NEPACCO plant manufactured its own 2,4,5-TCP feedstock. The papers published based upon the characterization of their waste report finding 2,3,7,8-TCDD and frequently make an encompassing statement describing TCDD as an impurity associated with hexachlorophene production. While 2,3,7,8-TCDD and HCX were both found in the NEPACCO waste, this finding does not establish that either impurity came from the preparation of hexachlorophene since NEPACCO also manufactured 2,4,5-TCP and used strongly basic conditions to purify their hexachlorophene after manufacture.

a. <u>Dioxin</u>

The statement that hexachlorophene manufacture forms dioxin describes a correlation, not a causal relationship. Some papers list 2,3,7,8-TCDD as part of a hexachlorophene waste stream. My research revealed no example in which any data were presented to demonstrate that 2,3,7,8-TCDD is actually formed during the hexachlorophene production processes described in the patent literature. Rather, the 2,3,7,8-TCDD, when present, is best explained as an impurity in the 2,4,5-TCP feedstock and/or a byproduct of 2,4,5-TCP manufacture. The extremely high levels of 2,3,7,8-TCDD reported in locations such as Times Beach resulted from a company that produced both 2,4,5-TCP and hexachlorophene at the same site and almost certainly co-mingled the waste streams. Once the waste streams have been mixed, it is no longer possible to determine where in the process any specific component was produced. Thus, a specific component will correlate with any step in the production process whether or not it is caused by that step.

Therefore, the assumption that 2,3,7,8-TCDD and/or PCDD/PCDF were generated through hexachlorophene manufacture by MA at the CMRP Site is inaccurate. It is my opinion that the 2,3,7,8-TCDD found at Times Beach and related sites was a result of the manufacture of 2,4,5-TCP, not hexachlorophene manufacture. Metro-Atlantic did not manufacture 2,4,5-TCP.

The presence of 2,3,7,8-TCDD as a byproduct of the manufacture of 2,4,5-TCP by the treatment of 1,2,4,5-tetrachlorobenzene with a strong base and at elevated temperatures is well

known.[36,37] Any 2,3,7,8-TCDD or PCDD/PCDF contaminating the 2,4,5-TCP used at Metro-Atlantic for hexachlorophene production would have been removed in the initial purification step of their process by tight binding (under the conditions present) to activated charcoal.[38,39,40] The solid material was disposed by removal from the Site by a waste hauler.[41,42] The analytical data presented are inconsistent with the resulting activated charcoal filter cake being disposed on the CMRP Site, the Woonasquatucket River, or Allendale Pond.

b. HCX

The hexachlorophene production method described in Cleary's patent utilizes acidic conditions for product preparation and purification.[6] Therefore, HCX is unlikely to be formed in the hexachlorophene process used by MA and hence would not have been associated with their hexachlorophene production waste.

Goethe and Wachtmeister, in their paper describing the synthesis of HCX, report that prolonged treatment of hexachlorophene with a strong acid (polyphosphoric) at an elevated temperature (220 °C) resulted in a complex mixture containing only small amounts of neutral products (such as the targeted HCX, as they note) whereas reaction in hot diglyme (135 to 140 °C bath temperature) with p-toluenesulphonyl chloride and excess sodium hydroxide resulted in a 77% yield of 1,2,4,5,7,8-hexachloroxanthene after a total of 28 hours, of which the majority was in a refrigerator to crystallize the product.[5] Note that no HCX was reported under acidic conditions, such as those used in the MA hexachlorophene process, even at temperatures substantially in excess of those employed at MA,[6,7] whereas a high yield was obtained under basic conditions, such as those used in the NEPACCO hexachlorophene purification process.[31,32,33]

HCX is a likely byproduct of the "purification" process described in the NEPACCO patents[31,32,33] and so is likely to be a component of their hexachlorophene waste stream. The Metro-Atlantic process used acidic conditions for hexachlorophene purification and would not have formed HCX. Thus, HCX could not have been in the Metro-Atlantic waste stream.

Cleary's patent describes a product purification based upon active charcoal under acidic conditions in contrast to the treatment with a strong base described in the NEPACCO patents. Given the similar chemical properties of HCX and dioxins,[4] there is no reason to believe that had any HCX been formed during the MA hexachlorophene synthesis, it would have escaped capture by the active charcoal. There is no basis to support the assumption that HCX could have been a component of the MA hexachlorophene waste stream.

Moreover, in my opinion, the data presented do not constitute adequate proof of the presence of HCX in the CMRP sample analyses reported in the RI, BHHRA, and BERA. The quantitative analyses reported for HCX in the CMRP samples do not meet normal quality standards. Therefore, the HCX results are invalid and must be disregarded.

None of the projects were designed to prove the presence of HCX in any of the samples. The Quality Assurance Project Plan (QAPP) for the work performed between 1998 and 2000 clearly states that HCX is a Tentatively Identified Compound (TIC).[43] The QAPP specifies the analyses be performed, even though no standard material is available to calibrate the instrument response, establish chromatography conditions, or even establish that HCX would be recovered by the sample preparation procedures used for the CMRP analyses.

The minimum requirements for the identification of a chemical in an environmental sample include substantial chemical properties such as solubility, acid/base behavior, dipole moment,

and polarizability. These determine the behavior of the chemical in the sample extraction and clean up procedures. In the absence of a valid standard reference material, it is impossible to know the efficacy of the extraction process or whether the sample preparation procedures even recover the target analyte.

In addition, it is normal to require the putative target compound to elute within a narrow relative retention time window established through the measurement of the same target compound under identical conditions for a standard reference compound. For the results reported prior to 2001, these conditions were not met on any of the analyses performed. No reporting laboratory had the benefit of a reference standard. As a consequence, no reporting laboratory had the means to know the recovery, if any, of the targeted HCX, or whether the measured signals even originated from HCX. Simply stated, there is no valid means to know whether or not HCX is present in any sample reported in this study, much less any idea of the quantity present.

The QAPP for the studies performed after 2002 does not include the minimum set of quality control measurements to establish data quality for HCX measurements.[44] The validation reports for these more recent studies clearly state that the purpose for including the HCX in the analysis was to evaluate its performance in Method 1613B.[45] The stated purpose of the measurements is to answer a research question, not to support a regulatory action. As such, the results are neither expected to nor actually do meet the standards necessary to support regulatory decisions.

The available data do not prove that HCX is present in any samples from the CMRP Site. The material reported as HCX in the earlier analyses did not meet basic requirements for target compound identification. The laboratories were instructed to report the response for masses that are nearly ½ a peak width different in spectral position than the monitored mass[b] at a specified relative retention time[c] compared to one of the Method 8290 internal standards without measuring that relative retention time themselves.[46]

As a specific example of typical requirements, the World Anti-Doping Agency (WADA) requires that the retention time for a candidate chemical on a capillary column equipped gas chromatograph match a contemporaneously analyzed reference substance within the smaller of ± 1% or 0.2 minutes retention time. In addition, the relative intensity of three "diagnostic" peaks must maximize together with relative intensity within ± 10% for ions > 50% relative abundance in

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[b] The mass spectrometer was not adjusted to the proper mass/charge ratio for the ions alleged to be HCX. This "misalignment" will have the effect of increasing the likelihood of some material with a different elemental composition than HCX providing the measured signals. New England Container Company recycled drums containing residual dyes. Xanthene based dyes were in widespread use during the period of NECC's operations at the CMRP. Little is known about the possible products of incomplete combustion that are formed when xanthene is burned in the presence of chlorine or chloride sources (for example, polyvinylchloride). The risk is that one of these products will have an elemental composition that gives an exact mass such that it provides the response alleged to arise from HCX. The authors should at least provide a profile of the peaks alleged to be HCX to ascertain whether they even have the correct exact mass to support their structural assignment.

[c] Retention indices are the proper means to transfer relative retention times for two compounds of differing class between chromatographic instruments. The quantity referred to as "relative retention time" in the HCX analyses is actually the same as α, the separation factor. It is intended to provide a measure of the difference between the centers of two peaks as part of the information to estimate the degree of separation. α is not intended to be an identification criterion for a material in the absence of a reference standard.

the mass spectrum or ± 20% for ions between 25% and 50% relative abundance in the mass spectrum.[47] The laboratory must use a reference material to meet these requirements. The earlier results produced for the CMRP samples did not have the required standard. As discussed below, the material alleged to be a HCX standard used in the later studies had sufficient issues that it is unlikely it actually was HCX. In their discussion of the problems with the alleged HCX standard, the authors presented no evidence other than the bottle label that the material was as claimed. Given the problems discussed by the authors, they should have verified structure by a means independent of GC/MS.

WADA requires the monitoring of three ions, not two ions as provided in the CMRP analyses. Although U.S. EPA lists similar criteria for the positive identification of target analytes in Methods 8280, 8290, and 1613 (monitoring of two ions, matching GC retention times with a standard), the authors provide no justification for adopting less stringent identification criteria for HCX. If the presence of HCX is intended to establish potential sources of 2,3,7,8-TCDD contamination, applying less stringent identification criteria for the HCX identification is completely unjustified and irresponsible, in my opinion.

The material alleged to be HCX in the BHHRA and BERA studies apparently does not have the same properties as HCX. As an example, the Third Party Data Validation Report for the ELS Tissue has a QA/QC discussion for QC Batch 48426-36.[48] On page 2 of the QA/QC discussion for Batch 48426-36, it is stated:

> A standard containing HCX and TCX was analyzed by HRMS in the selected-ion monitoring (SIM) mode using masses of TCDD to monitor TCX and masses of HxCDD to monitor HCX. Because the response for TCX and HCX using these masses was low, the standard was reanalyzed in the full scan mode in an attempt to identify masses of greater intensity and to confirm retention times. The masses determined from the full scan analysis were added to the acquisition program and, along with the TCDD and HxCDD ions, were used to monitor TCX and HCX in the subsequent sample runs. Reanalysis of the TCX/HCX standard during these runs showed that neither HCX nor TCX was detected at the masses identified in the full-scan analysis, but they were detected using the HxCDD masses and the TCDD masses, respectively. The sensitivity for TCX at the TCDD masses dropped significantly from the initial SIM analysis to the second SIM analysis acquired during the sample runs. Because of the low response for this TCX standard, the QAPP assumption that a response factor of 1 could be used to calculate TCX concentrations in absence of a calibration curve may be inappropriate. A very small peak detected in the TCDD masses at the TCX retention time appeared in all samples including blanks. In many instances the ion ratio was outside the expected range. Because this peak could not be positively identified as TCX and because the TCX standard response was low, estimated maximum possible concentrations for this possible TCX peak are not reported. No 1,2,3,7,8,9-HCX was found in the samples; however, a number of samples had a HCX response with the correct ion ratio, but different retention time from 1,2,3,7,8,9-HCX. This may indicate presence of a different HCX isomer. There are no concentrations reported for this possible HCX isomer since it is not the target isomer; however, the peak is flagged on the chromatograms included in the hard-copy of the data package.

The authors seem not to realize that the SIM (Selected Ion Monitoring) experiment would lead to an approximately 100-fold improvement in signal:noise ratio compared to the full scan analysis if they had properly entered the correct exact masses into the SIM descriptor.

The point can be readily understood by inspection of the peak positions and shapes for HCX and the HxCDD (hexachlorodibenzo-p-dioxin) ions discussed above:

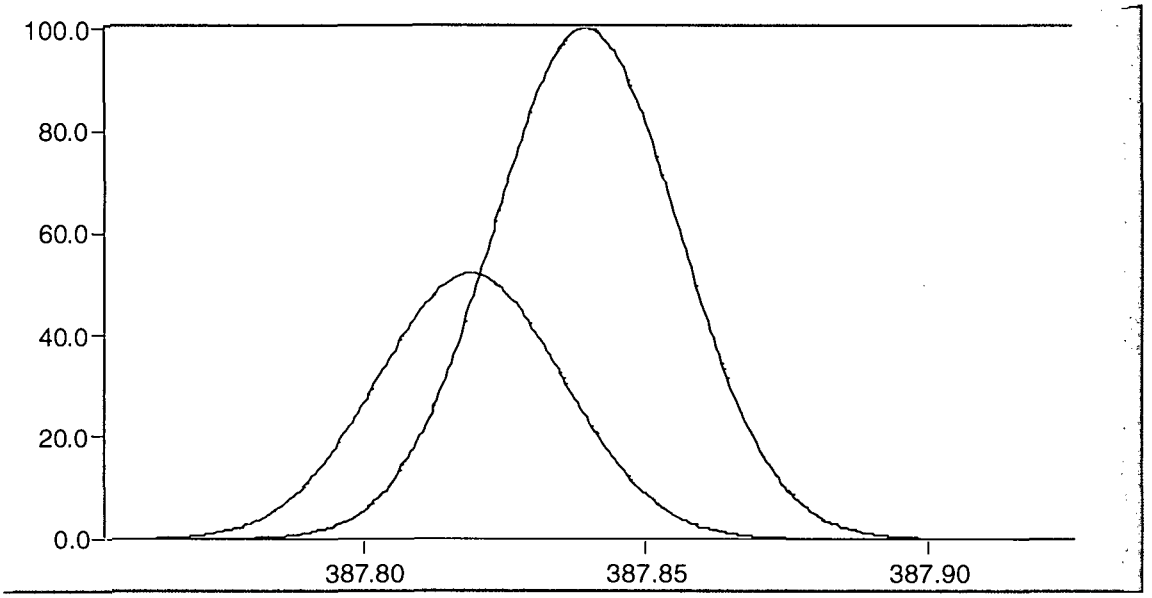

Figure 3: Plots of HCX (Red) and HxCDD (Blue) ions' intensity vs. mass/charge ratio at nominal m/z = 388 assuming a Gaussian peak shape and a resolving power of 10,000.

The peaks are poorly resolved so that monitoring for HxCDD would result in interference from HCX, as is reported. The concern is raised from the scanned mass spectrum that resulted in ion m/z's that failed to give a response in the subsequent selected ion monitoring experiment. I am aware of no explanation other than an error in the mass measurement or an error in editing the SIM descriptor to explain the authors' observation. Did the measured m/z agree with the calculated m/z for the elemental composition of HCX or not? If so, HCX should have been observed in the SIM experiment. If not, the compound is <u>not</u> HCX. Properly operated, the required mass measurement accuracy is well within the specifications of the equipment used for these measurements. The appropriate action at this point would have been to find and correct the problem with the mass measurements and/or structural assignment for the HCX reference standard.

It appears the authors performed a mass measurement on their HCX standard and found a value inconsistent with its elemental composition. If this occurred, the authors should have realized their standard was most likely misidentified. Accordingly, the standard should not have been used until the problem was understood and resolved.

The observation of a response in the samples for the alleged HCX at a different retention time than their standard is presented without comment. Is HCX such a widely distributed family of compounds that the occurrence of isomers other than the 1,2,4,5,7,8-HCX is an expected event? What is the source of these other signals? This observation raises questions that should have been addressed as part of a human health risk assessment.

It is much more likely than not that some entirely different compound than HCX is present, leading to the observed response. In fact, there are more than 40 chemically possible elemental compositions containing six chlorine atoms along with carbon, hydrogen, nitrogen, and oxygen within the resolution window of a mass spectrometer operated at 10,000 resolution at m/z 387.82. Each of these elemental compositions could result in one or more of hundreds of different chemical structures. Adding sulfur to the allowed compositions increases the number to approximately 100. Allowing the number of chlorines to vary between five and seven results in more than 200 possible elemental compositions being possible. In short, there are many thousands of compounds other than HCX that could give the observed results. Given the shortcomings described above, there is no way to know what chemical compound(s) are actually present in the CMRP samples.

The reported poor chromatography is a significant concern. The question of whether the chromatographic conditions would have resolved interfering compounds was not addressed. In their reports, the authors note that the column used does not resolve all TCDF (tetrachlorodibenzofuran) isomers from the 2,3,7,8-TCDF. This potential interference must be dealt with by analyzing any samples found to be positive on a different GC (Gas Chromatography) column. The authors should have obtained samples of all reasonable interferences (for example, all isomeric hexachloroxanthenes at an absolute minimum) and demonstrated which ones would not interfere. One could very reasonably ask, how do the authors know they are not looking at methylhexachlorodibenzofurans, chloromethylpentachlorodibenzofurans, or, dichloromethyltetrachlorodibenzofurans, which would have the same exact mass and similar chromatographic properties? There is no basis for calling the compound HCX until some of these other possibilities have been eliminated.

Other workers do not report similar chromatography issues. Archer and Crone report no chromatography issues with HCX.[2] Their paper includes a chromatogram showing HCX with essentially the same peak shape as the HxCDDs and HxCDFs (hexachlorodibenzofurans). Viswanathan et al. report on the measurement of HCX in samples derived from the NEPACCO hexachlorophene process without commenting on any chromatography issues.[3,4] Similarly, Freeman et al. report independent analyses of samples collected along a roadway for HCX with no reports of chromatography issues.[49] The data reported by the HCX standard vendor does not exhibit any evidence of poor chromatography.[50] This observation is especially noteworthy because the HCX standard is the same batch as was used in the CMRP sample analyses. Either the chromatographic system used by the authors was defective or their reference compound was not HCX. They should have determined which was the case and taken appropriate corrective action rather than continuing to analyze samples. In the absence of these actions to address the question concerning their standard, instrument, or both, all HCX measurements are invalid.

The Food and Drug Administration (FDA), for example, notes that: "A reference standard is a highly purified compound that is well characterized. Chromatographic methods rely heavily on a reference standard to provide accurate data. Therefore, the quality and purity of a reference standard is very important."[51] Other than any claim made by the vendor, the authors have presented no evidence supporting the assumed structure of their reference "HCX." These missing data are particularly troublesome in view of the report of stability problems with the same compound from the only vendor offering the compound[34] that are inconsistent with the published literature where no unusual stability problems are reported.[2,3,4,49]

Given all the issues the authors report with their attempts to use the HCX reference compound, my opinion is they have clearly demonstrated their reference compound has failed to meet either of the criteria discussed by FDA.

In addition to it being unlikely that the observed signals are coming from HCX, the RI, BHHRA and BERA provide no measure of accuracy for the HCX measurements. Other quality control measurements for the HCX analyses are erratic.

The Third Party Data Reviewers flagged all HCX results as estimated values ("J" flag). In my opinion, they were overly generous in their assessment of data quality given the gravity of issues they identified. Specifically, they cited non-compliant initial calibration and poor chromatography. Either issue should have resulted in the 2,3,7,8-TCDD results being rejected based upon the criteria described in Method 1613B. In my opinion, the HCX data should be rejected for failing to meet method criteria.

Further insight into the initial calibration issues is apparent from the literature. Tracy et al. report a modification of Method 1613B to allow for simultaneous measurement of HCX along with the usual PCDD/PCDF.[34] In addition to the stability problems discussed above, they were unable to obtain a linear calibration curve. The non-linearity is so severe that a fourth order polynomial is required to fit the data. The authors report dividing the calibration into two regions and assume each to be linear. They provide no justification for their action other than that it provides a means to get the percent relative standard deviation for the response factors below 50%. The response factors are clearly functions of concentration in both "linear" regions. That is, their "calibration" curve is made up of two discontinuous, non-linear functions. Stated simply, they have no initial calibration curve. Archer and Crone do not report any issues with linearity in their HCX calibration curve covering a factor of 100 in concentration.[2] Dr. Tracy is listed in Quality Assurance Project Plans (QAPPs) as the person responsible for the HCX analyses.[52]

My research revealed no evidence of a continuing calibration specification or any effort to demonstrate continuing calibration for HCX. Failure to demonstrate instrument calibration at the time of measurement is an adequate reason to reject any set of data. Data generated by uncalibrated instruments are invalid and are unsuitable for decision support.

Therefore, all HCX results must be disregarded. Accordingly, there is no basis to support U.S. EPA's contention in the RI that the alleged contamination by 2,3,7,8-TCDD on the CMRP Site is a consequence of waste discharge from the hexachlorophene manufacturing operation into the Woonasquatucket River in the mid-1960s.

B. <u>There is no evidence that furans are byproducts of hexachlorophene production.</u>

Neither NewFields nor U.S. EPA cite any publication or data that support the claim that furans (PCDF) are byproducts of hexachlorophene production or are related to such activity in any way. My research did not identify any publication that supports this assumption.

It is my opinion that no chemically plausible reaction mechanism exists that predicts PCDF formation under the conditions described for hexachlorophene manufacture. Dioxins (PCDD) and PCDF are widely reported as combustion byproducts from burning chlorinated organic compounds and/or organic compounds in the presence of chlorine, such as are described for the New England Container Company (NECC) drum recycling facility located on the CMRP Site.[53,54,55]

C. There is no evidence of "high variability" in the manufacturing process used at the CMRP Site.

The authors, along with the cited literature, attribute variations in the HCX:TCDD ratios to being a consequence in the variations in the manufacturing processes. Since there is no documented change in the chemical basis for hexachlorophene synthesis in either process,[6,7,31,32,33] the remaining possibilities are reaction conditions such as variations in feedstock loading, temperature, pressure and reaction time. The authors present no data or literature reference to support their assumption that conceivable variations in the reaction conditions could lead to the variation in impurity concentration ratios observed at the CMRP Site.

As noted above, HCX and TCDD each require similar strongly basic conditions to form from the available reactants. The rates of their formation reactions would both increase or decrease in unison with increases or deceases in temperature or pressure, respectively. Both are minor constituents of the overall reaction mixture so that their concentrations would not be affected by likely variations in the amounts of starting materials. Therefore, it is more likely than not that any change in conditions that affected one would affect the other in a similar manner so that their ratio would not change drastically. The HCX:TCDD ratio at the CMRP Site spans the range of 0.93 to 57. U.S. EPA presents no evidence that ordinary variation in the production process described in Cleary's patents could lead to such drastic changes in these impurity ratios.

My research revealed no evidence in the administrative record, Mr. Cleary's testimony, or former employee depositions that MA changed its hexachlorophene production process from the one described by Mr. Cleary. A more plausible explanation for the high variability of the 2,3,7,8-TCDD and HCX ratios, if real, is that these compounds were introduced to the Site *via* independent means, not as byproducts of a single process. U.S. EPA invokes a similar explanation for the poor correlation between PCB and dioxin TEQ (essentially TCDD concentration) at the CMRP Site.[56]

The observed variations in the TCDD:HCX ratio in waste generated by NEPACCO are more likely a result of variations in their waste management procedures than in their hexachlorophene manufacturing process.

D. The sampling results do not support the conclusion that dioxins and furans generated as hexachlorophene byproducts were discharged directly into the Woonasquatucket River and/or the Allendale Pond.

The Environmental Forensics Report hypothesizes a CSM in which hexachlorophene waste is deposited directly into Allendale Pond.[57] Page 4-12 of the RI substitutes "Woonasquatucket River" for "Allendale Pond" without explanation or justification. Putting aside the issues relating to TCDD and HCX formation or presence in any sample, it is my opinion that there is not adequate support for the assumption that either the TCDD or the HCX contamination found at the CMRP Site originated from hexachlorophene production waste being dumped into the Woonasquatucket River or Allendale Pond as hypothesized in the Interim Final Reports.

Based upon the maps in the RI, Allendale Pond is 500 to 2000 feet downstream from the site of the hexachlorophene production, depending upon location within the pond. The river is reported to be slow-moving much of the time through that area, especially in the winter, according to reports in the administrative record.

Table C-1 reports the Source Area sample with highest HCX concentration to be SD-30. According to Figure 3A of the Final Summary Report for the Expanded Site Inspection, dated March 9, 1999, Sample SD-30 comes from the tail race area below the NECC drum reconditioning facility and on the opposite side of the CMRP Site from the hexachlorophene production location and the Woonasquatucket River. Samples CMS-4104 through CMS-4111 also come from the tailrace area and are high in 2,3,7,8-TCDD and HCX. U.S. EPA offers no explanation for how the 2,3,7,8-TCDD and HCX were transported from the Woonasquatucket River and/or Allendale Pond to the tailrace area.

It is noteworthy that the same sample (SD-30) is listed as having the highest reported values of 2,3,7,8-TCDF and 1,2,3,7,8-PeCDF in the Source Area. High furan levels are consistent with a combustion source for the contamination, which, as discussed below, occurred in NECC's barrel reconditioning process conducted at the CMRP Site.

U.S. EPA provides no reference in the RI to support its contention that little settling of the alleged waste discharge occurred until after the "discharge" passed downstream of the highly contaminated area at the southern end of the property after which the majority of the "discharge" settled to the bottom.

The rather sparse data available for the Woonasquatucket River between the site of the hexachlorophene manufacturing building and Allendale Pond has one sample (SD-27) that was taken adjacent to Cap Area #2 with a 2,3,7,8-TCDD concentration just above the action level (1.3 ppb reported vs. 1.0 ppb for the action level) and a sample (SD-26) downstream of Sample SD-27 but upstream of the pond with low 2,3,7,8-TCDD levels (0.092 ppb, less than1/10 the action level). U.S. EPA provides no explanation for the contaminants being deposited at one location but not the other as would be required if their assumption of dumping 2,3,7,8-TCDD contaminated waste directly into the river were true.

In addition, there are a series of samples with the designation WRC-SD-XXXX where XXXX is 2009 to 2014, inclusive. The 2009 sample is at the approximate location of the former hexachlorophene plant and has a 2,3,7,8-TCDD value of .006 ppb. Going downstream, the levels rise to 0.19 ppb (2010), 0.31 ppb (2011), and 0.40 (2012) ppb as Cap Area #1 is passed. The level drops to 0.06 ppb between the cap areas (2013) then rises to 8300 ppb below Cap Area #2 (2014). U.S. EPA provides no explanation of the poor correlation of 2,3,7,8-TCDD levels with the hexachlorophene manufacturing location.

Higher 2,3,7,8-TCDD levels were reported in a number of samples from the tailrace area (SD-30, CMS-4104, CMS-4109, CMS-4110, and CMS-4111) than in the samples from the Woonasquatucket River just downstream of the hexachlorophene plant. U.S. EPA provides no explanation for these results.

As discussed earlier, any dioxin discharged during the production of hexachlorophene at MA would have been strongly bound to charcoal. It is well established that removal of 2,3,7,8-TCDD and/or PCDD/PCDF from charcoal is very difficult.[38,39,40,58,59] Had there been hexachlorophene waste discharge by MA, the charcoal present likely would have adversely affected the 2,3,7,8-TCDD and/or PCDD/PCDF analysis internal standard recoveries. This adverse effect was not observed.

Therefore, any 2,3,7,8-TCDD and/or PCDD/PCDF contamination found in Allendale Pond is unlikely to be a result of dumping hexachlorophene waste directly into the pond or the Woonasquatucket River by MA.

E. NECC's drum reconditioning operations are a more likely source than MA for the dioxins found at the CMRP Site.

NECC engaged in the business of recycling industrial barrels and drums starting in approximately 1948.[60] They served a substantial customer base throughout the northeastern U.S.[61] into Canada and possibly as far south as New Jersey.[62]

NECC's recycling process for "open head" drums (drums with removable lids) started with residue removal followed by burning the drum in a gas fired furnace.[63,64] Although there is conflicting testimony about the handling of residues in the drums, the weight of the testimony strongly favors the emptying and burning of residues with the combustion byproducts ending up in the pit at the head of the recycling furnace.[63,65,66,67,68] There is consensus that the remaining residue was burned in the furnace.

The quantity of residue is also the subject of conflicting testimony, sometimes in the testimony of a given witness. For example, Mr. Joseph Nadeau, a part-time laborer for NECC between approximately 1962 and 1963 and for MA between approximately 1963 and August 1965,[69] testified that sufficient material, which he describes as "minimum," was available that employees were recovering oil from drums for use in their vehicles.[70] Mr. Felix Palumbo, a truck driver for Metro-Atlantic from approximately 1951 until sometime after 1968,[71] initially testified that drums he brought to NECC might contain a "couple of gallons of stuff," then immediately changed to testify that he did not look nor did he know how much residue might be in the drums he returned.[72] Vincent Buonanno, the son of NECC's founder and later its owner,[73] testified, "...all empty drums have some traces of the prior residues."[74]

Mr. Joseph Cifelli, who was a laborer at NECC from approximately 1954 until approximately 1961, and then served as a truck driver until approximately 1970,[75] testified that the normal practice was not to open drums to examine them for residual material.[76] Mr. John Turcone worked full-time as a laborer for MA, and also for NECC on a part-time basis, from August 1963 to January 1965.[77] He testified that drums could be as much as ¼ full when tipped onto the conveyer belt leading to the furnace.[78] Thus, it is more likely than not that drums containing some residual material were accepted with the residue being combusted during the reconditioning procedures.

Mr. J. Nadeau testified that the pit was on fire most of the time.[79] Mr. Raymond Nadeau, who worked for NECC from approximately 1956 through approximately 1962 as a laborer, and then until approximately 1969 as a truck driver,[80] testified that cleaning the pit involved shoveling out the residual ash.[81,82] Thus, it is more likely than not that the material dumped in the pit was reduced in volume by combustion. Given the materials listed in the NECC 104(e) response,[61] 2,3,7,8-TCDD and PCDD/PCDF would have been formed during this process.

Conflicts also exist in the testimony concerning the disposal of the materials in the pit. Mr. Cifelli is adamant that all drums were emptied into the pit, or accompanying waste storage drums if the pit were full, and that all waste from the pit or these waste storage drums was pumped out into a "cesspool" truck and hauled away.[83,84] He testified that he had no recollection whatsoever of any dumping activity by any NECC or Metro-Atlantic employees on the southern end of the property. Mr. V. Buonanno also testified that he was unaware of any dumping at the CMRP Site and that all fire pit waste was removed from the NECC site.[85] However, there is no plausible means to reconcile their testimony with the contamination found at the CMRP Site.

There are a number of Fire Reports that are in response to a burning dump approximately 100 yards south of the NECC buildings and near the Woonasquatucket River.[86] The Providence Journal on June 19, 1964 quotes Mr. Bernard Buonanno, Sr. indicating that the company planned to eliminate the dump by August or September.[87]

In contrast to Mr. Cifelli's and Mr. V. Buonanno's testimony, Mr. R. Nadeau provided consistent and graphic testimony of his personal participation in waste removal from the pit and its disposal on the southern end of the property.[81,82] Mr. J. Nadeau supports his testimony.[88] Mr. Kenneth Neri, who lived at 6 Amber Street, Johnston, RI, across the Woonasquatucket River from the CMRP Site, from approximately 1955 until approximately 1968,[89] provided graphic and extensive testimony describing waste burial at the CMRP Site.[90] Further support comes from Mr. David Carbone, the son of a truck driver for MA, and for NECC on a part-time basis, who accompanied his father around the Site and on trips in the early 1960s.[91] Mr. Carbone testified that NECC disposed of drums that could not be repaired at a location approximately 100 yards south of the NECC facility on the eastern side of the property.[92] Mr. Turcone offers similar testimony regarding NECC using an area south of their facility to "stockpile" unusable drums.[93] Mr. Neri identifies the same area as the site of extensive drum burial.[94]

According to Mr. Turcone[41] and Mr. J. Nadeau,[42] waste was placed in dumpsters and hauled off-site for disposal starting sometime in late 1963 or early 1964. The bulk of the testimony involving on-site waste disposal comes from people whose primary direct observations occurred prior to 1964.[81,82,88,90,92] Recognizing that waste management practices at the Site changed at that juncture can eliminate much of the conflict in the testimony.

It is more likely than not that NECC disposed of the residue from the "fire pit" and waste drums by dumping on the CMRP Site, contributing to the 2,3,7,8-TCDD and PCDD/PCDF contamination at the CMRP Site.

The fire pit was outdoors where it would be exposed to rain and snow.[95] There is testimony that water and fire extinguishers were frequently used to extinguish fires in the pit.[96] Mr. Cifelli testified that the pit would overflow from time-to-time.[97] It is more likely than not that pit residue entered the environment through run-off, contributing to the 2,3,7,8-TCDD and PCDD/PCDF contamination at the CMRP Site.

NewFields Environmental Forensics, LLC provided an environmental forensics analysis of the CMRP data. As stated in a letter to Deidre Dahlen, dated June 20, 2005:[98]

> The primary objective of this analysis was a statistical review of these data for the identification of compositional relationships among analytes detected in the study area. Specifically, we explored the likelihood that analytes detected above background screening levels were consistent with the assemblages of contaminants present in the source area around the location of the Centredale Manor property.
>
> Alternatively, we evaluated the likelihood that the chlorinated compounds were better associated with background or independent human activity adjacent to Allendale or Lyman Mill Ponds.

There is no indication NewFields considered the hypothesis that combustion of 2,4,5-TCP was a source of the 2,3,7,8-TCDD at the CMRP. Of perhaps greater concern, there is no evidence

that NewFields recognized that the dioxin data available from the source area are inadequate to support establishing a dioxin fingerprint for either MA or NECC.

The RI hypothesis assumes that the 2,3,7,8-TCDD contamination originated by dumping of waste directly from the MA hexachlorophene facility into the Woonasquatucket River. Given that assumption, the sediment samples taken from the river adjacent to the former MA hexachlorophene processing plant and downstream to the lower cap area make a logical set to establish the MA process fingerprint for use in the identification of the 2,3,7,8-TCDD source for material in Allendale Pond and points downstream. If the RI assumption is true, then none of the 2,3,7,8-TCDD in the Source Area could have originated with MA since it is assumed that all of their waste went into the river. As discussed above, any waste water discharged by MA into the river from the hexachlorophene process should have met drinking water standards for 2,3,7,8-TCDD contamination.

Testimony that NECC disposed of substantial fire pit waste in the lower cap area[81,82] means any samples taken from that point or further downstream are highly likely to be mixed with material originating from NECC, if not being NECC generated 2,3,7,8-TCDD contamination in their entirety. The NewFields analysis apparently considered the entire source area as a single set for establishing a fingerprint. This experimental design is not capable of distinguishing between MA and NECC as possible sources of the 2,3,7,8-TCDD.

One alternate hypothesis is that combustion of 2,4,5-TCP contaminated drums recycled by NECC is the source of the 2,3,7,8-TCDD contaminated wastes at the CMRP Site. In this case, an appropriate set of samples to establish a fingerprint would be those around the NECC site, the tail race area and the area approximately 100 yards south of the NECC facility used to bury unusable drums.[92,93,94] It is necessary to exclude any samples originating from the fill dirt added during later construction.

Simple inspection of these data reveals that the hypothesis involving NECC combustion of 2,4,5-TCP contaminated wastes is far more consistent with the available data than the hypothesis that MA hexachlorophene waste disposal is the culprit.

The conclusion that NECC is the principal source is further supported by the sworn testimony of direct participants[6,8,12,15,16,17] that the 2,4,5-TCP was purified using what is in 2006 recognized as the Best Available Technology for removal of 2,3,7,8-TCDD from aqueous materials.[18] The solid material was disposed by removal from the Site by a waste hauler.[41,42]

My research revealed no evidence that MA made any use of the dumpsite located approximately 100 yards south of the NECC facility. This area overlaps some of the highest-level 2,3,7,8-TCDD and "HCX" samples reported at the CMRP Site,[99] strongly supporting the theory that NECC's practices were the most significant source of 2,3,7,8-TCDD and PCDD/PCDF contamination at the CMRP Site.

The cleaning of "tight head" drums is another likely source of 2,3,7,8-TCDD and PCDD/PCDF contamination at the CMRP Site. Mr. Cifelli testified that "tight head" drums were treated with a strongly caustic solution in a tank that was drained once or twice per month.[100] He testified that the tank was approximately 4.5 to 5 feet wide, approximately 30 feet long, and sufficiently deep to immerse a 55-gallon drum (approximately 2.5 feet).[101] We can estimate the total volume to be approximately 330 cubic feet, thus yielding approximately 9,000 kg of strongly caustic solution per tank.

Mr. Cifelli testified that the caustic solution was heated to near the boiling point of water by means of steam pipes.[102] Thus, the residue in these drums would be subjected to a strongly caustic solution for a period of 2 to 4 weeks. Mr. V. Buonanno testified that caustic, used for cleaning, was one of the main raw materials consumed by NECC.[103] Mr. R. Nadeau testified that "tight head" drums that were converted to "open" drums went through the furnace[104] so that any residual 2,4,5-TCP or related material would have been converted to 2,3,7,8-TCDD in high yield.

Typical conditions under which 2,3,7,8-TCDD is formed during the synthesis of 2,4,5-TCP from 1,2,4,5-tetrachlorobenzene involves strong caustic at 170 to 180 °C for four hours. At the beginning of the four-hour reaction period, no 2,4,5-TCP would be present and hence no 2,3,7,8-TCDD formation would occur. The reaction would be allowed to continue until the maximum yield of 2,4,5-TCP was obtained then halted to prevent avoidable formation of undesirable by-products, such as 2,4,5-trichloroanisole. That is, the period in which 2,4,5-TCP is available for further reaction, such as formation of 2,3,7,8-TCDD, is less than four hours.

Using the rule of thumb that a chemical reaction rate approximately doubles for every 10 °C rise in temperature,[105] we see that two weeks is sufficient time for any residual 2,4,5-TCP in the caustic bath to form 2,3,7,8-TCDD. Similar considerations result in the conclusion that chlorophenols other than 2,4,5-TCP that might have been present in the tight head drums would lead to PCDD/PCDF formation.

Any 2,3,7,8-TCDD or PCDD/PCDF contamination from residue in the drum would remain in the cleaning solution at the end of the cleaning procedure. Thus, it is more likely than not that the 9,000 kg of caustic solution being drained one or two times per month would be contaminated with 2,3,7,8-TCDD and possibly other PCDD/PCDF depending upon the drum residues' composition.

The NECC 104(e) response as well as the former employee testimony establishes that a substantial number of drums with residual dyes were recycled.[61,106] Xanthene based dyes were in common and widespread use at the time. The possibility that the "soup" in the closed-head drum cleaning solution could have resulted in the formation of the material being reported as HCX cannot be excluded based on the data available. Had any of the drums been used for hexachlorophene transport, the likelihood of HCX formation in the cleaning bath becomes a high probability.

The testimony is not dispositive as to whether the tank drains were or were not connected to the sewerage system. Let us explore both possibilities as related to the effect of draining the caustic waste on the observed contamination at the CMRP Site.

If the drains were not connected to the sewerage system, then the contaminated waste caustic solution would have quickly found its way into the tailrace to the east of the NECC facility[107] and then migrated down the tailrace and possibly into Allendale Pond. This may have been facilitated by the storm drain identified in the deposition of Mr. Leonard Pezza,[108] the President of C. Pezza & Son, the company that was contracted to perform certain grading and excavation activities during the construction of the structure currently on the CMRP Site.[109]

If the drains were connected to the sewerage system, the situation is more complex. While my research revealed no remaining record of when or if the NECC facility was attached to the city sewerage system,[110] testimony indicates that the city sewerage system was installed in the area sometime in the early to mid-1940s.[111] Thus it is more likely than not that any plumbing

attaching the caustic tanks to the sewerage system was installed prior to 1952 when PVC pipe was introduced in the United States.

The two materials in normal use for plumbing during the period of NECC's operation were clay and cast iron. My research revealed no testimony regarding any pipe at either Metro-Atlantic or NECC other than cast iron.[112] Cast iron pipe is readily corroded by alkali.[113] The caustic used to clean the tight head drums was an alkali.

The use of clay pipes in any connection to the sewerage system would not have avoided leakage. The material used to seal clay pipe joints is not stable to alkali.[114] As we know from common experience, roots and/or excessive weight can break clay pipes.

Therefore, it is more likely than not that any plumbing system that might have been used in the NECC tight drum processing facility would have developed leaks and thus introduced 2,3,7,8-TCDD and PCDD/PCDF contaminated liquid in the area of the NECC facility. This contamination would then have migrated into the tailrace through normal erosion and soil transport mechanisms.[115]

In summary, it is more likely than not that NECC introduced 2,3,7,8-TCDD and PCDD/PCDF to the CMRP Site through the use of combustion or strong alkali to treat residue in drums with the release of the accompanying waste through burial of ash, burial of defective drums and leakage of spent cleaning fluids.

Figures 4-2 through 4-6 in the RI summarize the TEQ (mostly 2,3,7,8-TCDD) at 0-3", 1-2', 2-3', 3-4', and 4-5' on the CMRP Site. These charts show a shallow, widespread distribution pattern of 2,3,7,8-TCDD contamination with a large fraction of the samples above the action limit found in the top 3" of soil and spread relatively uniformly over the lower portion of the Site, primarily below the NECC facility. One of the exceptions is one of the two samples that have higher reported 2,3,7,8-TCDD concentrations than any other CMRP samples (140 ppb 2,3,7,8-TCDD, CMS-451-F). This sample comes from a location that is approximately at a corner of the NECC building and several feet deep. This is reportedly the location of the NECC fire pit.[116] The matching sample (CMS-240-A, also 140 ppb 2,3,7,8-TCDD) is located in Cap Area #2, which Mr. R. Nadeau testified was used to dispose of approximately two 55-gallon drums of fire pit residue per week from NECC.[81,82] The NECC building is in the 1956 Sanborn Map. That is, it was built years before MA engaged in hexachlorophene manufacture.

It is my opinion that the portion of the TEQ distribution pattern near the surface is inconsistent with process waste burial by MA occurring for a period of less than 5% of the Site's occupancy. It is my opinion that this portion of the distribution pattern is more consistent with a finding that the 2,3,7,8-TCDD contamination resulted from combustion byproducts being distributed through airborne particulates formed in the NECC fire pit and the disposal of fire pit waste over a long time period (of approximately 20 years) than disposal of MA hexachlorophene waste over a short time period of approximately one year.

It is my opinion that the fires reported involving the dump used by NECC for drum disposal is highly likely to be a major source of the 2,3,7,8-TCDD and PCDD/PCDF contamination found at the CMRP Site. The location of that dump is one of the most highly contaminated areas on the CMRP Site.

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COMMENTS ON THE INTERIM-FINAL
BASELINE HUMAN HEALTH RISK ASSESSMENT REPORT
CENTREDALE MANOR RESTORATION PROJECT SUPERFUND SITE
NORTH PROVIDENCE, RHODE ISLAND
NOVEMBER 2005

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AMEC Earth and Environmental, Inc.
15 Franklin Street
Portland, ME 04101

1.0 INTRODUCTION

In November 2005, EPA Region I released the Interim-Final Baseline Human Health Risk Assessment (BHHRA) for the Centredale Manor Restoration Project Superfund Site (EPA, 2005). The BHHRA was prepared by MACTEC Engineering and Consulting, Inc., under contract to Battelle Corporation, and was submitted to the Department of the Army, U.S. Army Corps of Engineers, New England Division under Contract No. DACW33-01-D-0004, Delivery Order No. 1. The BHHRA evaluates a number of exposure pathways, including fish ingestion and direct contact with surface water, sediment and bank soil. Exposures and corresponding risks are separately estimated for five Exposure Areas (EAs) that consist of reaches and wetlands associated with the Woonasquatucket River: EA1, Allendale Pond; EA2, Lyman Mill Pond; EA3, Manton Pond; EA4, Dyerville Pond and EA5, the Fogarty Center. Greystone Mill Pond and Assapumpset Pond are designated as the upstream background area and reference area, respectively. The BHHRA identifies two exposure scenarios that evaluated fish consumption: neighborhood residents who live along the river and consume fish from the river, and individuals who do not live in the neighborhood but visit the area for recreational fishing. Additional pathways for both populations include dermal contact and incidental ingestion of sediment, surface water and, for some EAs, bank soil.

At the request of Emhart Industries, Inc., AMEC Earth & Environmental, Inc. prepared these comments on EPA's BHHRA. The comments focus on those exposure scenarios and risk assessment topics that have the greatest implications on setting target remediation goals and assessing remedial options. Accordingly, the comments do not address aspects of the report that do not appear to be influential in evaluating remedies or determining outcomes for the site. Because EPA's estimate of potential risks from fish ingestion dominates total risks and has the most significant impact on the establishment of preliminary remediation goals (PRGs), our comments focus primarily on the approach and methodology used to estimate those risks associated with fish ingestion. However, we also provide comments regarding the incidental soil ingestion exposure pathway, which is the exposure pathway that poses the greatest theoretical risk second to the fish ingestion exposure pathway.

Section 2.0 of this document addresses concerns with the assumptions used in the BHHRA to establish exposure point concentrations based on the available fish tissue data. Specifically, concerns are raised regarding species selection and the use of whole body rather than fillet data. Section 3.0 discusses other exposure assumptions used in the BHHRA to evaluate fish

ingestion. Among these concerns are the selection of a realistic and reasonable rate of fish ingestion from a non-destination fishery with limited potential to support an avid angling population, and fish preparation (i.e., trimming and cooking) methods. Section 4.0 provides comments on the direct contact exposure pathways. Finally, Section 5.0 considers the limitations of EPA's discussion of dioxin toxicology.

2.0 FISH TISSUE DATA

The BHHRA relies on fish tissue data collected in 2001 and bases exposures and risks associated with fish ingestion on the following limited collection of fish samples:

- Assapumpset Pond and Brook – 3 brown bullhead, 4 largemouth bass fillet, and 6 American eel;
- Greystone Mill Pond – 10 white suckers, 10 largemouth bass fillet, and 10 American eel;
- Allendale Pond – 10 white suckers, and 10 American eel;
- Lyman Mill Pond – 10 white suckers, 10 largemouth bass fillet, and 10 American eel;
- Manton Pond – 3 largemouth bass fillet; and
- Dyerville Pond – 3 American eel.

The use of the present fish tissue data adds uncertainty to the BHHRA for several reasons. First, there is a paucity of fish tissue data in existence for Manton and Dyerville Ponds. The risk estimates for Manton Pond, which is approximately two to three acres in size, are based on only three largemouth bass specimens in the EPA BHHRA. For Dyerville Pond, a water body that is essentially a riverine environment restricted only by the pilings of the former dam, EPA's human health risk estimates via fish ingestion are based on data for only three American eel samples. A sample of three individual fish from these ponds is clearly inadequate from a statistical, biological and common sense perspective for accurately assessing uptake and risk from ingestion of fish from these areas.

Second, there is unnecessary uncertainty introduced in the BHHRA through the use of data that are not consistent with the contemporary use and condition of the exposure area. In May 2001, the Allendale Dam was breached allowing unimpeded fish migration between Allendale Pond and Lyman Mill Pond until the February 2002 dam repair, which eliminated free passage of fish between the ponds. Rebuilding the dam, which EPA ordered the Centredale Manor Responding Parties to construct at considerable cost in order to reduce downstream exposure to dioxin, eliminated free passage between the ponds. However, all of the fish tissue samples

collected by EPA for Lyman Mill and Allendale Ponds were collected in the summer of 2001 – *after* the breach of the dam, but prior to its repair. While fish caught from each pond are used to assess the risks for each pond, there is no evidence to support the assumption that the fish caught in a specific pond have always resided in that pond. Hence, the chemical body burdens of fish in a specific pond (e.g., Lyman Mill Pond) could be due to sediments encountered in that pond or from an upstream reach. If the fish were from an upstream reach and had migrated downstream, the data would not be representative of the conditions that currently exist after the dam was repaired.

After their review, the Contaminated Sediments Technical Advisory Group (CSTAG) raised the concern that the fish tissue data may not be representative of current conditions and strongly recommended to EPA Region I that new, co-located sediment and fish tissue samples be collected to assess these ponds in their current condition (i.e., with Allendale Dam repaired) (EPA, 2004). EPA's response to this CSTAG comment was that they would conduct a review of data to further determine the potential that the fish tissue are not representative of current site conditions. After a thorough evaluation of this issue, EPA's contractor reported to EPA that the elevated chemical fish tissue concentrations in Lyman Mill Pond are from fish that likely migrated downstream from the more contaminated Allendale Pond to the less contaminated Lyman Mill Pond (MACTEC, 2005). Furthermore, this phenomenon is not unique to the upper ponds because eel may have been able to pass the dams freely and, consequently, their body burdens may originate from any of the ponds.

Nevertheless, with full knowledge of this potentially critical data flaw and having been advised to collect additional data that are consistent with current site conditions, EPA inappropriately uses the post-breach fish tissue data in the BHHRA. The use of the post-breach 2001 fish sampling data in the BHHRA is not consistent with the contemporary conditions of the exposure area.

Third, exposure point concentrations (EPCs) are derived for each species in each area (95% UCLs for each) and then averaged to derive a combined EPC. This approach assumes an equal likelihood of eating eel, bass, and sucker. Such an assumption is inappropriate since a review of the available recreational fishing survey data indicates that suckers are rarely, if ever, consumed, and most recreational anglers do not harvest eels. In fact, EPA's *Exposure Factors Handbook* shows that only 0.5 percent of fish consumed from rivers and streams consists of bottom fish such as suckers, carp, or sturgeon (EPA, 1997). EPA has determined that sucker consumption is only 5 percent of bass consumption from rivers and streams. Furthermore, the

technique used to catch eels differs from the technique used to catch the most desirable finfish species. As a result, eel consumers, if they exist on the river, are not likely to be the same population as finfish consumers. Thus, EPA's assertion in the BHHRA that there is an equal likelihood that a fish consumer would eat bass, eel and suckers is simply not correct. A vast majority of sport caught fish consists of trophic level 4 fish such as bass. In the BHHRA, EPA should have used a percent weighting more consistent with its *Exposure Factors Handbook*, such as 90:5:5, bass:sucker:eel in the assessment and not combined the different fish species with equal weighting in deriving the EPC. This is also consistent with EPA's own survey results as reported in the BHHRA, which show that anglers on the Woonasquatucket River are practicing catch and release, and none report catching white suckers or American eel.

In addition to the assumption that there is an equal likelihood of eating the three fish species, whole body concentrations of dioxins/furans and PCBs in eels and suckers are used. While it may be reasonable to assume that the entire eel is consumed in certain instances, it is not reasonable to assume that individuals eat the entire sucker, which is an extremely bony fish. Given the low likelihood that suckers will be consumed and the fact that any individuals who might consume sucker would certainly not consume the entire fish, whole body data for sucker cannot be considered representative of the fish tissues that will be consumed from the river.

Fourth, there is an inconsistency and lack of comparability in fish species for all the Ponds. For Assapumpset, Greystone Mill, Lyman Mill and Manton Ponds, fillet data for bass are available and should provide the basis for the EPCs for the fish ingestion pathway. Apparently, fillet data were not collected for Allendale Pond and as mentioned previously, only eel data were collected for Dyerville Pond. Thus, there are no fillet data available for use in these two areas. Although EPA recommends, "using fillets as the standard sample type for analyzing chemical contaminants," (EPA, 2000a), it is not uncommon to use a conversion factor on the order of 30 percent to convert whole body concentrations to fillet concentrations. However, when the 30 percent conversion factor is applied to the available data for Lyman Mill Pond, whole body concentrations of sucker do not yield concentrations that are representative of fillet concentration of edible fish species (i.e., bass). Therefore, when PRGs are developed, these PRGs should be applied to the fillet portions only of those species that are likely to be caught and consumed by recreational anglers (bass and any other desirable finfish species that may be present there). A comparison of the upper bound bass concentrations with the upper bound combined fish concentration in Lyman Mill Pond indicates that the EPC based on the bass fillet data is lower than the combined fish EPC that is currently used, by roughly an order of

magnitude. Thus, it appears that the use of bass fillet data to conduct the risk assessment would reduce risks by a factor of 10 in this reach. Where this same pattern occurs in other reaches, risks would be reduced similarly by the collection and use of fillet data as the basis for the EPCs in those reaches. Where eels are the only source of data (i.e., Dyerville Pond), the estimated risks are significantly higher than if largemouth bass data were used.

Lastly, throughout the BHHRA, including the initial screening steps, the concentration of 2,3,7,8-TCDD toxic equivalents (TEQ) is evaluated as a distinct entity, in lieu of evaluating the individual dioxins and furans. While each of the samples may have a TEQ concentration, it is not necessarily true that the TEQ in each of those samples is comprised of the same dioxin and furan congeners, nor in the same relative proportions to one another. As a result, there may be individual congeners that were non-detect in discrete samples or areas that are included at half their detection limits that would have been screened out if they had been evaluated separately. It is not possible to determine the impact that this approach may have had on the EPCs for TEQ, since the raw data are not available. It would be worthwhile, however, to conduct a discrete screen of each dioxin/furan congener to determine whether there are any compounds that should have been screened out (and thus not assumed to be present at one-half the detection limit when calculating TEQ).

3.0 FISH INGESTION PATHWAY

The BHHRA uses an approach for both the visiting and resident angler that substantially biases the risk estimates associated with the fish ingestion pathway. These biases result in an overestimation of the potential risk. These biased approaches are discussed in this section.

In its evaluation, the BHHRA assumes that both resident and visiting anglers consume a combined fish diet of American eel, largemouth bass fillet and white sucker at the following daily ingestion rates:

Resident and Visiting Angler	Fish Ingestion Rate (grams/day)	
	RME	**CT**
Adult	14	8.9
Older child	9.3	5.9
Young child	4.7	3.0

These ingestion rates are based on a sensitivity analysis using the Maine Angler Survey data (Ebert et al., 1993) for recreationally caught fish obtained from freshwater rivers and streams. They represent the arithmetic mean and 90th percentile rates from the distribution where only anglers eat the fish, i.e., no sharing with other members of the angler's household. EPA estimates the fish ingestion rates for children by setting them equal to 2/3 of the adult 'no sharing' rate and 1/3 of the adult 'no sharing' rate for the older and younger child, respectively. The Maine Angler Survey data provide an appropriate basis for fish consumption estimates for the Site. However, selecting rates that assume that all of the fish brought into each household are consumed by a single individual is not appropriate because the survey respondents specifically report the number of individuals with whom they shared their fish meals. Therefore, EPA's assumption that none of the fish are shared is a flagrant introduction of bias that leads to overstated risk estimates.

Despite an acknowledgement in the BHHRA that some sharing of the catch does occur among household members, these rates are based on the untenable assumption of no sharing. EPA justifies this biased approach by stating,

> "...there would be considerable uncertainty introduced into the fish consumption rates if assumptions were made concerning the number of household members who share the catch and if simplifying assumptions were made concerning the number of household members. In order to avoid introducing those additional uncertainties, it has been assumed the angler consumes all the fish brought into the household. Therefore, the selected fish consumption rates for adults are likely overestimates of fish consumption rates by recreational anglers." [p. 3-3].

EPA also states in the BHHRA, however, that sharing is assumed to occur for visiting recreational anglers where the catch from older children or an adult is shared with a young child. This is internally inconsistent with EPA's unfounded assumption that the data from Ebert et al. (1993) should not be evaluated with the underlying assumption of sharing the catch with other consuming family members. As shown below the uncertainty introduced when incorrectly using the non-sharing fish ingestion rates is greater than 200%.

Better estimates of consumption are the rates reported by Ebert et al. (1993) for consumption from rivers and streams and where sharing among all household consumers who consume fish were considered. Applying the approach used in the BHHRA (arithmetic mean and 90th percentile rates for adults; 2/3 and 1/3 adult rates for older and young child, respectively), the following table reports revised and more supportable fish ingestion rates. The use of these revised rates results in a reduction in risks by the fish ingestion pathway by a factor of 2.3.

Resident and Visiting Angler	**Revised Fish Ingestion Rate (grams/day)**	
	RME	**CT**
Adult	6.1	3.7
Older child	4.1	2.5
Young child	2.0	1.2

In addition to overestimating fish ingestion with biased ingestion rates, the BHHRA assumes that 100 percent of the recreationally-caught freshwater fish consumed are obtained from the Site. This is unlikely, even for the residents, given the limited appeal of the river as a fishery. While it is possible that low frequency consumers might consume all of their fish from the Site, higher frequency consumers (who are generally more avid anglers) will likely choose to fish alternative fishing locations that provide better conditions for fishing. Indeed, the survey data reported in Ebert et al. (1993) reported state-wide fish consumption rates among anglers and their families, and shows that over 80% of anglers fished two or more bodies of water per year, approximately 60% fished three or more, and nearly 40% fished four or more. Moreover, Ebert et al. (1993) conclude that it is likely that only a portion of the fish ingestion estimates for rivers and streams can be attributed to one water body. In addition, data presented in EPA's *Exposure Factors Handbook* show that only 32.5% of sport caught fish is from a local source. EPA has relied on this percent of locally caught fish in their assessment of dioxin in fish from landspread biosolids when formulating the technical basis in national rulemaking (EPA, 2002a). Based on this information, it is more reasonable to assume for these individuals that only one-third to one-half of the fish consumed every year will be obtained from the river. Assuming that one-half of the total consumption is derived from the Site, the risks from fish ingestion are reduced by an additional factor of 2.

Lastly, the BHHRA does not account for the loss of dioxins/furans during preparation and cooking of fish. There is substantial evidence to indicate that losses due to cooking range from 25 to 75 percent (Stachiw et al., 1988; Zabik and Zabik, 1995; Zabik et al., 1996; EPA, 2000a).

Increasing the cooking time, the cooking temperature and the surface area of the fish fillet enhances the amount of loss (Stachiw et al., 1988; Zabik et al., 1996; EPA, 2000a). A very conservative estimate of 25 percent loss would result in risk reduction for the fish ingestion pathway by a factor of 1.3. This loss from cooking does not include any reduction that occurs during fish preparation, such as trimming and skinning. Since dioxins as well as other lipophilic chemicals accumulate in fatty tissues, removal of skin and trimming the fat before cooking will decrease exposure (EPA, 1999).

The preceding paragraphs have presented more reasonable and realistic exposure parameters for the fish ingestion pathway. When these parameters are used in combination with the fillet concentrations in desirable fish species (roughly a factor of 10 below whole body concentrations in sucker and eel), risk estimates are significantly reduced and are within or very close (within a factor of 2) to EPA's acceptable risk range, even at current concentrations. While the issues raised here impact the baseline risk estimates, these issues also have substantial impact in the development of PRGs.

4.0 DIRECT CONTACT PATHWAYS

While the estimated risks associated with direct contact with surface water, sediment and bank soil are not as great as those estimated via fish ingestion, a number of the direct contact assumptions lead to overestimates and are discussed in this section.

For the older child and adult (RME), the BHHRA assumes that swimming in the ponds occurs 13 days per year and that wading in the river occurs an additional 39 days per year. For the young child, only wading is evaluated with a frequency of 52 days per year. Swimming frequency is based on one day per week during the three warm summer months (June, July and August). Wading frequency for the older child/adult is based on three days per week during June, July and August and four days per week for the young child.

The exposure frequency for swimming is inflated according to EPA's *Exposure Factors Handbook*, which recommends an exposure frequency for swimming of one day per month (EPA, 1997). Based on the approach used in the BHHRA, this would result in an exposure frequency of three days per year. However, it is possible that individuals who swim in the river might do so with a somewhat higher frequency due to close proximity to the water. According to the *Exposure Factors Handbook*, the maximum number of minutes that individuals reported swimming in a freshwater swimming pool (a substantially more attractive swimming location

than the ponds being evaluated in the BHHRA) was 181 minutes or three hours per month. If it is assumed, as it was in the BHHRA, that a person spends one hour swimming per event, this time equates to three swimming events per month or a total of nine one-hour swimming events during June, July and August. This is a more reasonable upper bound estimate.

It is unlikely that most individuals will spend substantial amounts of time wading in the river or ponds that do not have beach areas. Adults may wade while doing maintenance activities (cleaning out litter or vegetation) but would not generally be expected to wade unless they planned to swim as well. Thus, it would be more reasonable to assume that adults wade 2 days per month, for an additional exposure frequency of six days per year. Young children would not be permitted to play along the ponds without adult supervision. Thus, their water exposure frequency would be similar to adults (15 days).

The BHHRA reports that surface water data from Lyman Mill Pond are used as surrogate data for Manton and Dyerville Ponds, since no surface water data are available for these ponds. This is not appropriate and risk estimates based on this scenario cannot be considered representative of risks associated with those ponds, which are likely to have lower water concentrations than Lyman Mill Pond.

The BHHRA has assumed that one hundred percent of the soil contacted and ingested daily is from the bank soils. It is unlikely that residents will spend their outdoor recreational time along the banks of the ponds and no time in their yards. Some fraction should be applied to account for the time spent in other portions of their yards. EPA's *Risk Assessment Guidance for Superfund* (1989) stresses that the data used to evaluate potential exposures to surface soil should be representative of the entire exposure area and not limited to data that represents only the contaminated portion of an exposure area. EPA also states that it is reasonable to evaluate residential exposures by averaging data from the entire residential yard. EPA does not follow its own exposure assessment guidance when evaluating the potential surface soil exposures for bank soils. The BHHRA uses a factor of 50 percent in its evaluation of the visiting angler. A similar factor or perhaps a substantially lower factor should be used for the resident, depending upon the distribution of the sampling locations and the fraction of residential yards that the non-impacted soils represent.

The BHHRA uses soil and sediment ingestion rates that are not supported by EPA's own exposure assessment guidance and are not supported by current soil ingestion studies.

Similarly, the BHHRA has used these upper-bound soil ingestion rates to evaluate sediment exposures, despite its acknowledgement that sediment is not likely to adhere to the skin. This approach substantially overestimates potential exposure due to ingestion of sediment and also ignores the fact that on the days that sediment exposure occurs, it is only likely to represent a small fraction of the total soil/sediment ingested throughout the day. Furthermore, upper bound soil adherence factors are used in the BHHRA to evaluate potential dermal contact with sediment, despite the BHHRA's acknowledgment that sediment is not likely to adhere to skin because it is washed away by the water during wading and swimming activities.

Selection of Soil Ingestion Rates

In assessing exposures and risks from the ingestion of bank soil, the BHHRA evaluates the residential, recreational and industrial/commercial exposure scenarios. Depending on the exposure scenario, different age categories of potentially exposed human populations are assessed. The age groups considered include young children (aged 1 to 6 years), older children (aged 7 to 18 years), and/or adults (aged 19 to 30 years). When characterizing RME exposures to bank soils, the BHHRA uses upper-bound soil ingestion rates of 200 mg/day for young children (1 to 6 years of age) and 100 mg/day for older children and adults. For the CTE exposures, the BHHRA generally uses soil ingestion rates of 100 mg/day for young children and 50 mg/day for older children and adults.

As described in the BHHRA, the general soil ingestion rates are based on EPA New England Risk Update No. 2 (1994a). However, improved, more recent studies of soil ingestion by both children and adults have been published in the peer-reviewed literature. These studies show that the rates used in the BHHRA are overestimated. Because of improvements in study methodologies, the results of these more recent studies are more representative of potential exposures to these individuals. Specifically, two recent studies (discussed below), published by the authors of the studies upon which EPA has based its upper-bound estimates, provide the most objective information for use in deriving high-end estimates of daily soil intake. Adoption of these more recent data would be consistent with EPA's *Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility, and Integrity of Information Disseminated by the Environmental Protection Agency* (EPA, 2002b). These guidelines identify information suitable for inclusion in risk assessments as "the best available science and supporting studies conducted in accordance with sound and objective scientific practices, including, when available, peer reviewed science and supporting studies."

Soil Ingestion Rates for Younger Children

The BHHRA uses a soil ingestion rate of 200 mg/day to evaluate RME exposures to 1 to 6 year old children. Fifteen years ago, EPA recommended this rate in its *Risk Assessment Guidance for Superfund* (EPA, 1991) and reiterated that recommendation in its *Exposure Factors Handbook* (EPA, 1997) as a "conservative estimate of the mean." The latter recommendation was based primarily on tracer studies in children (ages 1 through 5) that were undertaken by Calabrese and his coworkers (Calabrese et al. 1989; Stanek and Calabrese, 1995a; 1995b). However, updated studies by these same authors, conducted using improved methodologies and published since that guidance was released, indicate that these previous estimates are overestimates and can be refined and improved.

The most recent such study in children was published by Stanek et al. (1999) and Stanek and Calabrese (2000). As described by Stanek and Calabrese (2000), this study implemented several improvements in study design and analytical procedures that occurred since the publication of their earlier papers and that led to an improved estimate of the 95th percentile soil ingestion estimate for this age group. The advantages of this recent study included: (1) a relatively large study group (n = 64 children); (2) improved particle size measurements that focused attention on soil of smaller particle size; (3) a longer study duration (365 days); (4) randomized selection of participants; (5) the use of a relevant age group (1 to 4 year old children); (6) use of a random sample of the population for that age group; and (7) better control for input/output error. The soil ingestion rates reported by Stanek and Calabrese (2000) for these children were:

- A 95th percentile rate of 106 mg/day (when evaluated over a 365-day period);
- An arithmetic mean ingestion rate of 31 mg/day; and
- A median (50th percentile) ingestion rate of 17 mg/day.

This study also calculated the best linear unbiased predictors of the 95th percentile of soil ingestion over different time periods and reported the following results:

- Over a 7-day exposure period, the 95th percentile soil ingestion rate was 133 mg/day;
- Over a 30-day exposure period, the 95th percentile soil ingestion rate was 112 mg/day;
- Over a 90-day exposure period, the 95th percentile soil ingestion rate was 108 mg/day; and
- Over a 365-day exposure period, the 95th percentile soil ingestion rate was 106 mg/day.

These data suggest that, as the length of time that the children are studied increases and as the precision of the analysis improves (i.e., reduced uncertainty), the daily ingestion rate estimates decline. This is reasonable due to the fact that daily fluctuations in soil ingestion rates will tend to average out over time. This narrowing of the distribution in the soil ingestion estimates when daily variability and uncertainty are reduced is not unexpected and is referred to as "regression to the mean" (Stanek and Calabrese, 2000). As noted by Stanek and Calabrese (2000), these longer-term estimates are more appropriate when assessing risks and hazards associated with chronic exposure, as is the case in the BHHRA.

In a presentation to EPA Region 1 in May 2002, Dr. Calabrese explained these points and recommended, based on this more recent study, that the soil ingestion rates to be used for young children in recreational scenarios should be 100 mg/day for the upper bound and 20 mg/day (based on the median in this study) for the central tendency estimate. Dr. Calabrese has reiterated these recommendations in written form, which has been submitted to EPA Region 1 in the past. *See* Exhibit 1. Based on the more recent and scientifically robust study data, the BHHRA should be revised to use these rates as the general soil ingestion rates for 1 to 6 year old children in residential and recreational exposure scenarios.

Soil Ingestion Rates for the Older Child and Adult

As noted in the BHHRA, the general soil ingestion rates used to evaluate exposures to older children and adults (100 mg/day for the RME and 50 mg/day for the CTE) are based on EPA New England Risk Update No. 2 (1994a). Again, however, there is now a more recent study of adults by the same investigators (Stanek et al., 1997), which included a number of improvements over previously published studies: (1) a larger number of subjects and days of participation; (2) improved study design that considered seven consecutive days of fecal sampling; (3) improved selection of soil tracers; (4) a broader range of soil ingestion validation; and (5) enhanced capacity for additional assessments including particle size of the soil ingested. The result was more reliable daily estimates of soil ingestion and a greater capacity for more reliable long-term modeling estimates.

The 1997 study was not without complications, however. Of the ten adults participating in the study, one had an unusually high soil ingestion estimate (2 grams) for the first day of the study week. This high estimate resulted in an inflated upper percentile estimate of the overall ingestion rates. In fact, Stanek et al. (1997) stated that "the 95th percentile soil ingestion

estimate was 331 mg/day, but based on present data, it is substantially uncertain" because of the results from this one subject. Further, as described by Dr. Calabrese (a co-author of the paper) in his attached letter, this subject had four times higher freeze-dried fecal weight on the first day than on any other day of the study, thus suggesting that his excretion on that day reflected a multi-day accumulation, instead of just one day, as assumed in the calculations. This fact confirms that the 95th percentile value from this study, which is driven by the result for this one subject, is not only uncertain but substantially overestimated. Due to this aberrant result from one participant, Dr. Calabrese has recommended, in his attached letter, that the upper 75th percentile (49 mg/day, rounded to 50 mg/day) from the Stanek et al. (1997) study is the most appropriate value to use as an estimate of high-end soil ingestion by adults. He has also recommended use of a value of 10 mg/day for the central tendency estimate. This is consistent with the results of Stanek et al. (1997), who reported an adult mean daily soil ingestion estimate of 6 mg/day, and it represents 50 percent of the young children's median rate. EPA should use these soil ingestion rates in the BHHRA.

For the reasons discussed above, EPA should revise the BHHRA to adopt general soil ingestion rates based on the more recent and improved studies (Stanek and Calabrese, 2000, for young children; Stanek et al., 1997, for older children and adults).

The BHHRA separately considers the ingestion of bank soil (78 days per year) and the ingestion of sediment (52 days per year). It is likely, however, that potential ingestion of soil/sediment from these sources occurs on the same day when individuals are wading or swimming. Thus, it is not appropriate to use total daily ingestion rates for both exposure pathways. Instead, either a fraction of the total ingestion should be assigned to each pathway or, since sediment is essentially being evaluated as soil, a combined EPC could be developed using both soil and sediment sampling data to derive a single exposure pathway to evaluate combined sediment and soil exposure on those days.

Finally, the location of the sediment samples that are used to derive the EPCs for the sediment exposure pathway cannot be identified in the BHHRA. It is important, however, that only the near-shore sediment data be included in the derivation of those EPCs as those are the only sediments with which people will be in contact during wading activities.

If exposure parameters for the soil and sediment contact pathways were modified to reflect more reasonable exposure conditions, estimated risks would be within EPA's acceptable risk range.

5.0 TOXICOLOGY OF DIOXIN AND DIOXIN-LIKE COMPOUNDS

EPA's discussion of dioxin toxicology fails to fully discuss the full range of potential dioxin cancer potency factors. EPA should include further discussion on the toxicology of dioxin, specifically identifying peer-reviewed scientific studies spanning the full range of plausible cancer slope factors for 2,3,7,8-TCDD. Moreover, the BHHRA should state that EPA has never published a cancer slope factor (CSF) for TCDD in IRIS and that the carcinogenic potential of TCDD is a matter of much debate in the dioxin reassessment process that has been underway at EPA for 15 years.

A wide range of CSFs based on animal studies, ranging from 9,000 to 156,000 $(mg/kg\text{-}day)^{-1}$, have been proposed by various groups under the linear, non-threshold cancer model, with differences resulting from the selection of tumor classification scheme and interspecies scaling factor used (EPA, 1994b, 2000b; FDA, 1993, 1994; Keenan et al., 1991). Recently, in two subsequent revisions of the draft Dioxin Reassessment, EPA proposed the use of a CSF of 1,000,000 $(mg/kg\text{-}day)^{-1}$ based on its evaluation of human epidemiological data (EPA, 2000b; 2003). However, there is substantial disagreement concerning the appropriate CSF for TCDD. In fact, in its review of EPA's draft final Dioxin Reassessment, the Agency's Science Advisory Board discussed the uncertainties associated with the available dose-response data and the extrapolation methods used, and stated that they could not "reach consensus on a single value for a dioxin potency factor" (EPA, 2001; p. 6). Subsequently, Congress directed EPA to undertake an agreement with the National Academy of Sciences (NAS) to review the draft Dioxin Reassessment (House of Representatives, 2003). Among other key issues, Congress asked NAS to evaluate "the validity of the non-threshold linear dose-response model in light of epidemiological studies and the corresponding cancer slope factor calculated by the Agency through use of this model" and "the appropriateness of including 'dioxin-like' chemicals in the risk assessment without independent empirical review of their effects" (House of Representatives, 2003, pp. 1445-46). Thus, there is substantial uncertainty associated with any selected CSF and, depending upon the CSF selected, the resulting regulatory standards and remedial decisions can vary by orders of magnitude.

NAS recently completed its review of the draft Dioxin Reassessment (NAS, 2006). Among its conclusions, the NAS committee determined that the available data support a nonlinear relationship rather than defaulting to a non-threshold linear model as EPA has in the past. NAS concluded,

> "The committee unanimously agrees that the current weight of evidence on TCDD, other dioxins, and DLCs [dioxin-like compounds] carcinogenicity favors the use of nonlinear methods for extrapolation below the point of departure (POD) of mathematically modeled human or animal data." [p. 135]

NAS further commented that,

> "a risk assessment can be conducted without resorting to default assumptions. To the extent that EPA favors using default assumptions for regulating dioxin as though it were a linear carcinogen, such a conclusion should be supported with scientific evidence." [p. 100]

Summarizing this issue, NAS stated that,

> "The selection of the default linear extrapolation approach was one of the most critical decisions in the Reassessment, but the decision to use this approach was not supported by a scientifically rigorous argument, nor was there a balanced presentation of arguments that would support the calculation and interpretation of a MOE [margin of exposure] with the same data. The committee determined that a balanced presentation of available data could support the use of a threshold model with subsequent calculations and interpretation of MOE. (For cancer risk assessment, the threshold approach should be used in addition to the linear approach.)" [p. 140]

While NAS (2006) commended EPA on its use of the benchmark dose (BMD) method as a replacement of the traditional approach of using the no-observed-adverse-effect level (NOAEL) and lowest-observed-adverse-effect level (LOAEL), NAS criticized EPA for using an effective dose (ED) at a 1% response level (ED01) for characterizing cancer and noncancer risks. NAS stated,

> "In its evaluation of ED01 used for cancer risk assessment, the committee concluded that EPA had not adequately justified use of the 1% response level as the POD for the analysis of either the epidemiological or the animal bioassay data....
>
> For the various noncancer end points, EPA should describe more clearly how and why the ED01 values were determined in animals and transferred to human equivalents. At the least, the risk assessment should provide more apparent and parallel calculations using a 5% response level as the POD to demonstrate the impact that this assumption might have on both the point estimates of risk at low doses and the range of uncertainty surrounding that point estimate. This

> recommendation applies to extrapolation for cancer risk estimates, as well as for noncancer risk estimates." [p. 135-136]

The NAS committee recommended that EPA consider the full range of animal bioassay data, "including the new NTP animal bioassay studies on TCDD, other dioxins, and DLCs for quantitative dose-response assessment." [p. 136]

While NAS agreed that use of body burden as the dose metric is a reasonable approach, the committee raised a number of uncertainties associated with using body burden to develop risk estimates. NAS (2006) concluded,

> "The magnitudes of the various uncertainties are not clearly defined. The most significant impact is the species differences in percentage body fat on the relationship between body burden and the concentrations present in nonadipose tissues. An analysis of the impact of possible uncertainties in the dose metric on the final risk estimates would be informative.
>
> It remains to be determined whether the current WHO TEFs, which were developed to assess the relative toxic potency of a mixture to which an organism is directly exposed by dietary intake, are appropriate for body burden toxic equivalent quotient (TEQ) determinations, which are derived from the concentrations of different congeners measured in BF. If body burdens are to be used as the dose metric, a separate set of body burden TEFs should be developed and applied for this evaluation. Without these corrected values, the overall TEQs estimated by use of intake TEFs might be substantially in error." [p. 138]

Furthermore, the high level of uncertainty associated with the application of the CSF for TCDD to dioxin-like PCBs is unnecessary, due to the fact that EPA has already established a CSF for PCBs that is based on empirical bioassay data (EPA, 1996). Depending upon how this is done, the approach may substantially overestimate the carcinogenic potential of PCBs as it may double-count the carcinogenic potential of the entire mixture. This is because the approach that is often used evaluates the "dioxin-like" PCBs using the CSF for TCDD but then evaluates the carcinogenic potential of the remaining "non-dioxin-like" PCBs using the CSF that EPA has established for PCBs. However, the CSF that has been derived for PCBs includes the carcinogenic activity of the entire PCB mixture tested, including both the "dioxin-like" and the "non-dioxin-like" congeners. Thus, the carcinogenic potential of the "dioxin-like" congeners is counted twice and added together when this approach is used.

6.0 CONCLUSIONS

This review has identified, documented and discussed the technical shortcomings of the analyses that comprise the EPA BHHRA report. We have focused on those exposure scenarios and risk assessment topics that have the greatest implications for setting target remediation goals and assessing remedial options. Our review does not comment on those elements of EPA's work that do not appear to be influential in evaluating remedies or determining outcomes for the site. Instead, we have discussed the substantial shortcomings and biases inherent in EPA's assessment of potential risks from fish ingestion. This pathway dominates the total risk estimate, and it has the most significant impact on the establishment of PRGs. We also have commented on the flaws inherent in EPA's assessment of potential risks via incidental soil and sediment ingestion, which is the exposure pathway that poses the next greatest theoretical risk after the fish ingestion exposure pathway.

Because these exposures pathways are used by EPA to develop PRGs in a separate document, these flawed risk assessments have resulted in flawed and inappropriate PRGs. The development of PRGs requires a much more robust and unbiased risk assessment in which state-of-the-art methods of risk analysis are coupled with competent interpretation and analyses of available data. The BHHRA does not meet this standard in its present form, and anything short of a complete overhaul and revision is unlikely to approach the level of sophistication required to serve as the basis for establishing technically defensible remedial cleanup goals for the Centredale Manor Restoration Project Superfund Site.

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Exhibit 1

July 23, 2003

Kevin W. Holtzclaw
Manager – PCB Issues
General Electric Company
3135 Easton Turnpike
Fairfield, CT 06431

Re: Soil Ingestion Rates

Dear Mr. Holtzclaw:

The General Electric Company (GE) has asked for my opinion on the soil ingestion rates used by the U.S. Environmental Protection Agency (EPA) for general residential and recreational exposures in its June 2003 draft of the Human Health Risk Assessment (HHRA) for the Housatonic River. For upper bound exposures, those rates are 200 mg/day for young children and 100 mg/day for older children and adults. The central tendency estimates are 100 mg/day for young children and 50 mg/day for older children and adults. These rates are based on prior studies by our group – Calabrese et al. (1989) and Stanek and Calabrese (1995a, b) for young children, and Calabrese et al. (1990) for adults.

As explained in a presentation that I made on this subject to EPA Region 1 in May 2002, I believe that these rates are overstated and can be significantly improved by reliance on newer soil ingestion studies from our group, which used improved methodologies. I have reviewed the discussion of this topic in the document entitled "Attachment E: Selection of Soil Ingestion Rates," which GE sent me and which I understand will be part of GE's comments on the HHRA. I entirely agree with that discussion. To summarize:

1. Soil Ingestion Rates for Young Children. Our most recent study of soil ingestion rates in young children (Stanek and Calabrese, 2000) included several improvements over our prior studies. These included: 1) a relatively large study group (64 children); 2) improved particle size measurements that focused attention on soil of smaller particle size; 3) a longer study duration (365 days); 4) the use of a relevant age group (1 to 4 year old children); 5) use of a random sample of the population for that age group; and 6) better control for input/output error. The results of this study showed a 95th percentile soil ingestion rate of 106 mg/day (when evaluated over a year), a median (50th percentile) ingestion rate of 17 mg/day, and an arithmetic average ingestion rate of 31 mg/day. Based on these results, I recommend that the most appropriate soil ingestion rates to use for chronic exposures to young children would be an upper bound rate of 100 mg/day (based on the year-long 95th percentile value from our study) and a central tendency estimate of 20 mg/day (based on the median value from our study).

2. Soil Ingestion Rates for Adults and Older Children. Our most recent study of soil ingestion rates in adults (Stanek et al., 1997) likewise included a number of improvements over our prior (1990) study. These included: 1) a larger number of subjects (10) and days (28) of participation; 2) an improved study design that considered seven consecutive days of fecal sampling; 3) improved selection of soil tracers; 4) a broader range of soil ingestion validation; and 5) an enhanced capacity for additional assessments including particle size of the soil ingested. In this study, one of the participating adults had an unusually high soil ingestion estimate (2 grams) on the first day of the study week. In fact, on that day, this subject had 4 times higher freeze-dried fecal weight than on any other day of the study, suggesting that his excretion on that day reflected a 3-4 day accumulation, instead of just one day, as assumed in the calculations. In consequence, the 95th percentile ingestion rate from this study (331 mg/day), which is driven by that result for one subject, is uncertain, unstable, and artificially inflated. In these circumstances, I recommend that EPA use the upper 75th percentile value from this study, which was 49 mg/day, as the basis for an upper bound soil ingestion rate of 50 mg/day for adults and older children. For the central tendency estimate, I recommend use of an ingestion rate of 10 mg/day. This is consistent with the

mean soil ingestion rate observed in our 1997 study (6 mg/day) and would represent half of the central tendency rate that I have recommended for young children.

I appreciate the opportunity to review these materials. Please do not hesitate to contact me if you have any further questions.

Sincerely,

Ed Calabrese

Edward J. Calabrese, Ph.D.
Professor of Toxicology
Director of the Northeast Regional
Environmental Public Health Center

EJC/ps

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COMMENTS ON THE INTERIM-FINAL
BASELINE ECOLOGICAL RISK ASSESSMENT REPORT
CENTREDALE MANOR RESTORATION PROJECT SUPERFUND SITE
NORTH PROVIDENCE, RHODE ISLAND
SEPTEMBER 2004

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AMEC Earth and Environmental, Inc.
15 Franklin Street
Portland, ME 04101

1.0 INTRODUCTION

In September 2004, EPA Region I released the Interim-Final Baseline Ecological Risk Assessment (BERA) Report for the Centredale Manor Restoration Project Superfund Site, North Providence, Rhode Island (EPA, 2004). The BERA report was prepared by MACTEC Engineering and Consulting, Inc., under contract to Battelle Corporation, and was submitted to the Department of the Army, U.S. Army Corps of Engineers, New England Division under Contract No. DACW33-01-D-0004, Delivery Order No. 1. The BERA document presents an assessment of potential hazards for a number of sensitive receptor communities and media. Receptors communities include floodplain invertebrates, lotic and lentic aquatic invertebrates, demersal (white sucker) and pelagic (largemouth bass) fish, and piscivorous (blue heron, belted king fisher and river otter), insectivorous (tree swallow and little brown bat), vermivorous (American woodcock and short-tailed shrew), and omnivorous wildlife. Media evaluated include soil, sediment and surface water. Weight of evidence hazards are separately estimated for receptors from five Exposure Areas (EAs) that consist of reaches and wetlands associated with the Woonasquatucket River: EA1, Allendale Pond; EA2, Lyman Mill Pond; EA3, Manton Pond; EA4, Dyerville Pond and EA5, the Fogarty Center. Ecological risks are also derived for surface soil at the Fogarty Center, EA5. Greystone Mill Pond and Assapumpset Pond are designated as the upstream background area and reference area, respectively.

At the request of Emhart Industries, Inc., AMEC Earth & Environmental, Inc. prepared these comments on EPA's BERA document. The comments focus on those calculations, exposure scenarios and risk assessment topics that have the greatest implications on setting target remediation goals and assessing remedial options. Accordingly, the comments do not address aspects of the report that do not appear to be influential in evaluating remedies or determining outcomes for the site.

Several significant errors were noted in our review of the report. In particular, in the Executive Summary and Conclusions of the BERA, EPA mischaracterizes the findings of the technical sections of the BERA for demersal and pelagic fish populations. Our review of the technical portions of the BERA shows that EPA *does not* find a significant risk of harm to these receptors from exposure to site-related chemicals. However, EPA erroneously reports the opposite of those findings in the Executive Summary and Conclusions. This erroneous conclusion is then propagated through to the ecological preliminary remediation goal (PRG) document, and EPA unnecessarily computes PRGs for these receptors (EPA, 2005a). This is so significant an error

that it by itself warrants that EPA revise the BERA and the PRG document to correct the text and remove these receptors. More detail regarding this substantial shortcoming is provided in the comments below along with other technical comments to EPA's BERA.

Section 2.0 of this document presents general comments regarding the BERA. Section 3.0 provides detailed comments on the problem formulation component. Section 4.0 provides detailed comments on the aquatic invertebrate community risk analysis. In Section 5.0, comments on the floodplain invertebrate community risk analysis are presented. Sections 6.0 and 7.0 present comments on the demersal and pelagic fish community risk evaluations, respectively. Section 8.0 presents comments on the piscivorous mammal and bird population risk evaluation. The insectivorous mammal and bird population risk evaluations are critiqued in Section 9.0, while Section 10.0 presents comments on the omnivorous mammal and bird population risk evaluation. Finally, Section 11.0 presents comments on the ecological risk assessment uncertainty assessment.

2.0 GENERAL COMMENTS

EPA incorrectly summarizes the technical findings of the BERA in the Executive Summary and Conclusions.

EPA states in the Executive Summary and in the report Conclusions (Section 11) that both demersal and pelagic fish populations may be at substantial risk of harm from site-related chemicals of concern. In stark contrast, however, the technical portion of EPA's BERA concludes the exact opposite. Specifically, Section 5.4 of the BERA states the following:

> "Integrating these results, it is concluded that the demersal fish community associated with the study area ***is not*** [emphasis added] at substantial risk of harm due to the presence of contaminant release from the Centredale Manor source area."

Similarly, Section 6.4 of the BERA concludes the following:

> "In summary, the pelagic fish populations associated with the study area are not at substantial risk of harm due to the presence of contaminant release from the Centredale Manor source area."

Additionally, Tables 89 and 98, which present the weight of evidence integration for these receptor populations, supports the idea of no significant risk. Also, the individual report

Appendices, which house the detailed data of the various measurement endpoints, also suggest that no significant risk is present to demersal and pelagic fish populations. Finally, Table 160, which is the Risk/Magnitude summary table, incorrectly reports the findings of the fish population studies and is inconsistent with the data reported in Tables 89 and 98.

Given the level of technical review that these documents reportedly undergo prior to release to the public, EPA and its contractors should not have made this error and allowed it to propagate through to subsequent documents, such as the PRG document. This is unsettling because this error was neither identified by the report authors nor by the other technical reviewers. If these technical reviewers did not find this error, it is highly unlikely that the public would have identified the errors because of the level of technical detail that would have to be sorted through in order to reach the correct conclusion; namely that there is no significant risk due to site-related compounds. Moreover, because the incorrect conclusions are reported in the Executive Summary, the Conclusions Section, and in the final risk assessment summary table – the sections of the report most often read by non-technical readers – this erroneous conclusion would likely be taken as an indisputable fact. Even EPA's contractor that conducted the BERA and wrote the report adopts this erroneous conclusion as fact as it inappropriately includes these receptors in the PRG document (EPA, 2005a). EPA's misrepresentation of its data, analysis and conclusions for these receptors is a gross error for both the BERA and the PRG document.

Editorial Comment

Tables ES-1 and ES-2 are not provided in the draft BERA.

3.0 PROBLEM FORMULATION

EPA fails to adequately incorporate important and relevant site-specific information in the life history summaries.

In the BERA, EPA fails to incorporate relevant and significantly important information in the potential receptor life history discussions. Additional discussion concerning the presence or absence of the evaluated receptors at the site should be added to the life history summaries as a means of verifying the conceptual site exposure model used in the BERA. For example, the discussion concerning the little brown bat does not address whether or not there are any daily roost locations or caves for habitat on or near the site. This becomes an important issue during the risk characterization of the receptors. In particular with the little brown bat, the calculated

hazard quotients (HQs), which exceed one for some chemicals, are not considered critical since bats are not reported in the area. EPA should augment the existing discussion with the relevant site-specific habitat information.

EPA should include a discussion of multivariate statistical analysis of the dioxin/furan congener results.

Although the concentration histograms shown in Figures 4 through 7 are useful to portray the relative dioxin/furan congener patterns, there may be some benefit to perform more detailed multivariate statistical analyses, such as principal components analysis (PCA) on these results. Since PCA is performed using normalized concentration data (such as to total dioxin/furans or total PCBs) it can be used to elucidate co-varying congener patterns that could be useful for determining the fate and transport of the congeners in the environment, as well as their interrelationships with other parameters. EPA has conducted a similar analysis as part of the Remedial Investigation (RI) report. Because the PCA analyses presented in the RI report were specifically designed to assess potential fate and transport issues (e.g., the data were not normalized prior to the assessment), those data may not be adequate for use in the BERA. If appropriate, EPA should incorporate to the extent possible the information from the PCA analysis already conducted. However, if the RI PCA analysis is not adequate for use in the BERA, EPA should conduct such an analysis and include the findings in the BERA.

The BERA does not adequately document the basis for selection of Assapumpset Pond as a suitable and appropriate reference area.

It is critically important that a reference area chosen for ecological comparisons is physically similar and also similar in hydrologic characteristic, structure, function, and ecological diversity, but without being impacted by site-related compounds. Based on the text provided by EPA in the BERA, there is no way to assess the adequacy of Assapumpset as a suitable and appropriate reference area. EPA needs to provide a more detailed description of why this water body is believed to provide an appropriate reference area for each reach evaluated in the BERA.

4.0 AQUATIC INVERTEBRATE COMMUNITY RISK EVALUATION

Additional detail concerning the calculation method for Toxic Equivalence Quotients should be provided.

There is insufficient discussion concerning the Toxic Equivalence Quotients (TEQs) in Section 3.1.1.1. In particular, it is not clear how non-detect congener results are used for the TEQ calculation. Depending upon the congener that is not detected, significantly different TEQ values could be calculated if different types of substitution methods are used. If a determination was made that the TEQ values were insensitive to the substitution methods, then this should be presented in the BERA text.

In addition, it is not clear how the 95th percentile exposure point concentration (EPC) values for TEQ are calculated. Two possible methods may result in substantially different estimates. These two methods may include 1) either calculating the TEQ for each individual sample and then calculating the summary statistics, or 2) calculating the 95th percentile for each congener and then summing those values. The specific method used to calculate the TEQ should be specified and the impact on risk estimates should be discussed thoroughly in the uncertainty section.

EPA should provide justification for normalizing the geometric mean values for sediments or biota by the geometric mean values for total organic carbon content or lipids (respectively) for use in deriving Biota-Sediment Accumulation Factors (BSAFs).

The BERA takes the approach (in Section 3.1.1.2) of deriving BSAFs by normalizing the geometric mean biota or sediment results using the geometric mean lipid content or total organic carbon (TOC) concentrations, respectively. It then calculates the ratio of the lipid-normalized fish concentration to the TOC-normalized sediment concentration. This yields a single BSAF value per chemical and per exposure area. EPA does not provide adequate justification for this approach. Moreover, it is unclear why this approach was taken since paired lipid and TOC data are available for many of the individual samples. It would have been more appropriate to first normalize the biota or sediment results to their respective lipid and TOC data, and then calculate BSAFs. In those cases where lipid or TOC data were lacking, a substitution method using the average lipid or TOC results could have been used, or the data could have been censored from the BSAF calculation. This would also allow the development of bounding estimates for the BSAFs which can then be evaluated quantitatively as part of the uncertainty assessment.

In addition, the BERA lacks any discussion that supports the presumption that the geometric means of the TOC or lipid data are representative of the central tendency value for these parameters. Also, EPA does not provide any supporting analysis or information to indicate that the chemical concentrations were log-normally distributed and, thus, the central tendency estimate would be best represented by the geometric mean sediment concentration. EPA should present an evaluation of the distribution of the TOC, lipid and chemical concentration data and identify the appropriate central tendency value for those data prior to their use in the BERA.

The BERA lacks specificity regarding whether filtered or unfiltered surface water sampling results were evaluated.

It is well known that the filtered fraction of the surface water represents the true exposure for potential toxicity or chemical uptake (the dissolved fraction). It is unclear from the discussion presented in Section 3.1.2 whether the EPCs are derived from filtered or unfiltered water samples. Since the benchmarks are typically derived from dissolved phase chemical concentrations, the use of unfiltered samples would likely lead to overestimated hazard quotient (HQ) values as reported in Section 3.3.1. EPA should provide additional information regarding whether the samples were filtered or simply grab samples. In addition, if the data are not filtered, EPA should discuss the limitations of using such data in the BERA.

EPA fails to adjust the sediment screening benchmarks for site-specific total organic carbon content of the sediment.

The sediment benchmarks (e.g., Table D-2) used for the HQ analysis of the potential impacts to aquatic benthic communities assume a total organic carbon (TOC) content of 1% for the sediments. This assumption is not unreasonable for use in a screening level ecological risk assessment (SLERA), but the BERA should correct these screening benchmark values with site-specific TOC data to minimize the uncertainty in the assessment. EPA should correct the screening benchmark values to account for site-specific TOC content.

The sediment bioassay was conducted using sediments that were selected based on concentration but there is no indication that grain size and TOC were also considered in the selection process.

There is no indication in the selection of sampling locations for the sediment bioassay that grain size and TOC were considered as part of the selection process. These factors can have a substantial effect on the habitat for the invertebrates and the bioavailability of the chemicals in

the sediments tested. As such, EPA should provide a discussion on the physical sediment characteristics and the limitations that may be placed on the bioassays as a result of the physical attributes.

EPA does not discuss the result of the reference area sediment bioassay assessment.
Section 3.3.3 of the BERA indicates that background/reference area samples did not meet the minimum survivability requirements for chironomids. The BERA would benefit from a discussion concerning the possible causes of this issue and how well the control sediment would be a reasonable surrogate for the background/reference area data. This discussion is especially important in light of the reported impacts for the sediment bioassay downstream of the site. These impacts may not be solely the result of chemical concentrations if the factors that impacted minimum survivability requirements for chironomids are also factors in the downstream areas.

The BERA should report the p-values that are obtained in the comparison of the sediment toxicity results and the chemical concentration data.
The correlation coefficients discussed in Section 3.3.3 (and presented in Tables 47 through 49) for the comparison of the sediment toxicity and chemical results should include the p-values. The latter, in conjunction with the reported correlation coefficients, are a better indicator of "significance" than merely reporting the correlation coefficients.

EPA's conclusion of an impact to the aquatic invertebrate lentic communities fails to incorporate the results from the benthic community assessment that showed no impact.
The conclusion in Section 3.4 of the BERA that there is an impact to the aquatic invertebrate lentic communities is based on a sediment toxicity evaluation using non-indigenous test organisms, and disregards the results from the biological assessments of the aquatic communities, which show no apparent impact (Appendix E). The site-specific results from the biological assessment should have been given greater weight in the assessment of whether the benthic macroinvertebrate communities are impaired by the chemicals found in the sediment.

The statement that there are no relevant published BSAF data for emergent/aquatic insects and earthworms is not supported.
The focus of Section 3.2.1 is on BSAF data derived for fish, but there are a number of publications in the peer-reviewed literature that derive BSAF data for emergent/aquatic insects and earthworms. In addition, BSAFs could be derived for similar species or from studies where

BSAF derivation may not have been the primary focus but where the data are available (e.g., Maul et al., 2006).

There are several studies which examined dioxin uptake by earthworms from sludge or biosolids disposal (e.g., Matscheko et al., 2002; Meyn et al., 1997) or from soils (e.g., Jager et al., 1998; Sample et al., 1998). These studies also provide information for organic chemicals other than dioxin. EPA should use these other sources of information to derive BSAFs for earthworms where empirical data are not available because these sources generally are more reliable than the use of default values.

There is limited discussion of the relative impact of the Waste Water Treatment Plant (WWTP) discharges on the aquatic community.

While the problem formulation provides a substantial discussion of the need to evaluate whether the WWTP is adversely impacting the river, there is limited discussion of the WWTP in the risk characterization and therefore no quantitative analysis of what impacts the WWTP may be causing. Since these impacts can be substantial in some water bodies, it is recommended that the risk summary in Section 3, the uncertainty analysis, and the risk characterization provide some discussion of the relative contribution of the WWTP releases to the reported risks.

In addition, EPA should discuss how discharges from combined sewer outfalls (CSOs), in addition to the WWTP discharge, may influence the chemical results of the BERA.

The substantial contribution of pesticides to the total hazard quotients is not discussed.

Pesticide residues in water and sediment contribute greatly to the HQs that have been calculated for surface water and sediment. For example, in the Lyman Mill reach, 67 percent of the HQ can be attributed to pesticides while at Dyerville, 65 percent of the HQ is due to pesticides. As pesticides may not be site-related, it is important to discuss their relative contribution to total risk estimates so that the risk manager and the public understand that even without the site-related chemicals, the ecological receptors evaluated may still be at risk due to the presence of other compounds.

Editorial Comments

Several of the citations included in the tables (e.g., Table D-2) are not included in the References section. These should be added for completeness.

Too many significant digits are reported for the correlation coefficients in Tables 47 through 49. Three should suffice, unless dictated by the input data.

5.0 FLOODPLAIN INVERTEBRATE COMMUNITY RISK EVALUATION

There is insufficient discussion of the representativeness of using several of the benchmarks in the uncertainty assessment.

The risk characterization (Section 4.4) and uncertainty (Section 4.3.4) discussions should contain a more detailed examination of the benchmarks used to assess the floodplain invertebrates. In particular, the benchmarks based on either EPA's EcoSAR software or the equilibrium partitioning (EqP) approaches (e.g., Table D-3), which are based on estimated surface water toxicity, may not be fully extrapolated to floodplain invertebrates without much uncertainty. This uncertainty may explain, in part, the high HQ values that are calculated, which do not support the results of the community assessments that show no adverse effects on the floodplain invertebrates.

EPA should provide additional discussion concerning the uncertainty in the critical body residue (CBR) values.

Although the uncertainty in the CBR values used to evaluate earthworm concentrations is discussed to a limited extent in the uncertainty section (Section 4.3.4), EPA should discuss other sources of uncertainty for using CBR data for earthworms. Lanno et al. (2004) summarized several of the key issues related to chemical bioavailability for earthworm uptake. This may become especially important when extrapolating CBR estimates of unrelated invertebrates to earthworm. For example, the 2,3,7,8-TCDD CBR benchmark for earthworms is based on crayfish (Table G-9) following aqueous exposure.

6.0 DEMERSAL FISH POPULATIONS RISK EVALUATION

There is no statistical assessment of the analytical results.

Section 5.1.1 (and the BERA in general) would benefit from some statistical analysis of the analytical results. Such analyses would provide insight to help answer a number of important questions. For example, of the species of demersal or pelagic fish collected, are there any statistically significant differences in their chemical concentrations? Are there any statistically significant differences by species between the different ponds? Such data would be helpful for purposes of pooling some of the fish results for further evaluation in the BERA. It does not appear that a statistical analysis of the fish data is performed elsewhere, such as part of the

Remedial Investigation Report (EPA, 2005b), although the RI report contains statistical summaries for other media, including soil, sediment, surface water and groundwater.

EPA's conclusion that the ichthyoplankton survey was inconclusive is not supported by the result of the study.

The uncertainty discussion (Section 5.3.5) states that the ichthyoplankton survey results were inconclusive since the survey was performed during two of the three planned survey events. Based on information provided in Appendix E, the ichthyoplankton survey was performed in early-May and mid-June, which should adequately capture most of the spawning period of the fish species present in the system. The ichthyoplankton report did indicate some logistical difficulties with plankton tows in Assapumpset Pond due to heavy macrophyte growth. However, the conclusion of Appendix E is that "no adverse impacts of the Centredale Manor Site on the ichthyoplankton communities in Greystone Mill Pond, Lyman Mill Pond, and Assapumpset Pond were evident." Based on these findings, EPA should not state that the study was inconclusive.

The estimated effect (EC) values from the Early Life Stage (ELS) Study may not reflect actual waterborne exposure to 2,3,7,8-TCDD.

Filtered water represents the bioavailable fraction for waterborne exposures. The exposure concentrations used in the ELS study (2 to18 ng/L) are similar to the unfiltered surface water samples collected during the remedial investigation. However, the unfiltered samples contain particles onto which the 2,3,7,8-TCDD is likely to be sorbed. The data from the unfiltered samples do not accurately represent the plausible dissolved phase concentrations of 2,3,7,8-TCDD because most, if not all, of the 2,3,7,8-TCDD are likely bound to particles in the water samples. EPA recognizes this fact in the Revised Interim-Final Baseline Human Health Risk Assessment (EPA, 2005c), which explains that "the dioxin/furans that have been measured in surface water may be associated with suspended particulate matter, perhaps derived from sediments. This is likely because 2,3,7,8-TCDD has such a low solubility in water and it has a high affinity for sediments that contain organic carbon as well as for high lipid materials such as fish."

The concentrations of dissolved (i.e., bioavailable) 2,3,7,8-TCDD in site surface water is likely to be much lower than the range of concentrations used in the ELS study. This is supported by the comparison of the eggs, larvae and fry pathological abnormalities between the ELS study and the field collected fish (as discussed in Appendix E). Although the latter were slightly older

than the fish from the ELS study by several weeks, they did not exhibit as many anomalies as the test organisms from the ELS study. Presumably, the differences are primarily due to the fact that the *in-situ* environment (TSS) renders some portion of the 2,3,7,8-TCDD non-bioavailable. Nonetheless, the EC values generated from the ELS study were used as the benchmark for the HQ analysis.

EPA's use of fish tissue concentrations in only large fish is likely to overestimate concentrations in the fish population as a whole.

The fish tissue concentrations used for comparison with benchmarks were measured in larger adult fish. Thus, these concentrations likely overestimate concentrations in younger life stages and, thus, may overestimate risks to those life stages. Ideally, EPA should have collected chemistry data on early life stage fish populations for use in the community assessment as well as for developing a more robust exposure point concentration for use in the piscivorous bird and mammal food chain modeling. EPA should discuss the fact that the fish tissue database is limited to mature fish samples and thus does not include samples from early life stages.

Editorial Comments

The ELS figure provided on page 5-6 has a very odd scale on the X-axis. The numeric values shown as major tick marks incremented as 10^0, 10^2, 10^4 and 10^6, yet a conventional log scaling is used for the minor tick marks. The comparable figure in Appendix H (ELS Toxicity Report) shows a proper log scale. The shape of the curves and placement of data points are comparable, but the X-axis should be corrected to show the proper scaling for the ELS figure provided on page 5-6.

7.0 PELAGIC FISH POPULATIONS RISK EVALUATION

The uncertainty assessment should include a discussion of the relevance of the ELS study on the pelagic fish.

The uncertainty section (Section 6.3.4) should include a discussion of the relevance and applicability of the ELS study, based on channel catfish, on the pelagic fish that were evaluated in Section 6. Differences in the morphometry of the eggs between the smallmouth bass (the representative receptor for the pelagic fish) and the channel catfish can influence the applicability and relevance of the ELS results.

EPA's weight-of-evidence assessment underweights the significance of the biological survey performed to support the pelagic fish risk assessment.

Although we agree with the overall conclusion that there are no apparent impacts to the pelagic fish in the different ponds due to the release of site-related chemicals, it appears that the weight-of-evidence assessment (Section 6.4 and Table 98) underweights the significance of the biological survey performed as part of this assessment. The HQ assessment focused on the use of CBRs, which are typically the lowest and most conservative values available from the referenced literature. Higher CBR values are available in many cases, and may be more relevant to the fish species that are present in the ponds at the site. Use of more realistic CBRs would support the observations from the biological survey that show no likely impact to the pelagic fish population. Therefore, the CBR results presented in Table 98 should be weighted less than the population results, especially if these results are used to derive PRGs.

The use of fish tissue concentrations in only large fish is likely to overestimate concentrations in the fish population as a whole.

As stated above, the fish tissue concentrations used for comparison with benchmarks were measured in larger adult fish. Thus, these concentrations likely overestimate concentrations in younger life stages and, thus, may overestimate risks to those life stages. EPA should include this fact in the uncertainty section.

The assumption for maternal to egg transfer used to derive the PRG for dioxins (0.7) in fish is inadequately supported.

The evaluation of the sensitive endpoint of eggs in fish assumes a maternal to egg transfer factor of 0.7. The limited discussion in the BERA document indicates that this number is derived from the literature, but specific references are not provided. A limited literature review indicates that a lower value was more supportable. For example, in their oral ingestion study of 2,3,7,8-TCDD using adult brook trout, Tietge et al. (1998) reported that the concentration in the spawned eggs was 39% of the whole body 2,3,7,8-TCDD concentrations. This value represented the slope of the regression between whole body concentrations, which ranged from approximately 100 to 1,900 pg/g fish, and spawned egg concentrations, which ranged from about 50 to 700 pg/g egg. The regression coefficient of determination was 93%. The authors did not report confidence bounds on the slope.

Heiden et al. (2005) reported that the concentration in eggs was 3% of the carcass 2,3,7,8-TCDD concentrations. Adult female zebrafish were exposed to 1,2,7,8-TCDD in their diets

using four treatment groups (10, 40, 100 and 270 ng/g diet) with tissue concentrations monitored after 5, 10, 15 and 20 days. As with the study by Tietge et al. (1998), this value represented the slope of the regression between carcass concentrations, which ranged from approximately 1 to 35 ng/g fish, and egg concentrations, which ranged from about 0.1 to 1.3 ng/g egg. The regression coefficient of determination was 87%, and the authors did not report confidence bounds on the slope.

Finally, the value of 0.7 is twice the original proposed value (0.35) from the BERA Work Plan. The latter is clearly more supportable by the references presented in this comment. EPA should not imply that the transfer factor of 0.7 is appropriate, and should instead use the site-specific factor of 0.35, which is supported by Tietge et al. (1998) and Heiden et al. (2005).

8.0 PISCIVOROUS MAMMAL AND BIRD POPULATIONS EVALUATION

EPA uses unrealistic Exposure Durations for the Great Blue Heron and Kingfisher.

Section 7.1.1 states that the Great Blue Heron (and also the Kingfisher) are seasonal migrants and would occupy the site area during a portion of the year, migrating to warmer climates during the winter. However, Table 18 does not reflect this seasonal use, and the exposure duration (ED) term (Tables I-1 though I-3) are assumed to be one for both species. This would clearly overestimate the potential exposure, and subsequent estimated risk, for these two species. EPA should use a seasonal use factor that is more representative of these species for this area. A reasonable seasonal use factor for these receptors would be 0.5.

EPCs used to estimate exposures to piscivorous birds and mammals are not likely to be representative of their dietary concentrations.

The BERA inappropriately models uptake in piscivorous birds and mammals using EPCs for fish tissues that are based on large fish only. Piscivorous birds and mammals will consume fish of all different sizes, including very small fish. Since smaller fish are likely to be more plentiful than larger fish, it is likely that their diets will be comprised largely of smaller sized fish. Since smaller (i.e., younger) fish tend to have lower concentrations of the COCs that tend to bioaccumulate over time, it is likely that the use of concentration data for only larger fish will substantially overestimate actual exposures. While this issue is acknowledged in the BERA, the magnitude of potential bias is not discussed. Ideally, EPA should have collected chemistry data on early life stage fish populations for use in the community assessment as well as for developing a more robust exposure point concentration for use in the piscivorous bird and

mammal food chain modeling. EPA should discuss the fact that the fish tissue database is limited to mature fish samples and, thus, does not include samples from early life stages. In addition, EPA should quantify the potential magnitude of the overestimation of exposure to COCs.

The assumption that 100% of the site foraging frequency (SFF) for Great Blue Herons occurs at each of the reaches evaluated is not realistic and is likely to greatly overestimate their potential for exposure.

Herons have very large foraging ranges. According to EPA's *Wildlife Exposure Factors Handbook*, they may forage as far as 24 km from their nesting areas and the mean foraging distance is 3.1 km (approx. 2 miles) (EPA, 1993). Given that there is substantial foraging habitat within a 2 mile radius from the site that is either upstream or is not part of the Woonasquatucket River, to assume that receptors spend 100 percent of their foraging effort in any of the reaches evaluated is unrealistic. For herons, it would be more appropriate to assume that they may feed in any of the ponds/reaches and to develop an EPC based on all reaches combined. This would then allow a SFF of 100% to be used with more credibility.

EPA should consider other sources for the PCB congener biomagnification factors (BMFs) for the avian receptors.

Although the referenced study (Braune and Norstrom, 1989) is fairly comprehensive, the BMFs were derived using one fish species (alewife). EPA should evaluate these and compare them to those reported for birds of other feeding guilds or from more contemporary references (Henny et al., 2003). These should be evaluated for completeness.

BMFs used to estimate total Aroclor PCB uptake may not be accurate for mammals.

Section 7.1.2 references the study from Leonards et al. (1997) to estimate the potential BMF from the diet to mink liver for Aroclor PCBs. Review of this paper shows that the authors did not assess Aroclor PCBs, but calculated total PCBs based on the sum of 28 PCB congeners. The potential uncertainty in using these BMFs to represent Aroclor PCB uptake should be discussed.

The study by Leonards et al. (1997) also reported 25 PCB congener BMFs, but 28 were reported in Table J-13. Based on review of this table it appears that the mean BMFs for the tetra- and penta- homolog groups were used as surrogate values for the three additional

congeners (PCB-81, PCB-114 and PCB-123). This should be discussed more fully in the revised text.

The source for the uncertainty factors used to derive the toxicity reference values (TRVs) is not provided.

The citation supporting the uncertainty factors for the derivation of the TRVs is not provided in Section 7.2.1. If this information was presented in the Risk Assessment Work Plan, then this document should be cited.

Additional uncertainties are not addressed.

There are a number of additional uncertainties that are not addressed and should be added to the discussion presented in Section 7.3.3. Some key additional items include: (1) the potential impact of the absence of any population data which may contradict the HQ analysis; and (2) the potential impact of the exposure assumptions on the HQ analysis results. The risk assessments performed for many of the other receptors evaluated in the BERA are not restricted to an HQ analysis alone, but include field observations and related activities which are able to show that potential risks are likely *de minimis.*

In the interest of transparency, EPA should disclose the fact that a comprehensive avian survey has not been conducted and that uncertainty in the avian risk evaluation is enhanced in the absence of such a survey. Potentially useful questions remain unresolved due to this methodological shortcoming; for example, (a) are herons frequently sighted in all of the evaluated ponds; (b) are the habitats suitable for forage by herons; and (c) are there known heron rookeries in the area?

There are several exposure assumptions (e.g., home range for herons, assumption of seasonal use factor of one for migrant species) that would bias high the resulting HQ calculations. It would be beneficial to discuss whether these key assumptions in this section are representative of regional conditions or conservative assumptions, since this clearly affects the interpretation of the risk results.

Editorial Comments

The three studies referenced in Section 7.1.2 (Braune and Norstrom, 1989; Leonards et al., 1997; and Tillit et al., 1997) are not included in the References section of the draft report. In addition, should the correct citation be Tillit et al, *1996*? These should be added and corrected for the final version.

Table J-12 (BMFs for avian receptors) is missing the column headers. Review of the referenced paper (Braune and Norstrom, 1989) shows that these should be "mean" and "SD" for the split columns under the biota or tissue types.

9.0 INSECTIVOROUS MAMMAL AND BIRD POPULATIONS EVALUATION

The estimated tissue concentrations in tree swallow eggs and nestlings, which are estimated using the same BMFs used for piscivorous mammals (Section 7.1.2), may not be appropriate due to differences in dietary components.

The BMFs used to calculated tissue concentrations in swallow eggs and nestlings are those BMFs that were calculated by Braune and Norstrom (1989) based on measured concentrations in whole body, liver and egg tissues of herring gulls and the measured concentrations in alewife tissue, which herring gulls commonly consume. There are, however, substantial differences between the lipid contents in alewives and emerging insects that are consumed by tree swallows. In addition, tree swallows consume a variety of invertebrates that may have substantially different potential to accumulate the COPCs. In addition, these BMFs have been combined with site-specific BSAFs that are also uncertain. The high level of uncertainty associated with these estimates should be thoroughly discussed.

EPA fails to use a duration factor for modeled exposures to account for the seasonal migration of tree swallows or the winter hibernation of bats.

Tree swallows are migratory birds that arrive in New England in early April and generally depart for their southward Fall migration in September. Thus, they would only be present in the study area for 5 or 6 months of the year. The exposure modeling does not, however, correct for the fact that only a portion of the year will involve potential exposure to site-related contaminants.

Similarly, brown bats are active from early spring to early fall but then move to their winter habitats for the remainder of the year. As a result, their only potential for exposure will occur during their active period. EPA should incorporate a duration factor of 0.5 to account for the migration of these receptors.

10.0 OMNIVOROUS MAMMAL AND BIRD POPULATIONS EVALUATION

It is unclear whether the home range of the raccoon was properly accounted for in the floodplain exposure risk estimates.

Section 9.1 of the document would benefit from some discussion of how the home range for the raccoon (108 hectares; Tables I-10 and I-11) is used in the dose calculations. It appears that if the SFF (the ratio of the site area divided by the home range) was greater than one, then no discussion is provided in the exposure assumptions development. For this particular case, the raccoon's home range would be equivalent to a length of about 3,800-ft (if home range is assumed to be a circle) or about 13,600-ft (if home range is assumed to be a square). If these values were greater than the available shoreline, then a value of less than one could be used as the SFF to assess potential risks. Assuming a value of one for the SFF when a smaller value would be more representative would overestimate the potential risks. At a minimum, it is recommended that the SFF calculation be addressed for each of the receptors and EAs, and that these be added to the appropriate tables for completeness.

Editorial Comment

There appears to be reference citation errors for the two EPA documents shown at the end of the paragraph in Section 9.3.

11.0 ECOLOGICAL RISK UNCERTAINTY ANALYSIS

The uncertainty associated with the high background mortality observed in the bioassay evaluating the survival, growth, and reproduction needs to be more fully discussed.

There is inadequate discussion of the high background mortality that was observed for assessment endpoint 1a. This discussion needs to be expanded and evaluated further to provide more perspective on the significance of the potential risks to support risk management decisions.

The uncertainty discussion is qualitative and no quantitative discussion of the relative importance of the different uncertainty components is provided.

The uncertainty discussion is general and qualitative, and only a minimal effort is made to estimate the potential biases in the input parameters and their impacts on the calculated risks. The use of a more quantitative assessment can provide bounding estimates for the risks, and therefore provide better supporting information for any risk management decisions. For example, often the lowest and most conservative CBR values are used in the BERA to assess

fish concentrations. Other higher CBR values are available in many cases that may be more relevant to the fish species that are present in the ponds at the site. The potential bias from using the more conservative CBR values to calculate the risks should be presented.

This section would also benefit by improving the tie-ins to the uncertainty discussions provided with each receptor. It is also unclear why the method used for each of the receptor evaluations, based loosely on the method from Menzie et al. (1996) is not applied to this section, at a minimum, for consistency.

The derivation of Exposure Point Concentrations does not include any discussion of the uncertainty related to using different methods to estimate the Upper Confidence Limits (UCLs).

The discussion presented in Section 10.2.1 [*Development of Exposure Point Concentrations*] focuses on the number (or type) of samples that are included in the EPC calculations, but does not address the potential effect of the different metrics used to estimate the UCLs (i.e., those based on the t-distribution, H-distribution, or the calculated mean). Such a discussion would be beneficial since there is mixing of the UCL types in the risk calculations, yet the hazard indices are reported as the sum of the hazard quotients. Depending upon the mixture of UCL estimates (normal, lognormal, non-parametric, Chebyshev, etc), the combined UCL may be overestimated. If, for the principal risk drivers, combining the different UCL metrics does not change the risk outcome (i.e., HQ values above a threshold are still calculated), then this should be stated in this section.

In addition, this subsection should address how alternative approaches to addressing non-detect results in the calculation of PCB- or dioxin-TEQs can affect the calculated risks. The methodology used to derive the TEQ was not presented in the BERA. If discussed in the preceding project plans, it would be beneficial to cite the appropriate section and document for completeness, and to address any of the uncertainty in this section. If the TEQs were derived from PCBs or dioxin congener results with a low detection frequency, there is the potential that the calculated TEQ may substantially overestimate the "true" value depending upon the method used for addressing the non-detect results.

The potential effect(s) of the "less-than" values for the dioxin and PCB congener TEFs for fish on the risk assessment are not discussed.

Although it is agreed that the TEF-TEQ approach represents the current policy for assessing potential dioxin risks, the referenced study (van den Berg et al., 1998) included "less-than" values for two dioxin/furan congener TEFs and eight PCB congener TEFs assigned to fish. Section 10.3.2 should include some discussion of whether these values are used as reported, and what potential impact they may have (if any) on the risk estimates.

There is no discussion of the cumulative impact of combining a number of conservatively biased assumptions and modeling approaches on the final risk estimates.

The lack of site-specific data for portions of many of the exposure pathways and receptors that are evaluated in the BERA results in the need to model uptake values and potential exposures to the receptors of concern. Generally, very conservative assumptions and parameters have been used to ensure that potential exposures and risks are not underestimated. While any one of these assumptions may be a reasonable upper-bound estimate for a given parameter, when multiple assumptions and parameters are combined and conservative TRVs are used as points of comparison, this may result in calculated HQs that are not reasonable or realistic. It is important to address this issue and the potential magnitude of the potential biases introduced by the approach in order to provide risk managers with all of the information that will be needed to make risk management decisions.

There are no tissue data available for several of the wildlife receptors evaluated.

Unfortunately, because tissue samples were not collected for many of the receptors, EPA found it necessary to calculate hazard quotients and model uptake for predators of those receptors using assumptions about what tissue concentrations were likely to be. Given the large amount of uncertainty associated with many of the values used to parameterize those uptake models and the tendency to make conservative assumptions in deriving estimated values, it is likely that these exposures and HQs have been overestimated. EPA should transparently acknowledge the fact that the lack of tissue data has greatly increased the uncertainty of the risk analysis.

The EC values from the ELS Study may not reflect actual waterborne exposure to 2,3,7,8-TCDD.

Filtered water represents the bioavailable fraction for waterborne exposures. The exposure concentrations used in the ELS study (2 to18 ng/L) are similar to the unfiltered surface water samples collected during the remedial investigation. However, the unfiltered samples contain particles onto which the 2,3,7,8-TCDD is likely to be sorbed. The data from the unfiltered samples do not accurately represent the plausible dissolved phase concentrations of 2,3,7,8-TCDD because most, if not all, of the 2,3,7,8-TCDD are likely bound to particles in the water samples. EPA recognizes this fact in the Revised Interim Final Baseline Human Health Risk Assessment (EPA, 2005c), which explains that "the dioxin/furans that have been measured in surface water may be associated with suspended particulate matter, perhaps derived from sediments. This is likely because 2,3,7,8-TCDD has such a low solubility in water and it has a high affinity for sediments that contain organic carbon as well as for high lipid materials such as fish."

The concentrations of dissolved (i.e., bioavailable) 2,3,7,8-TCDD in site surface water is likely to be much lower than the range of concentrations used in the ELS study. This is supported by the comparison of the eggs, larvae and fry pathological abnormalities between the ELS study and the field collected fish (as discussed in Appendix E). Although the latter were slightly older than the fish from the ELS study by several weeks, they did not exhibit as many anomalies as the test organisms from the ELS study. Presumably, the differences result primarily due to the fact that the *in-situ* environment (TSS) renders the some portion of the 2,3,7,8-TCDD non-bioavailable. Nonetheless, the EC values generated from the ELS study are used as a conservative benchmark for the HQ analysis, which may overestimate the potential impact.

The absence of eggs collected from two of the ponds as an implied impact is misleading.

Section 3.4.4 of the document states that fish eggs were not collected from two of the ponds, but implies that this was a potential impact. In contrast, close review of the field collection results provided in Appendix E shows that this was due to excessive macrophyte growth in the ponds precluding the successful use of the sampling gear. Thus the absence of eggs is attributable to sampling methodology issues, and not a potential impact as implied by this section. This was further supported by the ichthyoplankton study that concludes that there are no adverse effects observed from site compounds on the ichthyoplankton (Appendix E).

The comparison of the field collected white sucker egg concentrations to the measured egg concentrations in the channel catfish from the ELS study is inappropriate.

Section 4 of the ELS report attempts to substantiate the EC10 and EC50 egg concentrations calculated from channel catfish based on measured concentrations from field collected white sucker eggs. Review of the Elonen et al. (1998) study, which was used to develop the current study, does not indicate that there would be any effects at the reported concentration range in eggs. The table below compares the No Observed Effect Concentration (NOEC), the Lowest Observed Effect Concentrations (LOEC), the 10% lethal concentration (LC_{10}) and the 50% lethal concentrations (LC_{50}) in channel catfish and white sucker eggs (values from Table 5 in Elonen et al., 1998).

Fish Egg Concentrations ($pg/g_{wet\ weight}$)				
Species	NOEC	LOEC	LC_{10}	LC_{50}
Channel catfish	385	855	429	644
White sucker	848	1,220	1,590	1,890

The measured egg concentration for white sucker reported in this section of the document is 340 $pg/g_{wetweight}$, which is well below the NOEC value reported by Elonen et al. (1998). Since the ELS study values calculated for the channel catfish are comparable to those reported by Elonen et al. (1998), it is logical to assume that the values derived for the white sucker by these authors would be reasonable to apply to the Centredale project. Since the observed concentrations are below the NOEC reported by Elonen et al. (1998), it is appropriate to conclude that there would be no negative effects of the accumulated dioxin TEQs by this indicator species, which is in direct contrast to the conclusion presented in this section. Section 5 (Conclusions and Recommendations) should be revised to reflect this comparison. This would also likely affect the BERA conclusions concerning potential impacts to the pelagic and demersal fish populations.

The pathology frequency could be misleading when the sample size is not considered.

Section 4, page 4-2, discusses pathology frequencies but does not always include the sample size to put the values into context. For example, although a 5% lesion frequency may sound significant, its importance is reduced if the sample size was only 20 – i.e., 1 sample impacted out of 20 samples.

It is unclear why relative percent differences (RPDs) are used in Tables 3.7a and 3.7b to represent the potential reproducibility of the triplicate analyses.

It is unclear why RPDs were selected to reflect the variation of the triplicate analyses in Tables 3.7a and 3.7b. It is also noted that the RPDs calculated for the comparisons of rep2 and rep3 are consistently incorrect. For example, for the nominal TCDD dose of 0.018 ng/mL the reported RPD for rep2:rep3 is 113.7% while the recalculated value is 44.3%. The other paired comparisons for this dose are correctly calculated. As a comparison, the relative standard deviation (RSD) for this triplicate comparison is 29.5%.

A more appropriate metric to assess the reproducibility of more than two replicates is the RSD. The presumably "acceptable" RPD range reported in the two USACE documents (both documents were specific for this project; USACE 2002; 2003) could be re-calculated as RSDs to provide comparisons to this more appropriate metric.

Editorial Comments

Cross-referencing of the ToxCalc backup documentation would greatly improve the document clarity. It appears that this information is simply attached to this appendix without regard to including a suitable index to link the results to the discussion.

In Section 4, page 4-1, third paragraph, an incorrect appendix is referenced. The cited appendix (Appendix H) is actually to the Woods Hole Oceanographic Institute letter from Hahn that discusses the TEF for HCX. Supporting information is not provided in this document.

It would have been preferable for the multiple "sub-appendices" to be called "attachments" or "annexes" for clarity.

12.0 CONCLUSIONS

This review has identified, documented and discussed the technical shortcomings of the analyses that comprise the EPA BERA report. We have focused on those exposure scenarios and risk assessment topics that have the greatest implications for setting target remediation goals and assessing remedial options. Our review does not comment on those elements of EPA's work that do not appear to be influential in evaluating remedies or determining outcomes for the site.

Several significant errors were found in the report. In particular, in the Executive Summary and Conclusions of the BERA, EPA mischaracterizes the findings of the technical sections of the

BERA for demersal and pelagic fish populations. Our review of the technical portions of the BERA shows that EPA *did not* find a significant risk of harm to these receptors from exposure to site-related chemicals. However, EPA erroneously reports the opposite of those findings in the Executive Summary and Conclusions. This erroneous conclusion is then propagated through to the ecological preliminary remediation goal (PRG) document, and EPA unnecessarily computes PRGs for these receptors (EPA, 2005a). This is so significant an error that it by itself warrants that EPA revise the BERA and the PRG document to correct the text and remove these receptors.

The BERA lacks transparency and is inconsistent. Of primary importance is the inconsistent approach in evaluating avian populations, which were restricted to an HQ analysis alone. Other receptors evaluated in the BERA are not restricted to an HQ analysis, but also include field observations and related activities that show potential risks to be *de minimis*. In addition, the BERA fails to adequately incorporate important and relevant site-specific information in the life history summaries. Without this information, it is difficult to verify the conceptual site exposure model used in the BERA. With these and other shortcomings, PRG development is questionable at best.

The development of PRGs requires a much more robust and unbiased risk assessment in which state-of-the-art methods of risk analysis are coupled with proper interpretation and analyses of available data. In the absence of revisions to the BERA and PRG development documents, EPA's analysis lacks the level of sophistication required to serve as the basis for establishing technically defensible remedial cleanup goals for the Centredale Manor Restoration Project Superfund Site.

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COMMENTS ON INTERIM-FINAL PRELIMINARY REMEDIATION GOALS REPORT
PART I - HUMAN HEALTH
CENTREDALE MANOR RESTORATION PROJECT SUPERFUND SITE
NORTH PROVIDENCE, RHODE ISLAND
NOVEMBER 2005

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AMEC Earth and Environmental, Inc.
15 Franklin Street
Portland, ME 04101

1.0 INTRODUCTION

In November 2005, EPA Region I released the Interim Final Preliminary Remediation Goals (PRG) Report Part I - Human Health, Centredale Manor Restoration Project Superfund Site, North Providence, Rhode Island (EPA, 2005a). The PRG report was prepared by MACTEC Engineering and Consulting, Inc., under contract to Battelle Corporation, and was submitted to the Department of the Army, U.S. Army Corps of Engineers, New England Division under Contract No. DACW33-01-D-0004, Delivery Order No. 1. The human health PRG document presents the derivation of risk-based preliminary remediation goals for a number of exposure pathways, including fish ingestion and direct contact with surface water, sediment and bank soil. Media-specific PRGs are separately estimated for fish tissue and sediment from five Exposure Areas (EAs) that consist of reaches and wetlands associated with the Woonasquatucket River: EA1, Allendale Pond; EA2, Lyman Mill Pond; EA3, Manton Pond; EA4, Dyerville Pond and EA5, the Fogarty Center. PRGs are also derived for surface soil at the Fogarty Center, EA5. Greystone Mill Pond and Assapumpset Pond are designated as the upstream background area and reference area, respectively.

At the request of Emhart Industries, Inc., AMEC Earth & Environmental, Inc. prepared these comments on EPA's human health PRG document. In reviewing the EPA report and preparing comments for submittal to the Administrative Record, AMEC focused on those calculations, exposure scenarios and risk assessment topics that have the greatest implications on setting target remediation goals and assessing remedial options. Accordingly, the comments do not address aspects of the report that do not appear to be influential in evaluating remedies or determining outcomes for the site. The PRG report presents EPA's determination of preliminary remediation goals for Woonasquatucket River sediment and flood plain soils. The primary exposure pathways are ingestion of sport-caught fish, incidental ingestion of soil and dermal contact with soil. Because the fish ingestion pathway is most influential in driving the remedial discussions for this site, most of the comments provided on this document are focused on the development of PRGs related to this exposure pathway. However, we also provide specific comments regarding the incidental soil ingestion exposure pathway, which is the exposure pathway that poses the greatest theoretical risk second to the fish ingestion pathway.

Section 2.0 of this document presents comments regarding the general shortcomings of EPA's human health PRG document. Section 3.0 provides detailed comments on the technical approach and procedures for developing PRGs. Section 4.0 provides detailed comments on the calculations of the PRGs. Section 5.0 provides comments on the uncertainty analysis presented in the EPA document. Finally, Section 6.0 presents the conclusions of our review of the human health PRG document.

2.0 GENERAL REPORT COMMENTS

The Interim Final Preliminary Remediation Goals Report Part I - Human Health Human Report contains numerous internal inconsistencies which render the document very difficult if not impossible to follow. Moreover, the document cannot be used as a stand-alone document since it does not fully describe the data, methods, and results of the human health-based PRG development. Because of these deficiencies, the document lacks the transparency that is required of EPA documents, as specified by the Office of Management and Budget's (OMB) *Information Quality Guidelines* (OMB, 2002) and EPA's *Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility and Integrity of Information Disseminated by the Environmental Protection Agency* (EPA, 2002). The lack of transparency calls into question the veracity of the report's results and conclusions.

The report's internal inconsistencies include the following:

Step 1 - Section 2.1 details the specific steps that are to be used to develop the PRGs. However, these steps are not followed in Section 3. For example, Steps 3 and 5 in Section 2.1 discuss the use of the *mean* lipid-normalized tissue concentrations and the organic carbon normalized sediment concentrations, respectively, for developing biota-sediment accumulation factors (BSAFs). In contrast, in Section 3.3.3 and subsequent sections, the report discusses the use of the *geometric* mean lipid-normalized tissue concentrations and the organic carbon normalized sediment concentrations for determining BSAFs.

In addition, Section 2.1 does not detail and fails even to mention the methods that are used to calculate the interim step of developing the fish tissue PRGs. However, in Section 3.3.4.1, the reader is erroneously referred to Step 2 of the PRG development

process for the description of how fish tissue PRGs are computed. As mentioned above, no discussion of fish tissue PRG development is provided in any of the steps presented.

Another inconsistency in the report relates to the use or lack thereof of an outlier analysis. Section 3.2 of the PRG report discusses the use of the outlier analysis that was described in MACTEC (2005). The report then recommends the use of an outlier analysis and presents the results of such an analysis as Appendix A. However, it is entirely unclear whether the outlier analysis was ever substantively employed for limiting the data used in the PRG evaluation. The PRG document takes an unexplained turn away from the use of the outlier analysis as well as from the use of mean normalized sediment and tissue metrics. Instead, it turns towards the use of all data, including outliers and geometric means of normalized sediment and tissue data. There is no discussion as to the pros and cons of either method, the effect of using one method or the other, or any ranking of the technical correctness of one method or the other.

These shortcomings (and the others detailed below) create a document that is at best internally inconsistent and virtually impossible to follow in a manner that allows a reader, such as a risk manager or a member or the public, to understand the most important factors that affect PRG development. Technical considerations notwithstanding, the quality of this document is very poor. It should not have been released as an Interim Final public document in its current state without substantial modifications to make it more transparent to the reader.

3.0 TECHNICAL APPROACH AND PROCEDURE FOR DEVELOPMENT OF PRGS

Step 1 of Section 2.1 is not consistent with the BHHRA or the PRG document.
MACTEC states that, in addition to the combined fish diet analysis, a PRG will be developed for "the species with the highest calculated risks (Largemouth bass)." This statement is inconsistent with the Baseline Human Health Risk Assessment (BHHRA) and the rest of the PRG report. The BHHRA does not compute risks from the ingestion of a single fish species. For all exposure areas except Dyerville and Manton Ponds, which should not have been included in the PRG document due to the paucity of data for those areas, none of the other four areas have less than two species. The BHHRA combines the species data to yield an exposure point concentration (EPC). A species-specific assessment is not conducted for these four areas. In addition, had such an

assessment been conducted, Largemouth bass would not have been the species with the highest calculated risk in any case. In all cases, the fillet data from Largemouth bass would not have resulted in the highest computed risk. Where comparable data exist (i.e., within the same exposure area), whole body sucker and eel data are higher in dioxin than the Largemouth bass. Therefore, the Largemouth bass data would not be the species that exhibits the largest risk as incorrectly stated in the PRG document.

In addition, the stated approach (computing cancer and noncancer PRGs) for the species with the highest computed risks is not conducted anywhere in the PRG document. It appears that this step was never completed, nor should it be completed. However, the fact that step 1 of the PRG development process states that it will be completed is misleading and false. Like the other items discussed above in the General Comments, this type of an error in such a fundamentally important part of the PRG document seriously calls into question the report's veracity and authority.

No mention of a data outlier analysis is made in the list of tasks presented in Section 2.1.

As stated in the General Comments, EPA does not even mention the use of an outlier analysis in the steps presented for developing PRGs for this site. In fact, no data evaluation is presented in the steps that are to be followed. However, Section 3.1 is devoted to an analysis of data outliers. The presentation of the outlier analysis is not consistent with the step-by-step procedures detailed in Section 2.1. If EPA is going to apply an outlier analysis (see additional comments on this matter below), then this step should be added to the PRG development process.

EPA does not follow Step 3 in their own document.

Step 3 states that mean lipid-normalized fish tissue data are a component of the development of the fish consumption sediment PRGs. However, in Section 3.3.4.1, EPA states that *geometric* mean lipid-normalized concentrations are used. The switch to the use of a geometric mean from an arithmetic mean is not discussed at all. This is confusing and internally inconsistent.

EPA does not follow Step 5 in their own document.

Step 5 states that mean organic carbon normalized sediment data are a component of the development of the fish consumption sediment PRGs. However, in Section 3.3.4.1,

EPA states that *geometric* mean organic carbon normalized sediment data concentrations are used. This switch to the use of a geometric mean from an arithmetic mean is not discussed at all. This is confusing and internally inconsistent.

EPA should compute PRGs for both Central Tendency (CTE) and Reasonable Maximum Exposure (RME) scenarios.

EPA computes PRGs using exposure parameters for the RME scenarios. However, to give the risk manager more information for assessing cleanup values, EPA should also include an assessment of PRGs using a CTE scenario. This would be in keeping with EPA's stated *Risk Assessment Principles and Practices* (EPA, 2004).

> "In addition to evaluating the risks to the RME individual, EPA evaluates risks for the central tendency exposure (CTE) estimate, or average exposed individual. This approach is consistent with the Risk Characterization Policy and Handbook (USEPA, 1995a, 2000a). CTE estimates give the risk manager additional information to consider while making decisions at a site." (EPA, 2004, Sect. 5.1.2)

That EPA fails to calculate PRGs using a CTE evaluation is particularly troublesome in light of the fact that the exposure parameters used for the RME evaluation represent the extreme upper end of the range of all conceivable exposure factors. The RME analysis thus represents an unrealistic portrait of potential exposure, as documented in AMEC's *Comments on the Interim-Final Baseline Human Health Risk Assessment – Centredale Manor Restoration Project Superfund Site*, dated October 19, 2006.

EPA incorrectly states that the BSAF is unitless.

As computed in Section 2.1, the BSAF has units of kg organic carbon/kg lipid in fish tissue. EPA should change this in all equations and definitions.

EPA incorrectly switches between central tendency values for lipid-normalized tissue concentrations and organic carbon content-normalized sediment concentrations.

In Step 7, EPA shows the equation for estimating the PRG_{sed}. The equation calls for dividing the target fish concentration by the BSAF and multiplying by the mean total organic carbon and then dividing by the mean fish lipid content. The BSAF is computed using the geometric mean of the organic-carbon normalized sediment data and the lipid content normalized fish tissue data. The central tendency value (arithmetic or geometric

mean) used in the BSAF and the PRG_{sed} calculations should be the same. EPA should make these consistent throughout the document.

4.0 CALCULATION AND PRESENTATION OF PRGS

EPA introduces an outlier analysis that ultimately has no bearing on the calculations it performs.

In Section 3.1, EPA discusses a data outlier analysis which was supposed to identify data that is not consistent with other data of the same metric. In Appendix A and Table 3-1, EPA summarizes those samples which are considered outliers. Section 3.1 states that the outliers are to be removed from the data set because they "might otherwise have an inordinate and unexplained impact on the estimated BSAF for that COC at a particular exposure area." However, it does not appear that the outlier analysis was used substantively in any subsequent BSAF analysis.

EPA does not adequately define Appendix A charts.

In Section 3.1, EPA states that Appendix A contains normalized chemical data in biota and sediment. However, Appendix A contains charts with units simply stated as mg/kg. There is no indication from the chart title whether the data are normalized or not. In addition, Appendix A contains charts featuring metals data, which should not be evaluated using lipid- or organic carbon-normalized data, as these chemicals are not necessarily preferentially sorbed to those compartments.

EPA does not adequately describe the outlier analysis.

EPA states that an outlier analysis was performed to identify those situations where a chemical in a given media is five times higher or lower than other samples in that media. However, there is no citation or explanation to support the validity of such a "five times greater or lower" rule. It would appear that this type of an outlier analysis would only be valid if the distribution of the data is known. Without knowing the underlying distribution of the data, EPA could be truncating a data set (i.e., removing an outlier) based on the idea that the data set is normal when in fact it is not normally distributed. Without knowing the distribution of the data in advance, an outlier analysis centered on the idea of a normal distribution is not technically correct and should not be conducted.

In addition, the underlying assumption that EPA makes when conducting the outlier analysis is that the chemical data are somehow correlated to the organic carbon content

or tissue lipid analysis. However, for 2,3,7,8-TCDD, EPA found that there was no correlation between 2,3,7,8-TCDD and organic carbon in sediment (EPA, 2005).

Without justification, EPA changes the focus of the outlier analysis to just white sucker and largemouth bass.

In Section 3.2, EPA states that, "The calculation of BSAFs for sediment PRGs for white sucker and largemouth bass should include an outlier analysis for chemical concentration data in sediment and fish tissue." This statement is inconsistent with the outlier analysis presented in Appendix A, which illustrates the application of an outlier analysis to white sucker, largemouth bass, and American eel. EPA provides no rationale for limiting the species to which it is applied.

EPA does not evaluate the small subset of sediment data with corresponding TOC data in light of the larger data set to determine the representativeness of the smaller data set.

In Section 3.3.1, EPA presents the data set that is used for developing PRGs for sediment. The data set includes only those sediment samples that have corresponding TOC data. However, EPA never presents a comparison of the small data set with the larger dataset which contains samples with and without TOC data. EPA should compare and contrast these datasets. As it stands, there is no way to know whether the sediment data set used for the PRG analysis is consistent with the overall dataset.

EPA should not be calculating PRGs for Manton and Dyerville Ponds.

EPA computes sediment PRGs for all the ponds assessed in the RI. However, the PRGs that are presented for Manton and Dyerville Ponds are highly suspect and extremely uncertain. This results from the absolute paucity of fish tissue and sediment data that have been collected from these two ponds. As shown in Tables 3-2 and 3-3, EPA computes sediment PRGs for Manton and Dyerville Ponds based on three sediment samples and three fish tissue samples from each pond. Moreover, for Dyerville Pond, the fish tissue data are entirely from American eel, a fish that is not highly desirable. EPA should redact the sediment PRGs for these ponds given the lack of data upon which they are based.

EPA does not follow its own procedures for computing the BSAFs.

In section 3.3.3, EPA states that "BSAFs have been calculated in a manner consistent with the equation presented in Step 6 of Section 2.1." However, the discussions prior to Step 6 include the use of the arithmetic mean concentration. In Section 3.3.3, EPA uses the geometric mean concentrations rather than the arithmetic mean concentrations. The correct metric will depend on the distribution of the data, which has not been reported in the PRG document. EPA should determine the distribution of the data, and, based on those results, consistently choose the correct central tendency value for the BSAF calculation.

EPA gives undue weight to the ponds with little data when deriving the Lyman Mill BSAF.

EPA concludes that the data from Lyman Mill Pond is inadequate for computing a pond-specific BSAF. As a surrogate, EPA computes a BSAF from Lyman Mill Pond as the arithmetic average of the BSAFs computed for the other areas. EPA's method for calculating the BSAF for Lyman Mill Pond gives undue weight to those ponds (i.e., Manton and Dyerville) with little data. For example, the BSAF for American eel for Allendale Pond is computed with 10 fish tissue samples and 39 sediment samples. For this same species, the BSAF for Dyerville Pond is computed using only three eel samples and three sediment samples. Based on data set robustness alone, there should be more weight given to the BSAF computed for Allendale Pond when computing the BSAF for Lyman Mill Pond. However, due to the method that EPA is employing, the same weight is given to the data from Allendale Pond as that given the data from Dyerville Pond. In contrast, to develop a BSAF for Lyman Mill Pond, EPA should weight the pond-specific BSAFs by the limiting data factor, which is generally the number of tissue samples.

EPA inserts the fish tissue PRG without explanation.

Section 3.3.4.1, which describes the development of fish tissue PRGs, states that Step 2 describes the development of fish tissue PRGs. However, Step 2 (from Section 2.1 – the only place where Step 2 is enumerated) discusses identifying the lipid-normalized fish tissue concentrations. EPA does not discuss fish tissue PRG development anywhere in Section 2.0. This text is confusing and needs to be changed. In addition, EPA should add a discussion regarding fish tissue PRG development in Section 2.0.

The exposure parameters upon which the fish tissue PRGs are based are seriously flawed.

AMEC Earth & Environmental has prepared and submitted comments on the BHHRA and specifically on the shortcomings of the exposure parameters used in the BHHRA. These same comments apply to the derivation of the fish tissue PRGs.

5.0 UNCERTAINTIES

EPA should include a discussion of the exposure assessment inputs.

The PRGs developed for fish, sediment, and soils are highly dependent on the exposure parameters used in the PRG development process. As pointed out by the risk assessment results summarized in Table 1-2 of the PRG report, different exposure parameters (RME vs. CTE) can result in PRGs that differ by an order of magnitude. PRG estimates are likely to differ by at least two orders of magnitude if the exposure parameters recommended by AMEC were employed in this assessment. At a minimum, EPA should present an analysis of PRGs using the CTE exposure parameters to provide risk managers and the public with a sense of the uncertainty that EPA has introduced with the use of extreme and unrealistic RME exposure parameters.

EPA fails to discuss the full range of plausible dioxin toxicology.

EPA's discussion of dioxin toxicology fails to fully discuss the full range of potential dioxin cancer potency factors. EPA should include further discussion on the toxicology of dioxin, specifically identifying peer-reviewed scientific studies spanning the full range of plausible cancer slope factors for 2,3,7,8-TCDD. Moreover, the BHHRA should state that EPA has never published a cancer slope factor (CSF) for TCDD in IRIS and that the carcinogenic potential of TCDD is a matter of much debate in the dioxin reassessment process that has been underway at EPA for 15 years.

A wide range of CSFs based on animal studies, ranging from 9,000 to 156,000 $(mg/kg\text{-}day)^{-1}$, have been proposed by various groups under the linear, non-threshold cancer model, with differences resulting from the selection of tumor classification scheme and interspecies scaling factor used (EPA, 1994, 2000; FDA, 1993, 1994; Keenan et al., 1991). Recently, in two subsequent revisions of the draft Dioxin Reassessment, EPA proposed the use of a CSF of 1,000,000 $(mg/kg\text{-}day)^{-1}$ based on its evaluation of human epidemiological data (EPA, 2000; 2003). However, there is substantial disagreement

concerning the appropriate CSF for TCDD. In fact, in its review of EPA's draft final Dioxin Reassessment, the Agency's Science Advisory Board discussed the uncertainties associated with the available dose-response data and the extrapolation methods used, and stated that they could not "reach consensus on a single value for a dioxin potency factor" (EPA, 2001; p. 6). Subsequently, Congress directed EPA to undertake an agreement with the National Academy of Sciences (NAS) to review the draft Dioxin Reassessment (House of Representatives, 2003). Among other key issues, Congress asked NAS to evaluate "the validity of the non-threshold linear dose-response model in light of epidemiological studies and the corresponding cancer slope factor calculated by the Agency through use of this model" and "the appropriateness of including 'dioxin-like' chemicals in the risk assessment without independent empirical review of their effects" (House of Representatives, 2003, pp. 1445-46). Thus, there is substantial uncertainty associated with any selected CSF and, depending upon the CSF selected, the resulting regulatory standards and remedial decisions can vary by orders of magnitude.

NAS recently completed its review of the draft Dioxin Reassessment (NAS, 2006). Among its conclusions, the NAS committee determined that the available data support a nonlinear relationship rather than defaulting to a non-threshold linear model as EPA has in the past. NAS concluded,

> "The committee unanimously agrees that the current weight of evidence on TCDD, other dioxins, and DLCs [dioxin-like compounds] carcinogenicity favors the use of nonlinear methods for extrapolation below the point of departure (POD) of mathematically modeled human or animal data." [p. 135]

NAS further commented that,

> "a risk assessment can be conducted without resorting to default assumptions. To the extent that EPA favors using default assumptions for regulating dioxin as though it were a linear carcinogen, such a conclusion should be supported with scientific evidence." [p. 100]

Summarizing this issue, NAS stated that,

> "The selection of the default linear extrapolation approach was one of the most critical decisions in the Reassessment, but the decision to use this approach was not supported by a scientifically rigorous argument, nor was there a balanced presentation of arguments that would support the calculation and interpretation of a MOE [margin of exposure] with the same data. The committee determined that a balanced presentation of

> available data could support the use of a threshold model with subsequent calculations and interpretation of MOE. (For cancer risk assessment, the threshold approach should be used in addition to the linear approach.)" [p. 140]

While NAS (2006) commended EPA on its use of the benchmark dose (BMD) method as a replacement of the traditional approach of using the no-observed-adverse-effect level (NOAEL) and lowest-observed-adverse-effect level (LOAEL), NAS criticized EPA for using an effective dose (ED) at a 1% response level (ED01) for characterizing cancer and noncancer risks. NAS stated,

> "In its evaluation of ED01 used for cancer risk assessment, the committee concluded that EPA had not adequately justified use of the 1% response level as the POD for the analysis of either the epidemiological or the animal bioassay data....
>
> For the various noncancer end points, EPA should describe more clearly how and why the ED01 values were determined in animals and transferred to human equivalents. At the least, the risk assessment should provide more apparent and parallel calculations using a 5% response level as the POD to demonstrate the impact that this assumption might have on both the point estimates of risk at low doses and the range of uncertainty surrounding that point estimate. This recommendation applies to extrapolation for cancer risk estimates, as well as for noncancer risk estimates." [p. 135-136]

The NAS committee recommended that EPA consider the full range of animal bioassay data, "including the new NTP animal bioassay studies on TCDD, other dioxins, and DLCs for quantitative dose-response assessment." [p. 136]

While NAS agreed that use of body burden as the dose metric is a reasonable approach, the committee raised a number of uncertainties associated with using body burden to develop risk estimates. NAS (2006) concluded,

> "The magnitudes of the various uncertainties are not clearly defined. The most significant impact is the species differences in percentage body fat on the relationship between body burden and the concentrations present in nonadipose tissues. An analysis of the impact of possible uncertainties in the dose metric on the final risk estimates would be informative.
>
> It remains to be determined whether the current WHO TEFs, which were developed to assess the relative toxic potency of a mixture to which an organism is directly exposed by dietary intake, are appropriate for body burden toxic equivalent quotient (TEQ) determinations, which are derived from the concentrations of different congeners measured in BF. If body burdens are to be used as the dose metric, a separate set of body burden

> TEFs should be developed and applied for this evaluation. Without these corrected values, the overall TEQs estimated by use of intake TEFs might be substantially in error." [p. 138]

Furthermore, the high level of uncertainty associated with the application of the CSF for TCDD to dioxin-like PCBs is unnecessary, due to the fact that EPA has already established a CSF for PCBs that is based on empirical bioassay data (EPA, 1996). Depending upon how this is done, the approach may substantially overestimate the carcinogenic potential of PCBs as it may double-count the carcinogenic potential of the entire mixture. This is because the approach that is often used evaluates the "dioxin-like" PCBs using the CSF for TCDD but then evaluates the carcinogenic potential of the remaining "non-dioxin-like" PCBs using the CSF that EPA has established for PCBs. However, the CSF that has been derived for PCBs includes the carcinogenic activity of the entire PCB mixture tested, including both the "dioxin-like" and the "non-dioxin-like" congeners. Thus, the carcinogenic potential of the "dioxin-like" congeners is counted twice and added together when this approach is used.

EPA failed to compare the PRGs with known food-chain exposures of dioxin.

EPA computes biota-based human health PRGs for COCs without consideration of other dietary sources of dioxin, which are much more readily available to the receptor groups evaluated, and, in all likelihood, represent much larger potential sources of dioxin than fish from the Woonesquatucket River. For example, the biota-based human health PRG for dioxin TEQ in fish, computed at a target risk level of 1 in 1,000,000, is 0.072 pg/g wet weight. This PRG target concentration is approximately 25 times greater than the average concentration of dioxin TEQ found in farmed salmon fillet samples that are sold in U.S. supermarkets (Hites et al., 2004). EPA should present the PRGs in light of other possible sources of dioxin.

Use of a Non-Cancer Hazard Index Benchmark of 1.0.

While EPA has used a non-cancer Hazard Index (HI) benchmark of 1.0 as the limit for acceptable non-cancer exposures, a HI greater than 1.0 does not necessarily constitute a matter of concern or indicate that an adverse health effect will occur. It only indicates that a conservative threshold has been exceeded. The HI is the ratio of the predicted dose to the RfD. The RfD represents a daily intake level (or dose) that will not result in non-cancer health effects. That level is typically calculated by applying multiple uncertainty factors to the no-effect or lowest-effect level in the underlying study. Thus, if

the HI is less than 1.0, then the dose is less than the RfD and no risk is predicted. However, given the uncertainty factors and conservatism inherent in the derivation of the RfD, the converse is not true: a calculated HI greater than 1.0 does not necessarily mean that significant risks are predicted.

The RfD is itself defined by the EPA as "an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime" (EPA, 1988). With uncertainty spanning an order of magnitude or greater built into the very definition of the RfD, a calculated HI greater than 1.0 cannot and should not be interpreted automatically as warranting action – only as warranting further attention. The BHHRA should make that point explicitly. Correspondingly, in discussing the results of the various risk assessments, the BHHRA should not consider HIs greater than 1.0 as necessarily indicative of unacceptable non-cancer hazards.

6.0 CONCLUSION

This review has identified, documented and discussed the technical shortcomings of the analyses that comprise the EPA Human Health PRG report. In the interest of offering constructive criticism that focuses on those exposure scenarios and risk assessment topics that have the greatest implications for setting target remediation goals and assessing remedial options, we have refrained from compiling an exhaustive litany of shortcomings. Instead, we have discussed the substantial shortcomings and biases inherent in the methods and data EPA has used in developing the PRGs for the Centredale Manor site. Because the fish ingestion pathway has the most significant impact on the establishment of PRGs and this is the exposure pathway which is most significantly flawed, we have focused our efforts on reviewing PRG calculations related to this exposure pathway. We also have commented on the deficiencies inherent in EPA's assessment of potential risks via incidental soil and sediment ingestion, which is the exposure pathway that poses the next greatest theoretical risk after the fish ingestion exposure pathway.

A large number of technical issues arise because EPA has used overly conservative and unrealistic exposure assumptions in the BHRRA (EPA, 2005b). The deficiencies in the BHHRA propagate through to the PRG development document, resulting in seriously

flawed PRG values for fish tissue and sediment. The development of PRGs requires a much more robust and unbiased risk assessment in which state-of-the-art methods of risk analysis are coupled with competent interpretation and analyses of available data. The development of PRGs does not meet this standard in its present form, and anything short of a complete overhaul and revision of the BHHRA and its sister PRG development document is unlikely to approach the level of sophistication required to serve as the basis for establishing technically defensible remedial cleanup goals for the Centredale Manor Restoration Project Superfund Site.

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COMMENTS ON INTERIM FINAL PRELIMINARY REMEDIATION GOALS REPORT
PART II – ECOLOGICAL
CENTREDALE MANOR RESTORATION PROJECT SUPERFUND SITE
NORTH PROVIDENCE, RHODE ISLAND
NOVEMBER 2005

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AMEC Earth and Environmental, Inc.
15 Franklin Street
Portland, ME 04101

1.0 INTRODUCTION

In November 2005, EPA Region I released the Interim Final Preliminary Remediation Goals (PRG) Report Part II – Ecological for the Centredale Manor Restoration Project Superfund Site, North Providence, Rhode Island (EPA, 2005). The PRG report was prepared by MACTEC Engineering and Consulting, Inc., under contract to Battelle Corporation, and was submitted to the Department of the Army, U.S. Army Corps of Engineers, New England Division under Contract No. DACW33-01-D-0004, Delivery Order No. 1. The Ecological PRG document presents the derivation of risk-based preliminary remediation goals for a number of sensitive receptor communities and media. Receptor communities include lentic aquatic invertebrates, demersal (white sucker) and pelagic (largemouth bass) fish, and piscivorous (blue heron, belted king fisher and river otter), insectivorous (tree swallow and little brown bat), and vermivorous (american woodcock and short-tailed shrew) wildlife. Media for which PRGs are developed include soil and sediment. Receptor- and media-specific PRGs are separately estimated for soil and sediment from five Exposure Areas (EAs) that consist of reaches and wetlands associated with the Woonasquatucket River: EA1, Allendale Pond; EA2, Lyman Mill Pond; EA3, Manton Pond; EA4, Dyerville Pond and EA5, the Fogarty Center. Greystone Mill Pond and Assapumpset Pond are designated as the upstream background area and the reference area, respectively.

At the request of Emhart Industries, Inc., AMEC Earth & Environmental, Inc. prepared these comments on EPA's Ecological PRG document. The comments focus on those calculations, exposure scenarios and risk assessment topics that have the greatest implications on setting target remediation goals and assessing remedial options. Accordingly, the comments do not address aspects of the report that do not appear to be influential in evaluating remedies or determining outcomes for the site.

Several significant errors were noted in our review of the report. In particular, AMEC has determined that EPA mischaracterizes the findings of the BERA for demersal and pelagic fish populations. Our review of the BERA shows that EPA *does not* find a significant risk of harm to these receptors from exposure to site-related chemicals. However, EPA erroneously reports the opposite of those findings in the PRG document and unnecessarily computes PRGs for these receptors where none should have been computed. This is a significant error which by itself warrants that EPA revise the PRG document to remove these receptors. This substantial

shortcoming is examined below, along with other technical comments on EPA's Ecological PRG development.

Section 2.0 of this document presents general comments regarding the report. Section 3.0 provides detailed comments on the technical approach and procedures used by EPA for developing PRGs. Section 4.0 provides detailed comments on the PRG calculations. Section 5.0 discusses the uncertainty analysis as presented by EPA. Section 6.0 presents comments on the comparison of PRGs to media concentrations. Finally, Section 7.0 presents the conclusions of our review of the Ecological PRG document.

2.0 GENERAL REPORT COMMENTS

EPA incorrectly summarizes the findings of the BERA and computes PRGs for unwarranted receptors.

EPA states in Section 1.1 that both demersal and pelagic fish populations may be at substantial risk of harm from site-related chemicals of concern. In stark contrast, however, EPA's BERA concludes the exact opposite. Specifically, Section 5.4 of the BERA states the following:

> "Integrating these results, it is concluded that the demersal fish community associated with the study area ***is not*** [emphasis added] at substantial risk of harm due to the presence of contaminant release from the Centredale Manor source area" (EPA, 2004).

Similarly, Section 6.4 of the BERA concludes the following:

> "In summary, the pelagic fish populations associated with the study area are not at substantial risk of harm due to the presence of contaminant release from the Centredale Manor source area."

Presumably, the error in the PRG document stems from the fact that the BERA conclusions summary section is misaligned and internally inconsistent with the findings reported in the specific sections of the report. Nevertheless, given the level of technical review that these documents reportedly undergo prior to public release, EPA and its contractors should not have allowed this error to be made or to propagate through subsequent documents.

EPA's misrepresentation of their data, analysis and conclusions for these receptors is a gross error for both the BERA and the PRG document. These errors by themselves are enough to warrant that EPA revise both the BERA and the PRG document.

EPA's conclusion that there was an impact to the aquatic invertebrate lentic communities did not include the results from the benthic community assessment that showed no impact.

In Section 1.1 of the subject report, EPA states that the aquatic invertebrate community in lentic habitats appears to be at substantial risk to harm. This is consistent with the conclusion from the BERA that reported an impact to the aquatic invertebrate lentic communities, which was based on a hazard quotient analysis. However, EPA disregarded the results from the biological assessments of the aquatic communities which showed no apparent impact to the aquatic invertebrate lentic community. In fact, the conclusion that the aquatic invertebrate community in lentic waters is at substantial risk of harm flies in the face of the results of the benthic community assessment, which by all accounts showed no significant biological impact. This comment is discussed in greater detail in the comments to the BERA document.

3.0 TECHNICAL APPROACH AND PROCEDURE FOR DEVELOPMENT OF PRGS

EPA has failed to discuss how the PRGs derived from several measures of potential toxicity should be evaluated from a risk management perspective.

In the PRG document, EPA computes PRGs based on a variety of ecological benchmarks including the No–Observed-Adverse-Effect Level (NOAEL), Lowest-Observed-Adverse-Effect Level (LOAEL) and Maximum Allowable Threshold Concentration (MATC). The use of a variety of toxicity thresholds results in multiple PRGs for the same endpoint and media. Sometimes those PRGs differ significantly (by an order of magnitude or greater). However, EPA does not provide any discussion concerning how these different PRGs should be viewed from a risk management perspective. Without such a discussion, the lowest PRG value may be deemed appropriate even though the community assessments conducted for the BERA did not indicate any significant impacts to the receptors evaluated. In addition, the fact that non-site related chemicals, such as chlordane and mercury, are prevalent throughout the watershed at concentrations that could by themselves pose a theoretical risk to ecological receptors argues against the implementation of the lowest computed PRG levels.

EPA methodology used to derive PRGs for fishes was overly conservative and inappropriately biased low.

The benchmarks used to derive the demersal and pelagic fish PRGs were the Critical Body Residue (CBR) values used in the BERA. The same CBR values used to derive PRGs were also used in the BERA. However, in most cases, the BERA inappropriately used only the lowest, most conservative reported CBR value. Other CBR values were available in many cases, reflecting the range and distribution of values that could be applied. Almost certainly, some of these values have the potential of being more relevant to the fish species present in the ponds at the site. In addition, the application of a less conservative CBR value would be more consistent with the results of the fish community assessments, which did not observe the occurrence of significant effects to either the pelagic or demersal fish populations. Since conservative CBRs were used, overly protective PRGs were calculated. At a minimum, EPA should evaluate a range of CBRs for the PRG calculation to develop bounding estimates for the PRGs. Such an approach would reduce the potential for unnecessary remediation, and also better define and target key areas where remediation may be advisable.

EPA did not consider the bioavailability of 2,3,7,8-TCDD in surface water when designing the Early Life Stage (ELS) Study.

Filtered water represents the bioavailable fraction for waterborne exposures. The exposure concentrations used in the ELS study (2 to18 ng/L) are similar to the surface water grab sample data from unfiltered surface water samples collected during the remedial investigation. However, the unfiltered samples contain suspended particles onto which the 2,3,7,8-TCDD is likely to be sorbed. The data from the unfiltered samples do not accurately represent the plausible dissolved phase concentrations of 2,3,7,8-TCDD because most, if not all, of the 2,3,7,8-TCDD is likely bound to particles in the water samples. EPA recognized this fact in the Revised Interim Final Baseline Human Health Risk Assessment, which explained that "the dioxin/furans that have been measured in surface water may be associated with suspended particulate matter, perhaps derived from sediments. This is likely because 2,3,7,8-TCDD has such a low solubility in water and it has a high affinity for sediments that contain organic carbon as well as for high lipid materials such as fish." Additionally, the Remedial Investigation Report states that the 2,3,7,8-TCDD in surface water is likely associated with suspended solids and is not likely to become dissolved in the water.

The concentrations of dissolved (i.e., bioavailable) 2,3,7,8-TCDD in site surface water is likely to be much lower than the range of concentrations used in the ELS study. This is supported by

the comparison of the eggs, larvae and fry pathological abnormalities between the ELS study and the field collected fish (as discussed in Appendix H of the BERA). Although the latter were slightly older than the fish from the ELS study by several weeks, they did not exhibit as many anomalies as the test organisms from the ELS study. Presumably, the differences result primarily due to the fact that the *in-situ* environment (TSS) renders some portion of the 2,3,7,8-TCDD non-bioavailable. Nonetheless, the EC values generated from the ELS study were used as the benchmark for the HQ analysis.

EPA's assumption for maternal to egg transfer used to derive the PRG for dioxin (0.7) in fish is inadequately supported.

The PRG derived for the sensitive endpoint of eggs in fish assumed a maternal-to-egg transfer factor of 0.7. The limited discussion in the PRG document indicates that this number was derived from the literature, but specific references were not provided in the PRG documents or in the BERA.

A limited literature review supports the use of a value lower than 0.7. For example, in their oral ingestion study of 2,3,7,8-TCDD using adult brook trout, Tietge et al. (1998) reported that the concentration in the spawned eggs was 39% (0.39) of the whole body 2,3,7,8-TCDD concentrations. This value represented the slope of the regression between whole body concentrations, which ranged from approximately 100 to 1,900 pg/g fish, and spawned egg concentrations, which ranged from about 50 to 700 pg/g egg. The regression coefficient of determination was 93%. The authors did not report confidence bounds on the slope.

Heiden et al. (2005) reported that the concentration in eggs was 3% (0.03) of the carcass 2,3,7,8-TCDD concentrations. Adult female zebrafish were exposed to 1,2,7,8-TCDD in their diets using four treatment groups (10, 40, 100 and 270 ng/g diet) with tissue concentrations monitored after 5, 10, 15 and 20 days. As with the study by Tietge et al. (1998), this value represented the slope of the regression between carcass concentrations, which ranged from approximately 1 to 35 ng/g fish, and egg concentrations, which ranged from about 0.1 to 1.3 ng/g egg. The regression coefficient of determination was 87%, and the authors did not report confidence bounds on the slope.

Also noteworthy is the fact that EPA's site-specific maternal tissue-to-egg transfer factor was 0.35 (EPA, 2004). Thus, this site-specific factor, which was the originally proposed value (0.35)

in the BERA Work Plan, was essentially doubled and replaced by the value of 0.7. We believe that the 0.35 value is much more supportable by the references presented above.

As stated earlier in these comments, PRG development for demersal and pelagic fish are not warranted based on the results of the BERA. Notwithstanding the commission of that substantial error, EPA should have used the transfer factor of 0.35 based on the site-specific data, which is supported by Tietge et al. (1998).

EPA does not provide support for normalizing the geometric mean values for sediments or biota by the geometric mean values for total organic carbon content or lipids, respectively, to derive the biota-sediment accumulation factors (BSAFs).

EPA's approach for deriving the BSAFs was to normalize the geometric mean biota or sediment results using the geometric mean lipid content or total organic carbon (TOC) concentrations, respectively. However, EPA does not provide any justification for this approach. Moreover, it is unclear why this approach was taken since lipid and TOC data were available for many of the individual samples. It is more appropriate to first normalize the biota or sediment results to their respective lipid and TOC data, and then calculate BSAFs. In those cases where lipid or TOC data were lacking, a substitution method using the average lipid or TOC results could have been used, or the data could have been censored from the BSAF derivation.

In addition, the presumption that the geometric mean of the TOC or lipid data would be representative of the central tendency value for these parameters was not presented in this document. EPA should conduct an assessment of the data to determine the metric most appropriate for describing the central tendency of the data distribution.

4.0 CALCULATION AND PRESENTATION OF PRGS

EPA's statement that relevant published BSAF data for emergent/aquatic insects and earthworms are not available is not supported.

The focus of Section 3.2.1 of the PRG document was on BSAF data derived for fish, but there are a number of publications in the peer-reviewed literature that derive BSAF data for emergent/aquatic insects and earthworms.

There are several studies which examine dioxin uptake by earthworms from sludge or biosolids disposal (e.g., Matscheko et al., 2002; Meyn et al., 1997) or from soils (e.g., Jager et al., 1998; Sample et al., 1998). These studies also provide information for non-dioxin organic chemicals.

In addition, EPA should have taken the initiative to derive BSAFs for similar species or from studies where BSAFs are not derived but where the data are available to do so (e.g., Maul et al., 2006). EPA should consider the use of this alternative approach for developing BSAFs for emergent/aquatic insects and earthworms.

5.0 DISCUSSION OF UNCERTAINTIES

EPA's uncertainty discussion is inadequate and provides scant information useful to a risk manager or the public.

EPA's uncertainty discussion is qualitative and provides no information of the relative importance of the different uncertainty components. The uncertainty discussion was highly qualitative and only a minimal effort was made to estimate the potential biases in the input parameters and their impact on the calculated PRGs. For example, as noted in an earlier comment often the lowest and most conservative CBR values were used in the BERA. Other higher CBR values were available in many cases that may be more relevant to the fish species that were present in the ponds at the site. The potential bias from using the more conservative CBR values to derive the PRGs should be presented. The use of a more quantitative assessment can provide bounding estimates for the PRGs, as well as assist in the implementation of the PRGs during the interpretation of the compliance data during the remedial action. Therefore, EPA should quantify the uncertainty in the PRG estimates.

6.0 COMPARISON OF PRGS TO BACKGROUND CONDITIONS AND SITE CONCENTRATIONS

Minor reformatting of the summary tables would be beneficial.

It is appreciated that there was a discussion of the comparison of the PRGs to background conditions. To assist in reviewing the information presented in the tables, we would recommend that the background concentrations be repeated on each of the pages of the tables for clarity, since the current formatting (bold font) does not show adequately on photocopied pages.

Although not explicitly stated, the implication of this section is that exceedances of the PRGS at individual sample locations would require remediation.

Both subsections of this document compare the individual sample results to the PRG values. Although useful for initially locating potential remediation areas, there are instances where there are sporadic exceedances but where other nearby samples are below the PRGs. It is unclear whether the planned approach would therefore involve multiple "spot" remediation, or be restricted to remediation of larger areas. Perhaps more importantly, since average

concentrations were used to assess potential risks, then average media concentrations should be used to assess candidate areas for remediation. EPA should provide a discussion of the application of PRGs as surface area weighted concentrations and not bright line, not-to-exceed concentrations.

7.0 CONCLUSIONS

This review has identified, documented and discussed the technical shortcomings of the analyses that comprise the EPA Ecological PRG report. We have focused on those exposure scenarios and risk assessment topics that have the greatest implications for setting target remediation goals and assessing remedial options. Therefore, we have discussed the substantial shortcomings and biases inherent in the methods and data EPA has used in developing the PRGs for the Centredale Manor site.

The most critical finding of our review is that EPA has mischaracterized the findings of the BERA for demersal and pelagic fish populations. Our review of the BERA shows that EPA *did not* find a significant risk of harm to these receptors from exposure to site-related chemicals. However, EPA erroneously reported the opposite of those findings in the PRG document and unnecessarily computed PRGs for these receptors. This is a substantial error which by itself warrants that EPA revise the PRG document to remove these receptors.

The aforementioned error and the other technical shortcomings noted in our review indicate that the present PRG document is seriously flawed and requires revision. The development of PRGs requires a much more robust and unbiased risk assessment in which state-of-the-art methods of risk analysis are coupled with proper interpretation and analyses of available data. The development of PRGs does not meet this standard in its present form. In the absence of revisions to the BERA and PRG development documents, EPA's analysis lacks the level of sophistication required to serve as the basis for establishing technically defensible remedial cleanup goals for the Centredale Manor Restoration Project Superfund Site.

8.0 REFERENCES

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