SDMS DocID 273415

Superfund Records Center
SITE: Centredale
BREAK: 3.10
OTHER: 273415

Comments on Baseline Human Health Risk Assessment for the Centredale Manor Superfund Site

This report provides Sciences International, Inc.'s (Sciences) review of the *Baseline Human Health Risk Assessment – Interim Final, August 2004* (BHHRA) for the Centredale Manor Restoration Project Superfund Site, North Providence, Rhode Island (Site). The review was conducted to determine whether the BHHRA conforms to applicable risk assessment guidance. It also identifies the risk driving pathways and chemicals and contains a preliminary analysis of the data as it pertains to the original source(s) of dioxin and other contaminants.

Overall, the BHHRA was well developed. It evaluates potential human health risks for current and future uses of the site. It presents quantitative risk estimates for the following exposure pathways: consumption of locally caught fish and contact with surface water, sediment and soil (e.g., while someone swims and wades in the on-Site portion of the Woonasquatucket River). It evaluates risk separately for five Exposure Areas of the Site: Allendale Pond, Lyman Mill Pond, Manton Pond, Dyerville Pond and the Fogarty Center. One area was evaluated for risk to represent background, Greystone Mill Pond, located upriver of the Site. Another area, Assapumpset Brook and Pond was designated as a "reference' area for identification of background-related compounds; this area is located on a tributary to the Woonasquatucket River which discharges into Lyman Mill Pond. Figure 1 shows these "exposure" areas on a topographic map of the site and surrounding areas. Figure 2 shows this information on an aerial photograph of the Site and environs. The potentially exposed populations evaluated by the BHHRA include current and future residents along the river and local and visiting recreational anglers (subsistence fishers are separately evaluated in an Appendix). Risks were computed to both adults and children. Risks for employees at the Fogarty Center were calculated assuming exposure to soil only.

We understand from documents other than the BHHRA that the areas of Brook Village and Centredale Manor (shown in Figures 1 and 2) had been used in the past for woolens manufacturing (prior to 1936), by the Atlantic Chemical Company/Metro Atlantic Inc for soap/hexachlorophene manufacture (1940-early 1970s) and by the New England Container Company (NECC) (1952-1969) for drum reconditioning. The BHHRA indicates that the "main area of the Site" was used by a chemical manufacturing company (which produced hexachlorophene, among other chemicals) and a drum recycling facility. It notes that the areas have been capped and currently contain apartments for the elderly. The BHHRA does not evaluate risks at the "source" areas.

The BHHRA finds that the risk of cancer and non-cancer end-points to current and future residents along the river and recreational anglers which it associates with the Site are high. Of the scenarios evaluated, fishing and exposure to surface water give the highest risks. Although the BHHRA considers the effect of a number of contaminants, it finds that the majority of the cancer risk is associated with 2,3,7,8-tetrachlorodibenzodioxin (TCDD), one of the 17 member family of dioxins and dioxin-like furans, and to a lesser degree polychlorinated bipheynls (PCBs). Non-cancer risks are largely associated with PCBs in fish. The BHHRA's cancer and non-cancer risk estimates exceed the EPA's presumptively acceptable risk range of 1 in one million (1×10^{-6}) to one in ten thousand (1×10^{-4}) for cancer risks and, for non-cancer risks a hazard index of 1. The highest cancer risks ($\sim 10^{-2}$) are 100 times greater than the upper end of the presumptively acceptable range and non-cancer risks exceed the presumptively acceptable hazard index level of 1 by almost a factor of 30.

This report is split into the following sections: 1) conformance with EPA guidance, 2) hazard identification, 3) exposure assessment, 4) toxicity assessment, 5) risk characterization and results and 6) sources of contamination.

1. Conformance with Risk Assessment Guidance

In general the BHHRA conforms to federal EPA risk assessment guidance and EPA Region 1 risk assessment guidance ("Risk Updates"). We have checked key equations and parameters used in the risk assessment and, apart from certain site-specific parameter inputs which deviate from published default values and a few issues on which we comment in the following sections, all the assumptions, equations and values were found to be consistent with the guidance.

- *Comment:* The BHHRA references the Interim Review Draft version of the Risk Assessment Guidance for Superfund, Part E (RAGS Part E) which is related to dermal exposure risk assessment. The finalized version of this document, published in July 2004, prior to the date of the BHHRA should have been referred instead. However, the methodology and critical parameter value (the aqueous permeability rate for dioxin) are unchanged in the final version.

2. Hazard Identification

Hazard Identification involves selection of chemicals of potential concern (COPCs) based on comparison of the concentrations to health-based values. The BHHRA clearly sets forth the protocol it used to determine COPCs, which it listed for each media (soils, sediments, surface water and fish) separately at each exposure area and combined for the background and reference areas.

- *Comment:* The BHHRA sets forth and implements reasonable hazard identification for the Site. As a separate COPC list was derived for the combined background and reference areas, we question why these were treated as if it were an exposure area. The BHHRA states that the number of COPCs are "reasonably consistent" among the combined background/reference area and the four river exposure areas. It would be more logical to include the same COPCs in the background and reference areas that were identified in the exposure areas.

3. Exposure Assessment

In general the BHHRA presents a detailed exposure assessment. Exposure assessment provides the assumptions and calculations of the dose received by individuals engaged in certain activities that result in exposure to contaminants in various media. It is standard practice to assume activities at the site are not restricted by the knowledge that the site may be contaminated. Accordingly the BHHRA postulates that individuals can fish, swim and wade in and around the Woonasquatucket River at the defined exposure areas. It is worth noting, however, that the fact that river is in an urban setting may affect the extent of these activities.

Exposure assessment considers both the potential environmental concentrations of chemicals and the degree to which contact occurs. The former are estimated using sampling data. In accordance with EPA policy and risk assessment guidance, the concentration values assigned to a pathway of exposure (exposure point concentration or EPC; for example, the concentration of a chemical in surface water), for a reasonable maximum exposure (RME) evaluation is calculated as the 95th upper confidence limit on the mean (95%UCL) of all surface water samples. The 95%UCL is a statistical value that expresses a 95% confidence that the true mean of a sample set is not lower than the value; thus the exposure point concentration is unlikely to be underestimated. Arithmetic average values are used for the typical or central tendency exposure evaluation.

- *Comment:* The BHHRA conforms to EPA guidance for derivation of exposure point concentrations.

The degree to which an individual is exposed is governed by a combination of exposure factors. For example, in a fishing scenario, the exposure factors are the daily fish consumption rate, number of exposure events and the duration of the exposure period. Combined with the EPC and taking into account body weight and the time over which the exposure is averaged, the individual's chemical dose from the pathway is determined. The dose is expressed in terms of the mass of chemical intake per mass of bodyweight per day. The BHHRA clearly presents the equations and parameters for the calculation of dose.

- *Comment:* The BHHRA conforms to EPA guidance for derivation of chemical intakes and dose.

We raise the following specific issues:

Ingestion of Contaminated Fish

Ingestion of fish is the major risk driver for both the residential and recreational angler populations. The resident who wades and swims is also a recreational angler but the recreational angler only fishes. The fish consumption rates were chosen using data from a study of recreational anglers in Maine (Ebert et al. 1993). The study did not present consumption rates for an individual but measured catch per household. For adult anglers, the BHHRA assumed that 100% of the catch was consumed by a single individual. An upper bound ingestion rate of 14 g/day was chosen for the RME for adults[1]. The central tendency (CT) consumption rate of 8.9 g/day for an adult was derived from the arithmetic mean of household catch in the Ebert et al. study. The BHHRA assumes that children eat only a portion of the household catch based on consideration of body weight.

- Comment: The BHHRA appropriately considers regional fishing habits in its derivation of site-specific fish ingestion rates. It points out that the assumption for adults that the entire catch is consumed by one adult introduces a likely overestimate in the dose but to account for sharing would introduce additional uncertainty in the estimates. The magnitude of this simplifying assumption is, in our estimation, not likely to affect the risk outcomes by more than a factor of about 2.

Dermal Contact with Surface Water and Sediments

The dermal contact with surface water is the other major contributor to risk at exposure areas. The dose to the exposed individual is based on the time exposed, the exposed skin surface area and the compound's dermal permeability rate.

---

[1] EPA recommends a consumption rate of 17.5 g/day in *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health* (October 2000)

The RME risk calculations assume that a resident visits the site 4 days per week for 13 weeks during the summer (52 days per year). During each of those visits an exposure time of 1 hour is assumed. The BHHRA assumes that an individual (adult and older child resident) is expected to go wading three out four days in each week (39 hours per year) and go swimming on the remaining day (13 hours per year). The younger child only wades.

- *Comment:* EPA's Risk Assessment Guidance for Superfund (RAGS, Part A) gives the national average for swimming as 7 days/yr and 2.6 hrs/day (18 hours per year). EPA Exposure Factors Handbook (1997) recommends 12 days/yr and 1 hr/day (12 hours per year). It also gives a 90th percentile of 3 hrs/event and 12 events/year (36 hours per year). The assumptions of the BHHRA in this regard are reasonable.

For adults and older children, the BHHRA assumes that for both wading and swimming activities, the full body surface area is exposed. For the young child wading, the BHHRA assumes exposure only to the hands, legs and feet.

- *Comment:* The assumption made for adults and older children that the same skin surface area is exposed during wading and swimming is characterized as "conservative" in the BHHRA. However, unless wading for these individuals involves wading up to the neck (for the duration of each and every event), this assumption is unrealistic. The BHHRA should have assumed less than full body surface area for wading. Correction of this issue would reduce the surface water-related risks to adult and older child residents by a factor of about 1.5.

Another key component of the dermal exposure calculations is the permeability constant of the chemical being examined. This factor determines the rate at which a chemical in water penetrates the dermal barrier and is absorbed into the body. Table 4s presented in the BHHRA refers to a "PCevent" table where apparently the interim calculation of chemical-specific absorbed dose is presented, but the table is absent from the copy of the report we reviewed.

- *Comment:* We cannot assess the permeability constants used in the dermal exposure pathway risk evaluation.

Surface water exposure point concentrations from the Lyman Mill reach were used to represent surface water concentrations for the Manton and Dyerville reaches because there were no site-specific concentrations for those areas. The BHHRA's authors consider the Lyman Mill reach to be an adequate surrogate because of the proximity of the downstream locations to the upstream surrogate.

The BHHRA points out that the surface water concentrations for TCCD (which alone represents over 99% of the risk from dioxin-like compounds in water) are likely "associated with suspended particulate matter, perhaps derived from sediments." It points out that TCCD has a very low solubility in water and consequently can be expected to have a high affinity for organic matter in suspended sediments. The BHHRA acknowledges that the equation for the dose from dermal contact with water relies on a permeability constant (Kp) which is valid only when the water concentration is expressed in terms of the dissolved concentration of the chemical. The BHHRA for TCCD notes that no empirical Kp value has been measured; the published Kp value recommended by US EPA and published in RAGS Part E is estimated by a model and the estimated value is outside of the model's effective predictive domain. Furthermore the BHHRA authors state that the model "would likely estimate absorption rates when in fact no absorption was taking place."

- *Comment:* While it is appropriate to compute exposures from incidental ingestion of surface water while swimming using total water concentrations, it is not appropriate to use these values when estimating dermal absorption which require knowledge of the concentration in the dissolved phase. The exposure and risk associated with the dermal contact pathway computed using flawed concentration values should not have been included in the BHHRA until appropriate data have been collected. This issue has a potentially large effect on the aggregate risk estimates for potentially exposed individuals as we will describe in Section 5 of this report.

4. Toxicity Assessment

The aim of the Toxicity Assessment section of the BHHRA is to characterize the relationship between exposures to chemicals of potential concern (COPCs) and the incidence of adverse health effects. For each COPC, the BHHRA presents dose-response information divided into two major categories: 1) non-carcinogenic health effects and toxicity, and 2) carcinogenicity and toxicity information from human epidemiologic data or laboratory studies. The BHHRA evaluated all the COPCs for potential non-carcinogenic health effects, and those classified by USEPA as *known, probable* or *possible* human carcinogens were evaluated for potential carcinogenic effects.

Carcinogenic effects are considered non-threshold, meaning that any dose is assumed to pose a defined probability of generating a biological response. A two-part evaluation system is used where the substance is assigned a weight-of-evidence classification and then a numerical factor (slope factor) is calculated to reflect carcinogenic potency. Cancer slope factors and unit risks are calculated for known or suspected carcinogens. The cancer slope factor is an upper bound estimate of the slope of the dose-response curve

which describes the potency of the chemical. It is presented in units of inverse of dose $(mg/kg\text{-}day)^{-1}$ and is based on a lifetime average daily dose.

- *Comment:* The dose-response assessment for carcinogenic effects adhered to the conservative, public health protective, standards set forth by USEPA in its Superfund guidance.

Non-carcinogenic endpoints, such as liver toxicity, reproductive and developmental effects, neurotoxicity, are treated under the assumption that threshold exposure levels exist below which adverse effects are not expected. USEPA has standards and guidelines for determining safe levels of exposure to noncarcinogens. This is accomplished by calculating a reference dose for oral routes of exposure[2].

- *Comment:* The dose-response assessment for non-carcinogenic effects adhered to the standards and guidelines set forth by USEPA's standards and guidelines for the dose-response assessment for non-carcinogenic effects.

Dermal dose-response values are calculated from oral dose-response values for cancer (slope factors) and non-cancer effects (reference doses) using an absorption factor. The absorption factor accounts for the amount of the chemical that is absorbed from the gastrointestinal tract after oral exposure. Therefore, the absorbed dose is an internal dose that is available for biological interaction and effects.

- *Comment:* The dose-response assessment for dermal toxicity adheres to the standards and guidelines set forth by USEPA.

We checked all the values and sources of the toxicity factors used in the BHHRA as provided in Tables 5.1 (non-cancer) and 6.1 (cancer).

- *Comment:* The toxicity factors used in the BHHRA are generally correct. In those few instances where we could not readily confirm the values exist for certain non-cancer agents, however, the affected chemicals do not appear to contribute significantly to the risk outcome.

The following compound-specific comments are made:

---

[2] The oral reference dose (RfD) is defined by USEPA "as an estimate (with uncertainty spanning perhaps an order magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime."

Dioxins

With respect to the major risk driver at the site, 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD), it is generally recognized that this compound is the most potent member of the family of dioxin-like compounds which include dioxin-like furans and certain PCBs. In the body, TCDD binds with a protein known as the aryl hydrocarbon receptor (AhR) which is the mechanism through which TCDD exerts its effects. Other dioxins, dioxin-like furans, and co-planar (dioxin-like) PCBs exert their toxicity through the same mechanism. However, the magnitude of the toxic effects is proportional to the affinity with which these compounds bind to the AhR. The toxicity of these other compounds is expressed using Toxicity Equivalent Factors (TEFs) which relate their toxic potential to that of TCDD

The toxicity of TCDD has been extensively studied and characterized and a cancer potency factor has been published in the Health Effects Assessment Summary Tables (USEPA, 1997). This cancer slope factor is 150,000 per mg/kg-day, which is very high compared to other carcinogens. Potency factors have not been determined for the other AhR agonists (dioxin-like compounds), but are calculated based on the system of TEFs. TCDD is arbitrarily assigned a TEF of 1, and the other dioxin-like compounds are assigned TEFs that are fractions of 1 based on their relative potency in binding the AhR. The potency of the dioxin-like compound is estimated by multiplying the measured media concentration by the TEF for the particular compound to yield a toxic equivalent quotient (TEQ). The TEQ concentration is determined by summing the TCDD normalized concentrations for each dioxin-like (AhR-binding) compound. This procedure does not account for toxicological effects that are not mediated by the AhR. However, the TEF/TEQ methodology is widely regarded as the state-of-the-art for assessing the risk of TCDD and dioxin-like compounds.

USEPA has released a preliminary draft document, *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin and Related Compounds* (2000). For cancer effects, USEPA recommended a revised cancer slope factor of 1,000,000 per mg/kg-day for the upper-bound cancer risk. This represents a 7-fold increase in cancer risk estimates to the current upper-bound slope factor of 150,000 per mg/Kg-day.

- *Comment:* The BHHRA uses the current published toxicity factor for TCDD (which form the basis for the potencies of all other dioxin-like compounds) instead of the revised draft value. This election is appropriate since the revised value is still in draft status.

Hexachloroxanthene (HCX) is identified by the BHHRA as a dioxin-like compound but notes that USEPA has not formally released dose-response values for HCX, and toxicity

information is limited. In Appendix H, the BHHRA estimates a TEF of 0.0002 for HCX based on a preliminary and unpublished report, and despite the lack of chronic toxicity information, limited metabolism information, and uncertainties with the available studies. However, the risks estimated for HCX, which were not carried through into the main body of the report, were below levels of potential concern and therefore would not contribute significantly to the overall risk values.

For non-cancer effects of dioxin and dioxin-like substances, the BHHRA notes that a reference dose has not been developed. The BHHRA does not assess quantitatively the non cancer risk for dioxins.

Lead

Dose-response values for lead are not available. USEPA recommends using lead biokinetic uptake models and compare these estimated intakes with threshold blood lead level models for children and adults. In Appendix E, the BHHRA evaluated lead in soil and sediment using USEPA's Integrated Exposure Uptake Biokinetic Model and concluded that blood levels are not likely to be of concern. For lead exposure via fish consumption, the BHHRA compares the conditions at the Site to the results of a lead modeling study done for the Columbia River and concludes that lead in fish tissue at the Site is unlikely to result in blood lead levels in children or fetuses that are above thresholds of concern.

Chromium

Speciation analyses for chromium were not performed. The BHHRA attempted to provide a conservative assessment of risk by treating the total chromium concentrations as if they were hexavalent chromium, the most toxic form of chromium.

- *Comment:* The BHHRA's assumption that all of the measured chromium is in its hexavalent form is overly conservative and not appropriate. Although the BHHRA acknowledges this shortfall, at a minimum some factor should have been applied to parse the hexavalent form from the less toxic trivalent and elemental forms. However, this metal had very little influence on the aggregate risk results.

5. Risk Characterization

Risks are computed by comparing the dose estimates to potency factors. Cancer risks are the product of the cancer slope factor and the lifetime average daily dose estimates. A cancer risk is the upper bound on the probability that an excess cancer will arise from the exposure or a combination of exposures. For example an estimated aggregate cancer risk of 1×10^{-3} indicates the exposed individual is not likely to have greater than a one in one thousand chance of contracting an additional cancer from all site-related exposures combined.

For non-cancer effects, average daily dose estimates are compared to non-cancer reference doses. If the received dose is less than the reference dose (a hazard index less than one) then adverse non-cancer effects are unlikely; if the dose is higher than the reference dose, an adverse health effect is possible but not inevitable (this is due to the level of safety built into the reference dose).

The BHHRA's cancer and non-cancer risk estimates exceed the EPA's presumptively acceptable risk range of 1 in one million (1×10^{-6}) to one in ten thousand (1×10^{-4}) for cancer risks and, for non-cancer risks a hazard index of 1. The highest cancer risks ($\sim 10^{-2}$) are 100 times greater than the upper end of the presumptively acceptable range and non-cancer risks exceed the presumptively acceptable hazard index level of 1 by almost a factor of 30.

- *Comment:* We found no computational errors in the risk estimates of the BHHRA.

The BHHRA finds high levels of Site-related potential cancer and non-cancer risk to current and future residents along the river and recreational anglers. It should be borne in mind that the RME risk levels are plausible upper bound estimates, the true risks being lower. Fishing and exposure to surface water give the highest risks. The risks to residents are higher than recreational anglers because the residents are assumed to be recreational anglers with the addition of swimming and wading exposures.

Tables 1 and 2 show the risk driving chemicals at the various areas of the site (note that Greystone Mill Pond was designated as a background area). The chemicals have been grouped by risk to provide an indication of the relative importance of risk driving chemicals in each area. The majority of the cancer risk is associated with dioxins and dioxin like furans expressed as TCDD (i.e., toxic equivalent quotient, TEQ) followed by

PCB-Aroclors and dioxin-like PCB congeners[3]. Non-cancer risks, not shown in the tables, are largely associated with PCBs in fish.

Incremental risks were presented in the risk assessment by subtracting the aggregate risk at the background location (Greystone Mill Pond) from the aggregate risk at each of the individual exposure areas.

- *Comment:* This approach may be flawed due to differences in the selection of chemicals of potential concern (COPC) for each exposure location. According to the risk assessment COPCs were included in the risk calculation only if the concentrations at the location exceeded screening values. Therefore, chemicals present at the background area may not be included in the risk calculations for that area, despite being included in risk calculations for other areas. It would be more accurate to subtract the background risks on a chemical by chemical basis from the risks at the exposure areas but this would require the background location to include all COPCs in the exposure areas. Computationally it is much easier to simply calculate total risks independently for each area and subtract the background total from another area, but valuable information regarding risk driving chemicals can be lost. Despite this we do not believe the incremental risk calculation is misleading in this case since the major chemicals driving risk in the exposure areas are identified in the background area. However the origin of certain chemicals with possible off-site sources, for example PCBs, may be obscured by this method.

Figures 3 through 12 (Appendix A) depict the relative contribution of risk driving chemicals for the resident and recreational angler and the magnitude of the pathway-specific risks. It is notable that for both PCBs and dioxins the risks are significantly higher in Allendale Pond than in the background location. The BHHRA points out that this is strongly suggestive of a source on the main area of the Site (i.e., the property on which Atlantic Chemical and NECC were located). With regard to the Fogarty Center, the BHHRA concludes that the risks to workers are within the acceptable range.

- *Comment:* The effect of assuming that surface water concentrations in Manton Pond and Dyerville Pond are equivalent to those of Lyman Mill Pond is seen in the increasing predominance of surface water related risk in these downstream areas which have less fish consumption related risk. This may be an artifact of the use of Lyman Mill as a surrogate and may not reflect conditions in these areas.

[3] PCB Aroclors (commercial mixtures of PCBs) typically contain both non-dioxin like and dioxin like PCB congeners but are often evaluated separately because concentrations are expressed as Aroclors and Aroclors and dioxin like PCB congeners have separate published toxicity factors.

It is important to note that although dioxin and furan congeners other than TCDD have been detected in site media their contribution to the TEQ value is negligible; TCCD alone is responsible for the dioxin/furan risk. This can be seen in Figures 13 though 15 where we depict the concentrations of individual dioxin/furan congeners alongside the TEQ profile. These figures, based on the more highly impacted samples in the EPA database that are expected to drive risk, indicate little if any contribution to the total TEQ of congeners other than TCDD.

- *Comment:* Dioxins and furans other than TCCD are not responsible for the dioxin risk.

As described in the exposure assessment comments the risks associated with dermal contact with surface water are flawed.

Comment: For residents, the risk associated with contact with surface water is approximately equal to that of fish consumption (see, for example, Figures 9 and 10 in Appendix A). The surface water risks are dominated by the dermal exposure pathway. The risks computed for this pathway have a large degree of uncertainty and are acknowledged in the BHHRA to be overestimated. Although it is appropriate to evaluate swimming and wading activities, the quantitative estimation of risks associated with dermal exposure should not have been included in the risk assessment because the dissolved water concentrations required in the exposure model were not available. The dissolved water concentrations are likely to be significantly lower than the total water column concentrations because TCDD is only sparingly soluble and has a high affinity for binding to suspended sediment.

Since other river activities (ingestion of surface water, soil and sediment ingestion and dermal contact) are estimated by the BHHRA at levels far below those of dermal contact with surface water, in most cases under 1×10^{-4} (the upper end of the presumptively acceptable risk range), the inclusion of this uncertain exposure biases the decision making process towards unnecessary remedial actions.

6. Sourcing Issues

Past use of what the BHHRA assumed to be source areas included woolens manufacturing, soap/hexachlorophene manufacture and drum reconditioning. With regard to sources of site-related contamination we provide here only a preliminary review, focusing on potential sources of dioxin, specifically the manufacture of hexachlorophene.

Hexachlorophene is prepared from 2,4,5-trichlorophenol (2,4,5-T)[4]. Hexachlorophene contains dioxin as an impurity, specifically 2,3,7,8-TCDD (TCDD) at concentrations <15,000 parts per trillion (ppt).

As we currently understand it, during the manufacturing process the hexachlorophene (or 2,4,5-T) is purified by removing TCDD (the amount of TCCD remaining in the hexachlorophene depends on the effectiveness of the purification process). The removed TCDD ends up in wastewater, clay, and still bottom waste, the latter being a thick residue containing highly concentrated dioxin. These wastes were responsible for the contamination and subsequent closing down of the town of Times Beach, Missouri in the early 1980s. In that case still bottom wastes were mixed with other waste oils and sprayed on roads and farms residences. The concentration of TCCD in still bottom wastes can be 2,000,000,000 ppt or greater; this concentration equates to about 2 grams TCDD per liter of waste.

- To demonstrate just how this waste could have affected the area, we conducted an exercise to predict the concentration of TCDD in soils (or sediments) assuming a single 55-gallon drum of still bottom waste (containing about 1 pound of TCDD) was released and incorporated uniformly into a volume of soil/sediment approximately equal to the aerial extent of the entire Site (260 acres, based on the extent of the soil and sediment sampling area from Brook Village to Dyerville Pond) by a mixing depth of 1 ft. This rough calculation results in a concentration of TCDD in all soil or sediment close to 1 μg/Kg[5]. This result is comparable to the average sediment concentrations detected at the various sites investigated: Allendale Pond 5.74 μg/Kg; Lyman Mill Pond 1.79 μg/Kg; Manton Reach 0.43 μg/Kg; Dyerville Reach 0.11 μg/Kg.

It is not only the still bottom waste that could have contributed to site dioxin contamination; the raw materials, wastewater, and hexachlorophene itself contain TCDD. We currently do not know if other dioxin congeners other than TCCD are associated with the process but have found no reference other than to TCDD. The BHHRA notes the similarity between

[4] 2,4,5-T is the agent that was used to produce herbicides including Agent Orange.
[5] 1 μg, or micro-gram, is one millionth of a gram.

the TCDD contamination in the environment and the facility in Verona, Missouri that manufactured hexachlorophene[6].

Figures 16 and 17 show dioxin congener profiles for highly impacted sediment and surface water samples at the site. Preliminarily these profiles contrast with those typically found in emissions from combustion sources as shown in Figures 18 and 19[7]. Since dioxin-like congeners will undergo environmental transformations that may significantly alter their profile in environmental media, we suggest that a fingerprinting evaluation be conducted to better understand the likely source of the contamination in the environment. Although our analysis is preliminary, the information suggests that the source of dioxin contamination is the chemical manufacturing process and not the drum reconditioning activities.

Hexachloroxanthene (HCX)

Also associated with hexachlorophene production and found with TCCD-bearing wastes is hexachloroxanthene (HCX). Hexachlorophene contains "about 100 mg/Kg" HCX (International Programme on Chemical Safety, Environmental health criteria 88, 1989).

As shown in Figures 20 and 21, both TCDD and HCX are found in all areas of the site. HCX is present in all media (sediment, soil, surface water and fish) including the more highly impacted samples that drive the risk.

EPA Region 1 comments on finding HCX at the Site and associates it with "hexachlorophene production" at the Site (EPA Region 1 Action Memorandum, January 2001).

HCX is not on list of analytes for combustion sources burning hazardous waste (EPA 1998). In our experience with an actual hazardous waste burning facility, HCX was not detected in air emissions from a hazardous waste burning cement kiln facility evaluated by Sciences (dioxins were detected).

The BHHRA comments on the presence of HCX both from a standpoint of it being a potential chemical of concern for health risk (although no potency factor has been developed for this compound) and from the perspective of HCX as a marker for the source

[6] The Verona site that was responsible for the TCDD contamination at Times Beach, Missouri.

[7] For example, the dominance of TCCD is inconsistent with the profiles for emissions from incinerators. Although NECC did not operate an incinerator (we understand that drums were subject to heat at temperatures around 500°F, a temperature well below that of hazardous waste incinerators), the profiles for incinerator emissions provide at least a rough basis for comparison.

of the contamination. The BHHRA's comments on the sourcing issues are quoted in the following bullets in the order in which they appear:

- Page 1-17

  In sediment, 2,3,7,8-TCDD and HCX, and 2,3,7,8-TCDD equivalent concentrations are at least 100 times higher in Allendale Reach than in background and reference areas. "This suggests a Site-related impact on dioxins/furans and HCX in sediments in areas adjacent to and downstream of the source area."

- Page 1-22

  "Sediment concentrations of 2,3,7,8-TCDD, HCX and PCB-77 are dramatically higher" in the four areas of Allendale, Lyman, Manton and Dyerville ponds. Sediment concentrations were "highest in the two ponds (Allendale Pond and Lyman Mill Pond) adjacent to and immediately downstream of the source area". "This pattern strongly indicates that these contaminants are related to the source area."

  "HCX was detected in white sucker tissue from Allendale Pond and Lyman Mill Pond but not at either the Greystone Mill Pond upstream background location or at the Assapumpset Pond reference location"

  "There is information that indicates that 2,3,7,8-TCDD and HCX would have been expected to be present at the source area due to the manufacture of hexachlorophene...Mr. Cleary indicated in his deposition that crude trichlorophenol was treated with chemicals at the source area in order to purify it"

  "It is also reported that HCX is a by-product of the synthesis of hexachlorophene and that hexachlorophene contains approximately 100 mg/kg of 1,2,4,6,8,9-hexachloroxanthene (WHO,1989)."

- The final conclusions do not discuss further the issue of HCX and 2,3,7,8-TCDD.

7. Conclusions

During our review of the BHHRA we identified a number of issues which result in risks being overestimated. Most importantly is the application of a dermal exposure equation using total water concentrations rather than the dissolved concentrations required by the formula. Since surface water exposure pathway related risks are at least equal to those of fish ingestion in areas near the presumed source areas (and in the lower reaches much higher than the fish ingestion risks), correction of this issue might have a significant impact on the risk assessment perhaps to the point of affecting the determination of final remedies.

The BHHRA is distinct from the risk assessment that will be required during the remedial design stage according to the recommendations of the National Academy of Science. USEPA has developed eleven principles to be followed when managing the cleanup of sites with contaminated sediments. These principles closely follow the National Research Council's risk-management strategy for PCB-contaminated sediments which provides a framework for assessing the risks and managing the remediation of PCB-contaminated sediments. Both long- and short-term risks must be evaluated in the decision-making process to generate the basis for a risk-based remedy selection.

Sciences concludes that the former manufacture of hexachlorophene at the Site is likely responsible for the site related dioxin contamination. This conclusion is consistent with statements made in the BHHRA report.

We recommend an in-depth analysis of the data at the Site using fingerprinting, GIS and other forensic techniques to provide a better and more defensible understanding of the possible sources of the contamination. We also recommend a review of available documents related to the historical use of both the Site itself and upstream areas. Upstream sources may have impacted the Site with contaminants that are common in industrial areas such as dioxins, polycyclic aromatic hydrocarbons, and polychlorinated biphenyls (PCBs). All of these contaminants are of concern at the subject property.

Appendix A

Tables and Figures

Table 1. Chemical-Specific Cancer Risks for the Recreational Angler

Risks 10^{-6} - 10^{-4}

Chemical of Concern	Allendale Pond	Lyman Mill Pond	Manton Reach	Dyerville Reach	Greystone Mill Pond
4,4'-DDE		1.0E-06			
Aroclor-1254			4.0E-05	8.4E-05	2.9E-05
Aroclor-1268	5.0E-06	4.0E-06	1.3E-05		1.6E-05
Arsenic		2.4E-06			
Benzo(a)pyrene		3.7E-06		2.6E-06	
Dieldrin	1.3E-05	8.4E-06		1.6E-05	3.0E-06
Technical Chlordane	1.5E-05	3.3E-05		1.3E-05	6.0E-06
TEQ (Dioxins/Furans)					2.3E-05

Risks 10^{-4} - 10^{-3}

Chemical of Concern	Allendale Pond	Lyman Mill Pond	Manton Pond	Dyerville Pond	Greystone Mill Pond
Aroclor-1254	3.5E-04	3.9E-04			
TEQ (PCB Congeners)	4.4E-04	5.7E-04			

Risks >10^{-3}

Chemical of Concern	Allendale Pond	Lyman Mill Pond	Manton Pond	Dyerville Pond	Greystone Mill Pond
TEQ (Dioxins/Furans)	5.0E-03	5.8E-03	1.1E-03	1.7E-03	

Total Risks

Chemical of Concern	Allendale Pond	Lyman Mill Pond	Manton Pond	Dyerville Pond	Greystone Mill Pond
All COCs	5.8E-03	6.8E-03	1.1E-03	1.8E-03	7.7E-05

Table 2. Chemical-Specific Cancer Risks for the Resident

Risks 10^{-6} - 10^{-4}

Chemical of Concern	Allendale Pond	Lyman Mill Pond	Manton Pond	Dyerville Pond	Greystone Mill Pond
2,3,7,8-TCDD	1.6E-05				
4,4'-DDE		1.0E-06			
Aroclor-1254			4.0E-05	8.4E-05	2.9E-05
Aroclor-1268	5.0E-06	4.0E-06	1.3E-05		1.6E-05
Arsenic	1.4E-06	4.0E-06			
Benzo(a)pyrene	1.1E-05	8.4E-06			
Dibenzo(a,h) anthracene	2.4E-06	1.3E-06			
Dieldrin	1.3E-05	8.4E-06		1.6E-05	3.0E-06
N-Nitroso-di-n-propylamine		1.5E-06			
Technical Chlordane	1.5E-05	3.3E-05		1.3E-05	6.0E-06
TEQ (Dioxins/Furans)					6.5E-05

Risks 10^{-4} - 10^{-3}

Chemical of Concern	Allendale Pond	Lyman Mill Pond	Manton Pond	Dyerville Pond	Greystone Mill Pond
Aroclor-1254	3.5E-04	3.9E-04			
TEQ (PCB Congeners)	4.4E-04	5.7E-04			

Risks >10^{-3}

Chemical of Concern	Allendale Pond	Lyman Mill Pond	Manton Pond	Dyerville Pond	Greystone Mill Pond
TEQ (Dioxins/Furans)	1.3E-02	1.2E-02	7.2E-03	7.8E-03	

Total Risks

Chemical of Concern	Allendale Pond	Lyman Mill Pond	Manton Pond	Dyerville Pond	Greystone Mill Pond
All COCs	1.4E-02	1.3E-02	7.2E-03	7.9E-03	1.2E-04

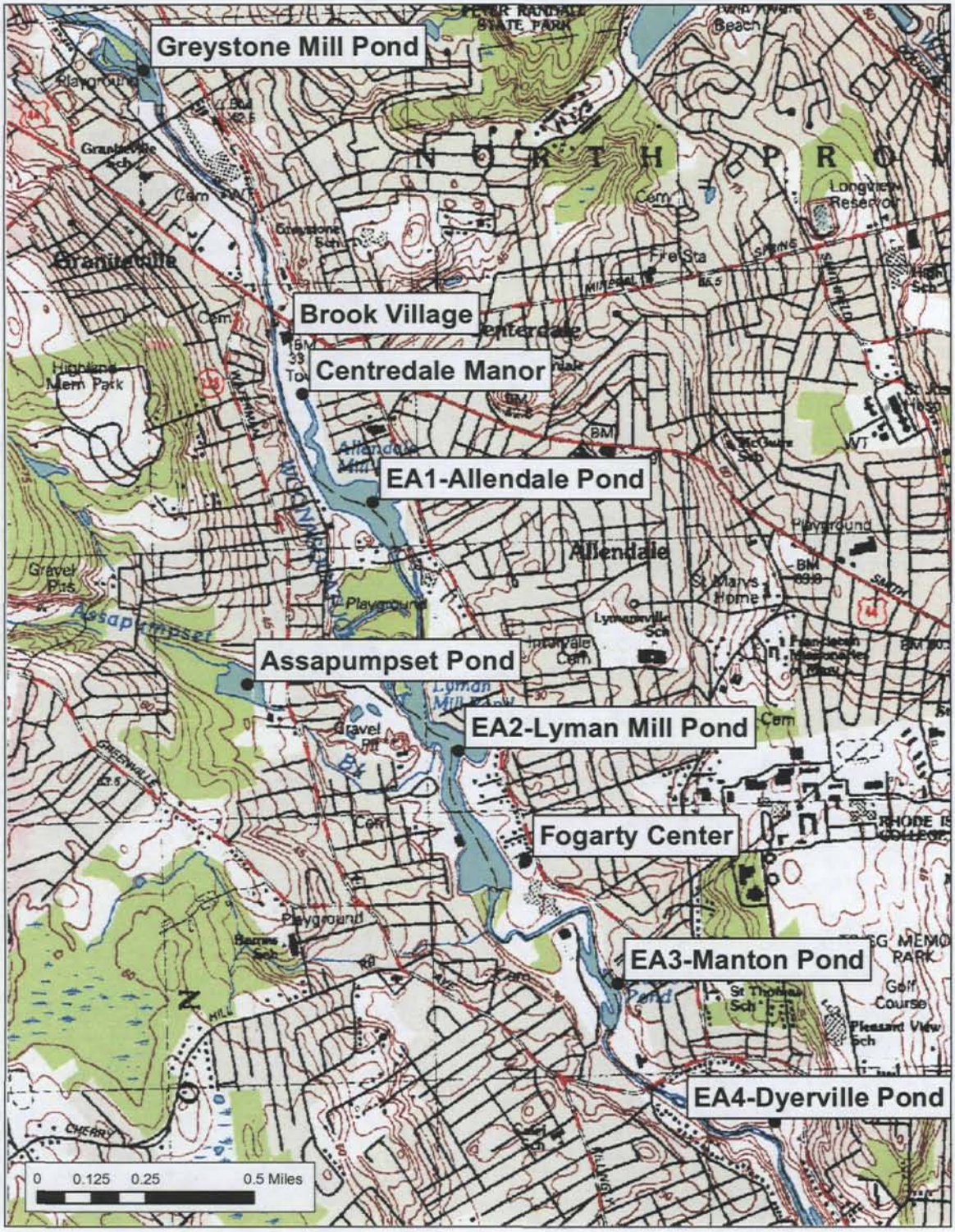


Figure 1. Site Overview

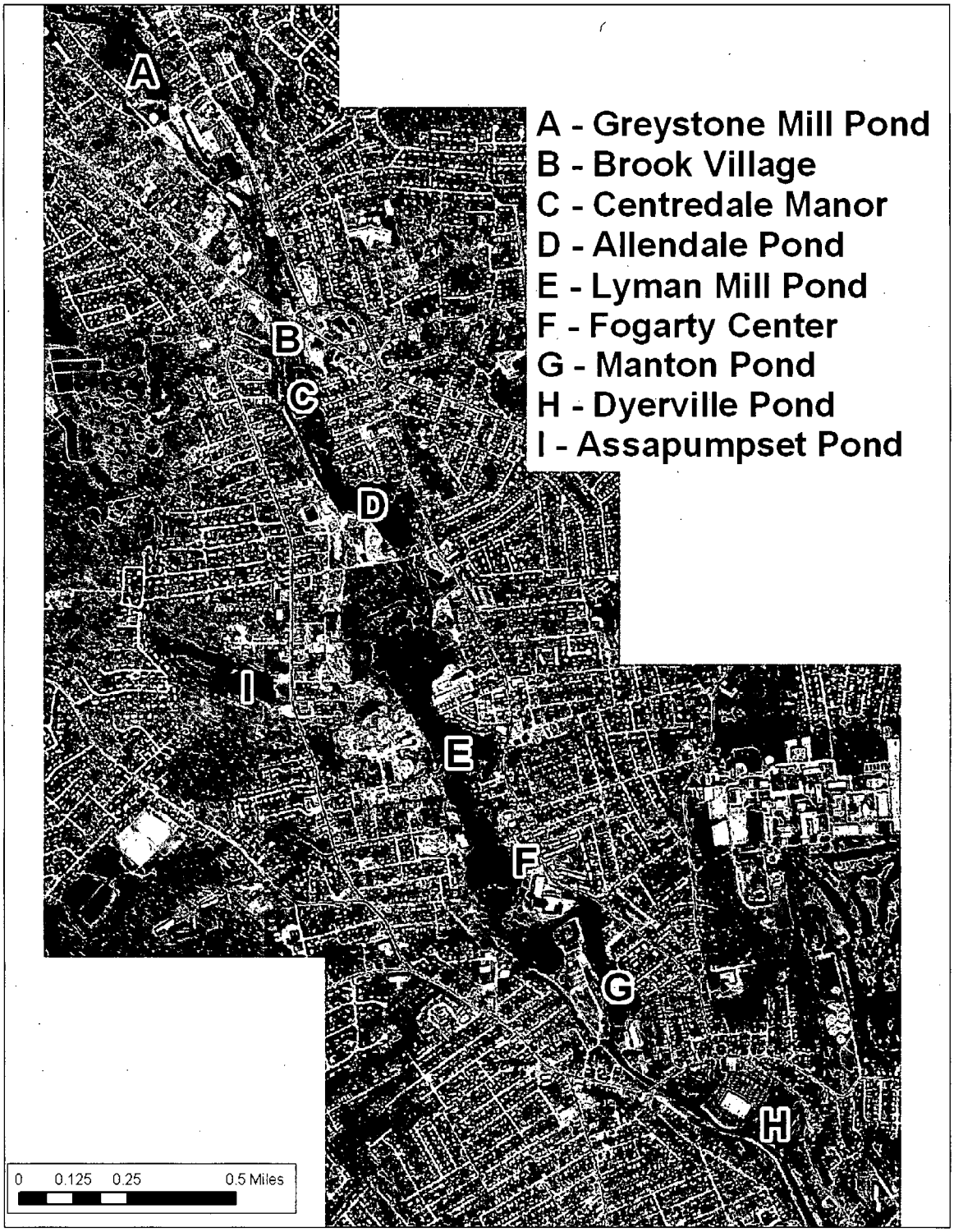
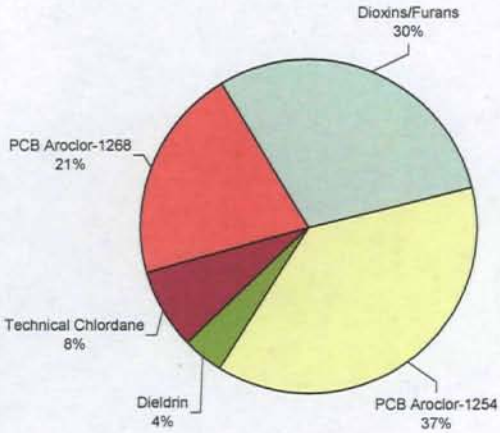
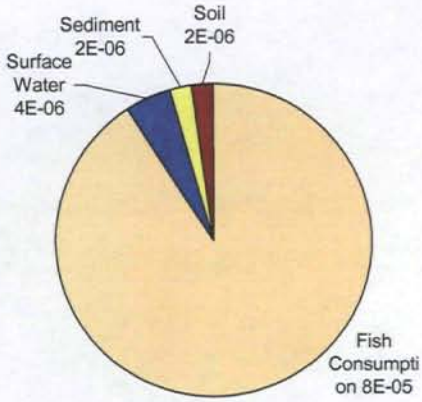


Figure 2. Site Areas

Figure 3. Risk Distribution for Greystone Mill Pond Recreational Angler

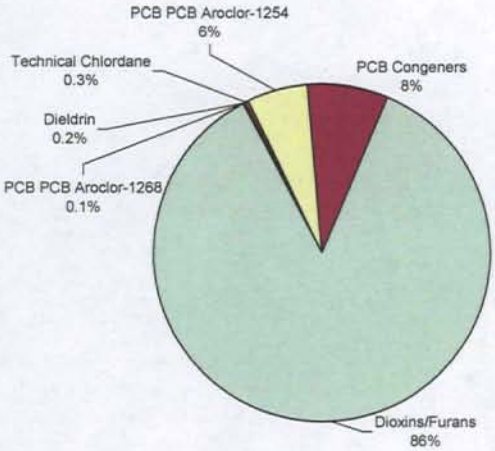


Note: Chart only includes COCs

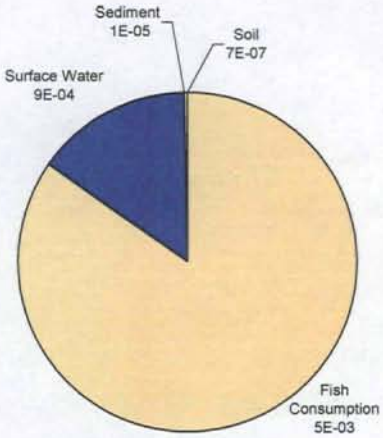


TOTAL RISK = 9E-05
-Fish Consumption Risk = 8E-05
-PCBs = 5E-05
-Dioxins/Furans = 2E-05

Figure 4. Risk Distribution for Allendale Pond Recreational Angler

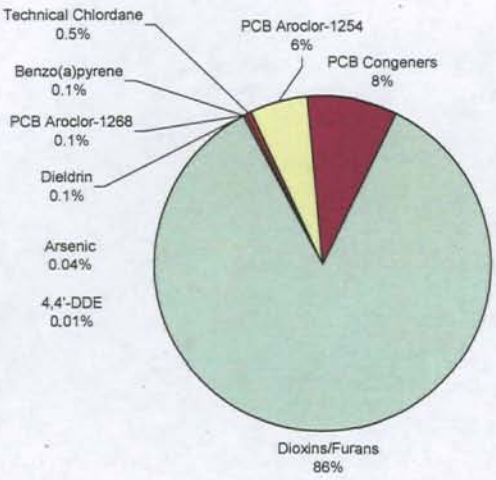


Note: Chart only includes COCs

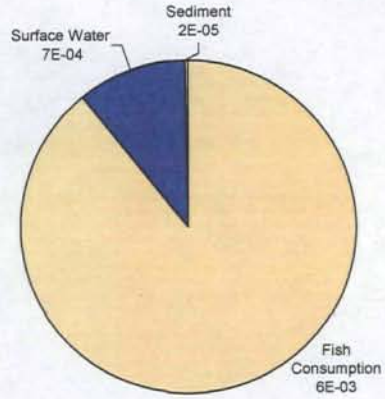


TOTAL RISK = 6E-03
-Fish Consumption Risk = 5E-03
-Dioxins/Furans = 4E-03
-PCBs = 1E-03

Figure 5. Risk Distribution for Lyman Mill Pond Recreational Angler

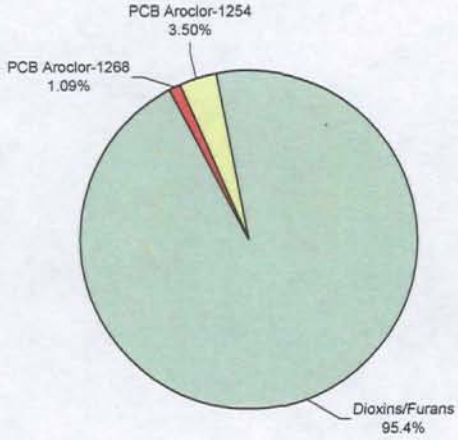


Note: Chart only includes COCs

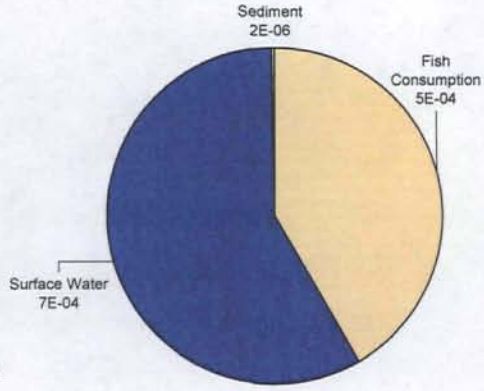


TOTAL RISK = 7E-03
-Fish Consumption Risk = 6E-03
-Dioxins/Furans = 5E-03
-PCBs = 1E-03

Figure 6. Risk Distribution for Manton Pond Recreational Angler

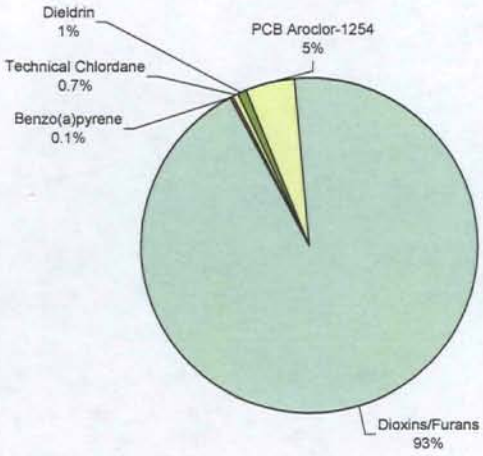


Note: Chart only includes COCs

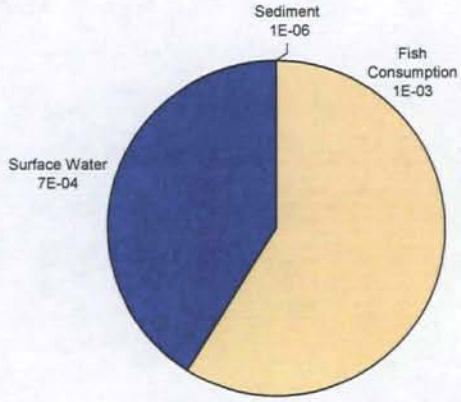


TOTAL RISK = 1E-03
-Surface Water Risk = 7E-04
-Dioxins/Furans = 7E-04
-Fish Consumption Risk = 5E-04
-Dioxins/Furans = 4E-04

Figure 7. Risk Distribution for Dyerville Pond Recreational Angler

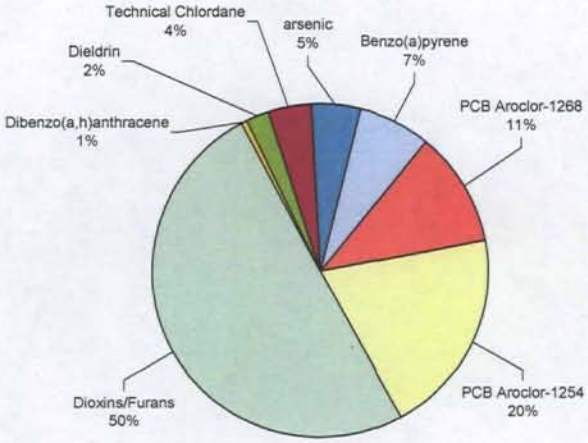


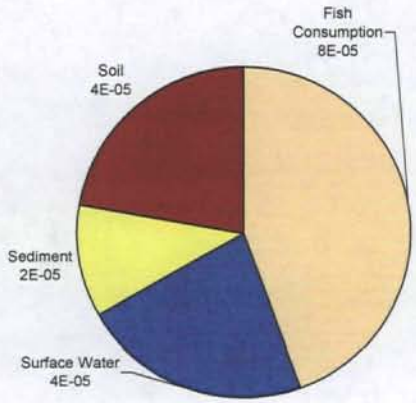
Note: Chart only includes COCs



TOTAL RISK = 2E-03
-Fish Consumption Risk = 1E-03
-Dioxins/Furans = 1E-03
-Surface Water Risk = 7E-04
-Dioxins/Furans = 7E-04

Figure 8. Risk Distribution for Greystone Mill Pond Resident

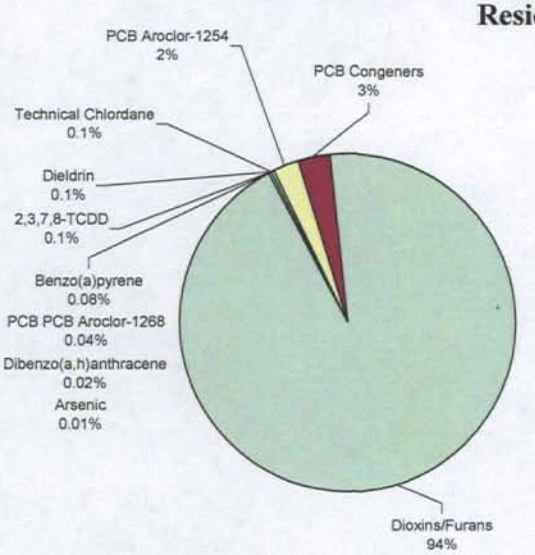


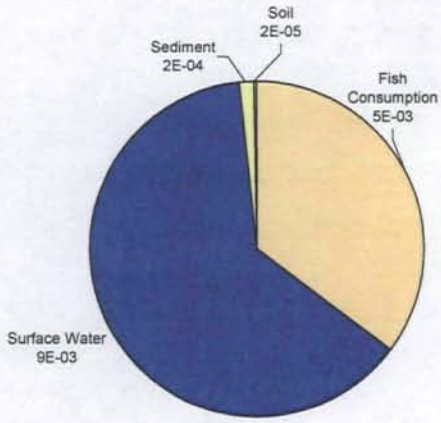


Note: Chart only includes COCs

TOTAL RISK = 2E-04
-Fish Consumption Risk = 8E-05
-Dioxins/Furans = 2E-05
-Aroclor-1254 = 3E-05

Figure 9. Risk Distribution for Allendale Pond Resident

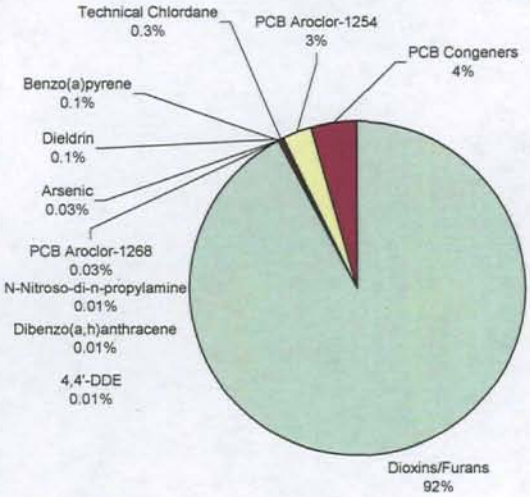




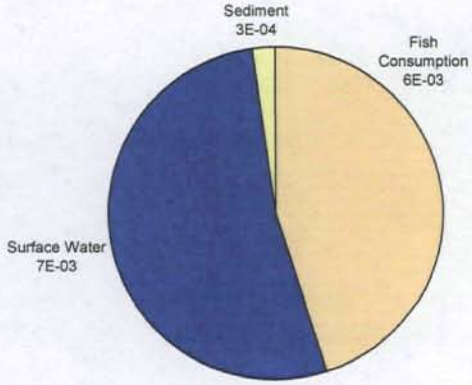
Note: Chart only includes COCs

TOTAL RISK = 1E-02
-Surface Water Risk = 9E-03
-Dioxins/Furans = 9E-03

Figure 10. Risk Distribution for Lyman Mill Pond Resident

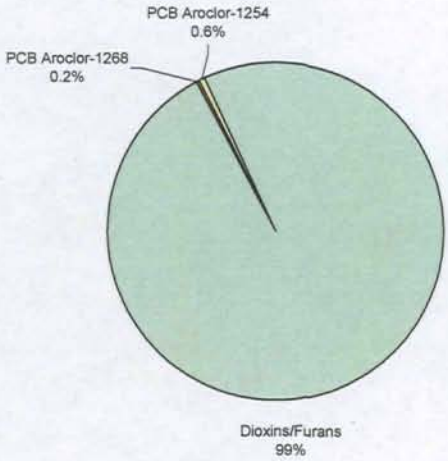


Note: Chart only includes COCs

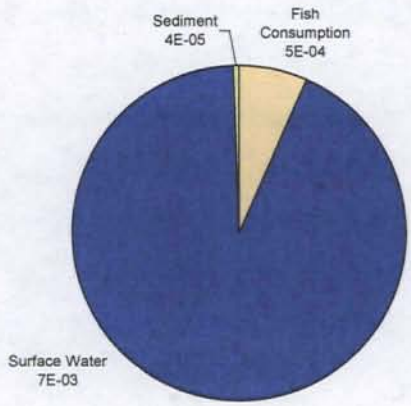


TOTAL RISK = 1E-02
-Surface Water Risk = 7E-03
-Dioxins/Furans = 7E-03
-Fish Consumption Risk = 6E-03
-Dioxins/Furans = 5e-03
-PCBs = 1E-03

Figure 11. Risk Distribution for Manton Pond Resident

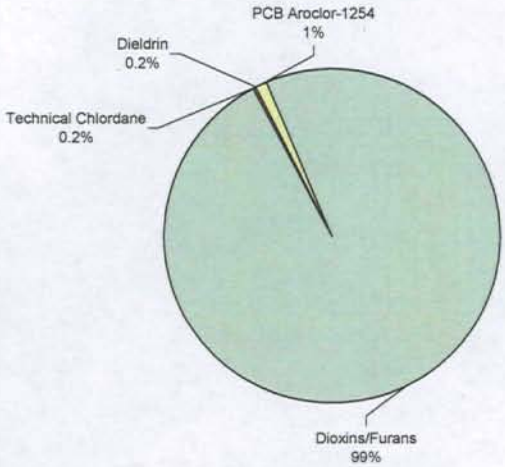


Note: Chart only includes COCs

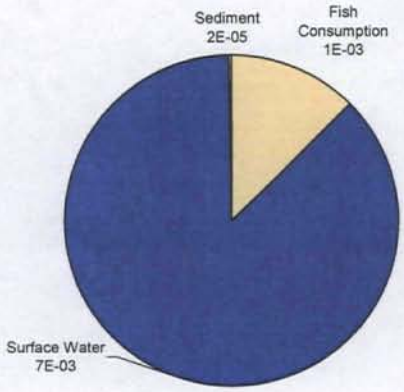


TOTAL RISK = 7E-03
-Surface Water Risk = 7E-03
-Dioxins/Furans = 7E-03

Figure 12. Risk Distribution for Dyerville Pond Resident



Note: Chart only includes COCs



TOTAL RISK = 8E-03
-Surface Water Risk = 7E-03
-Dioxins/Furans = 7E-03

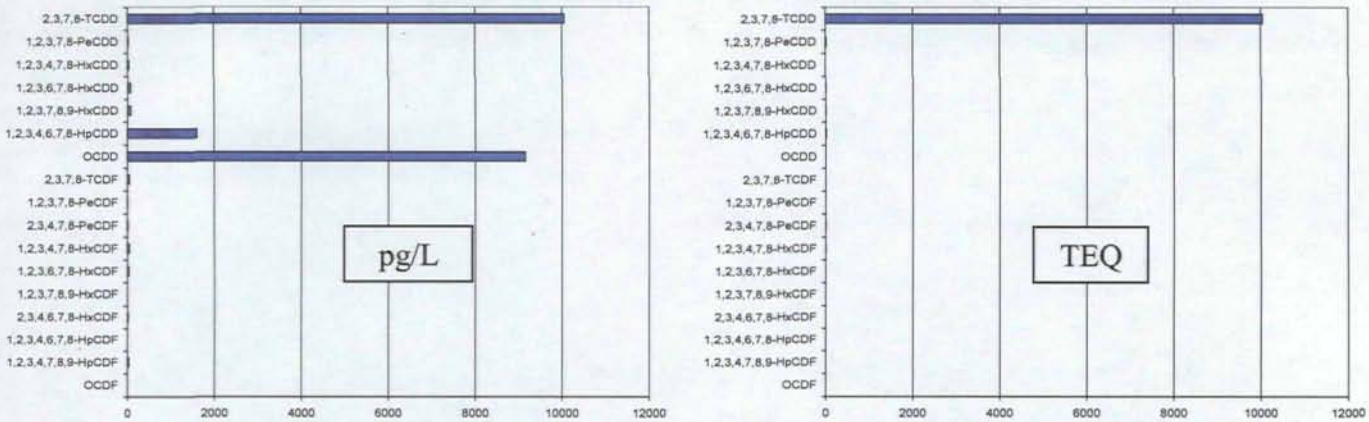


Figure 13. Surface Water Dioxins/Furan Concentrations (pg/L) and TEQ[8]

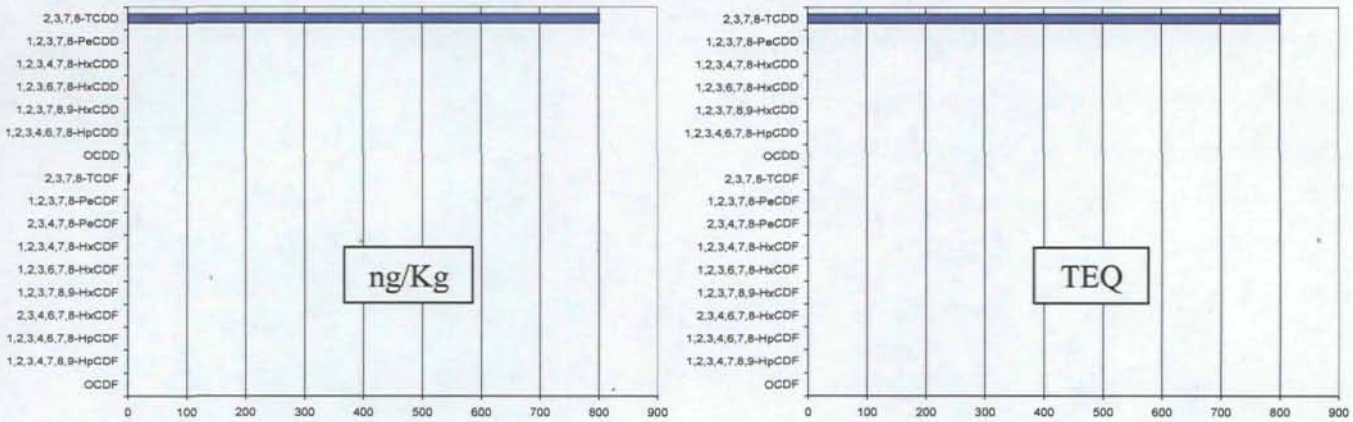


Figure 14. Sediment Dioxins/Furan Concentrations (ng/Kg) and TEQ[1]

Figure 15. White Sucker Tissue Dioxins/Furan Concentrations (ng/kg wet) and TEQ[1]

---

[8] Source: Centredale Master Database File created by Battelle dated July 22, 2004. Surface Water Sample # CMW-SW-2019-01; Sediment Sample # SD-23; White Sucker Sample # APC-WS-4003-0000-01-W

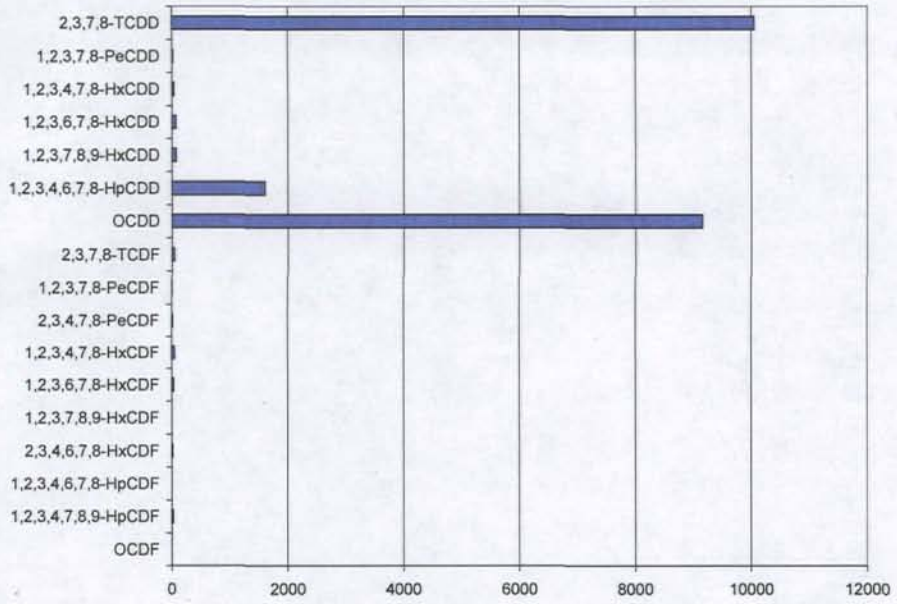


Figure 16. Dioxin Congener Profile - Sediment Concentrations (ng/Kg)[9]

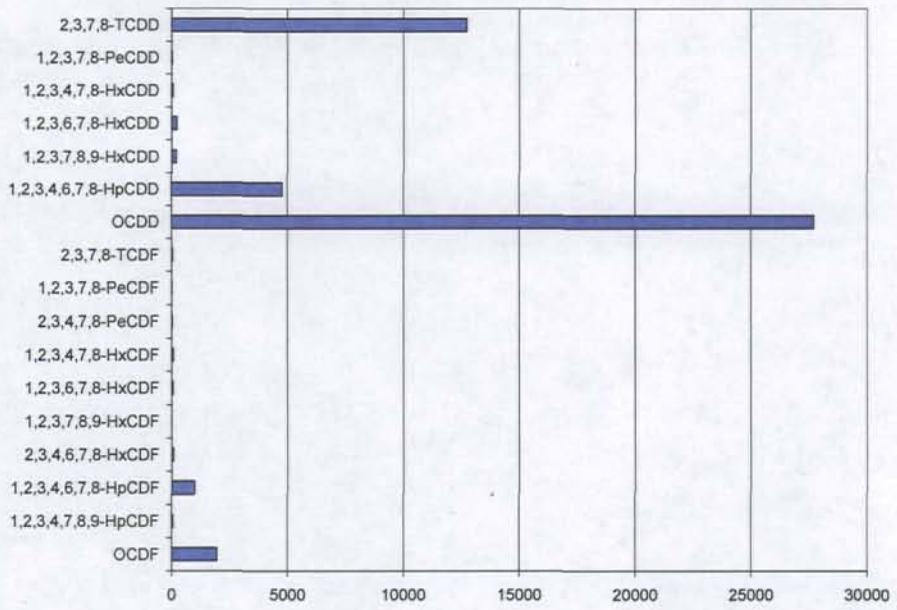


Figure 17. Dioxin Congener Profile - Surface Water (pg/L)[1]

[9] Source: Centredale Master Database File created by Battelle dated July 22, 2004. Sediment Sample # SD-23; Surface Water Sample # CMW-SW-2019-01

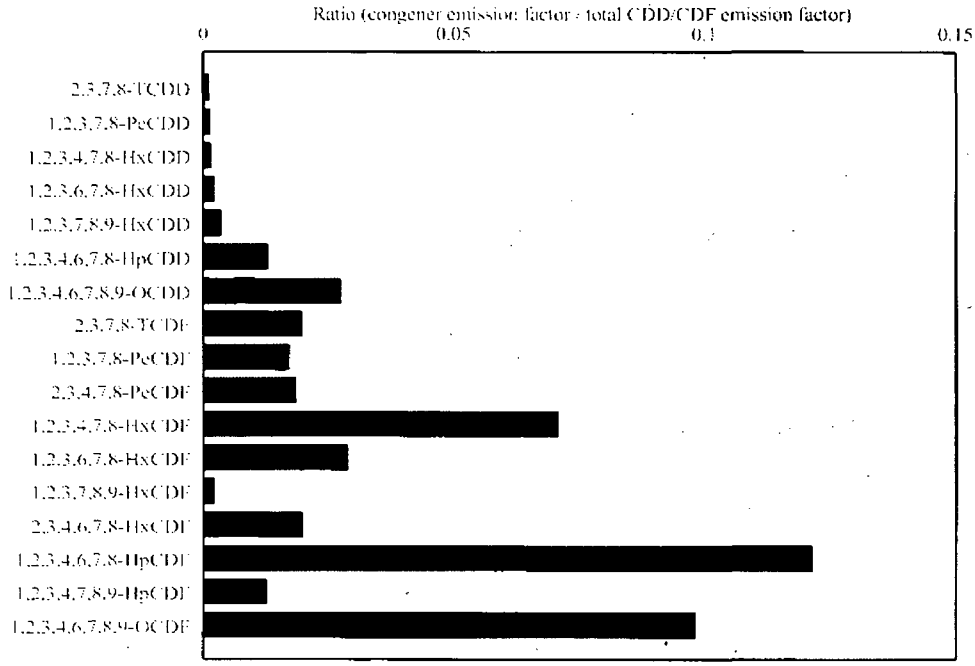


Figure 18. Congener Profile for Air Emissions from Hazardous Waste Incinerators[10]

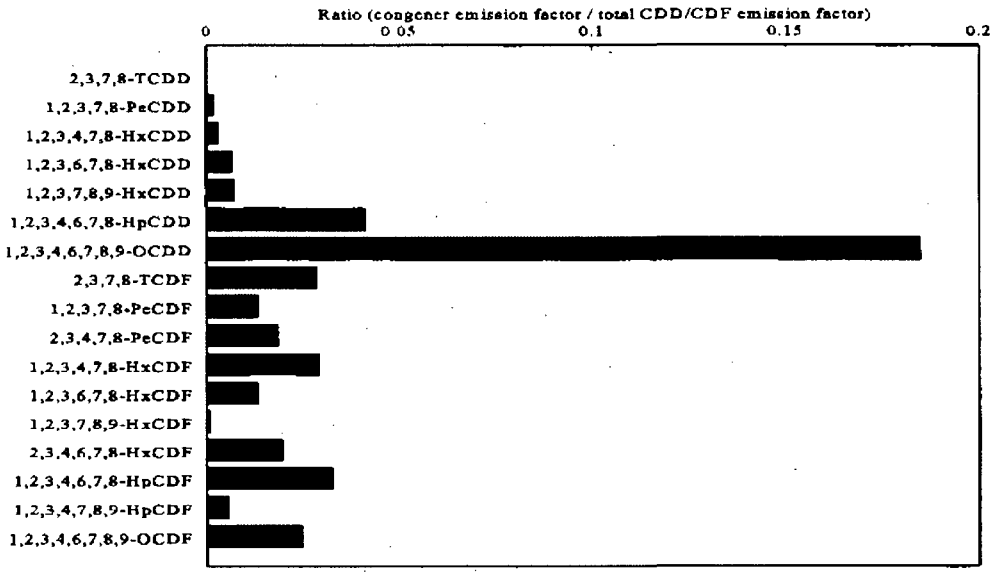


Figure 19. Congener Profile for Air Emissions from Boilers and Industrial Furnaces Burinig Hazardous Waste[1]

---

[10] Source: *Draft Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds*, EPA Office of Research and Development, September 2000, Draft Final Report

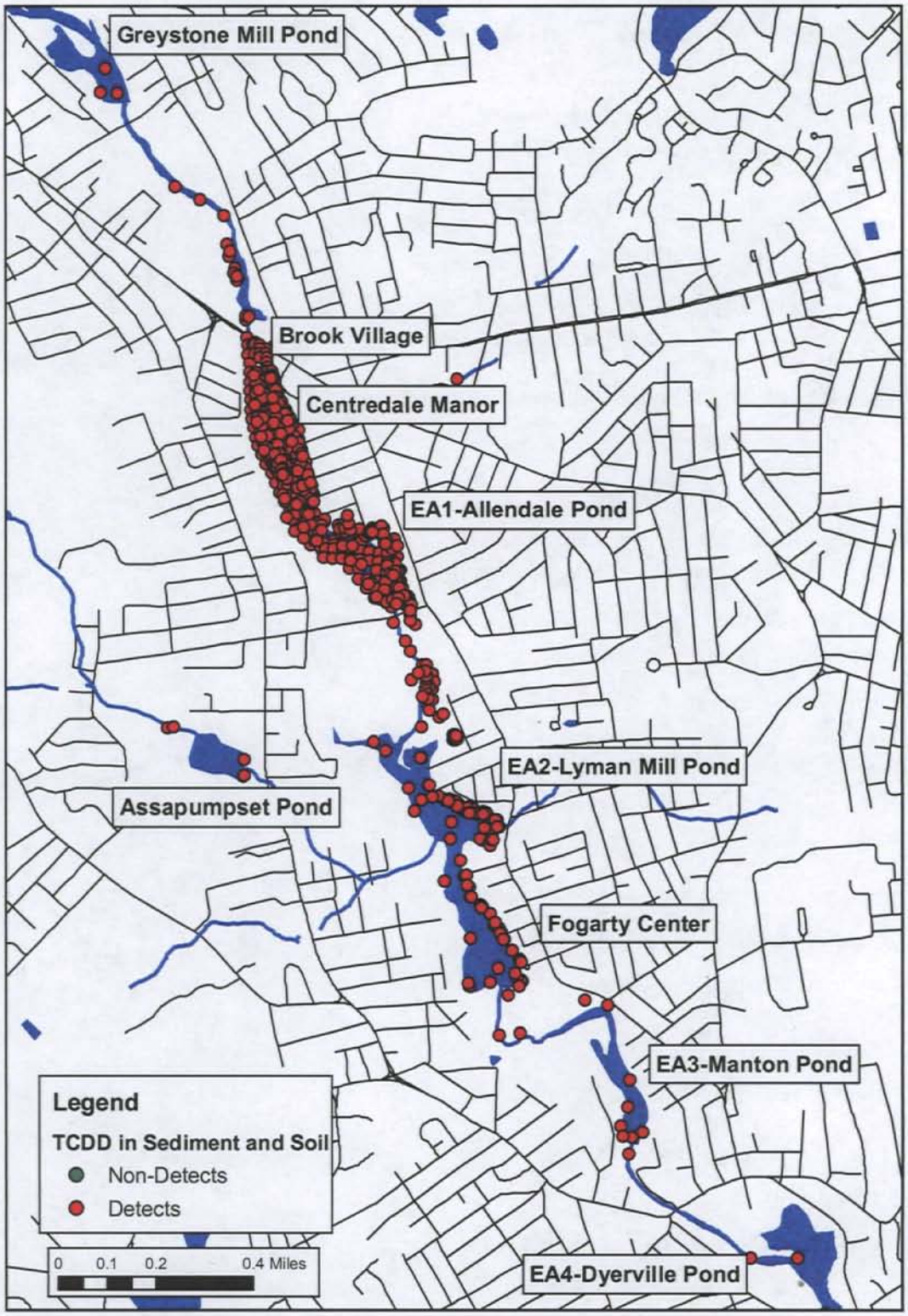


Figure 20. Pattern of TCDD Detections

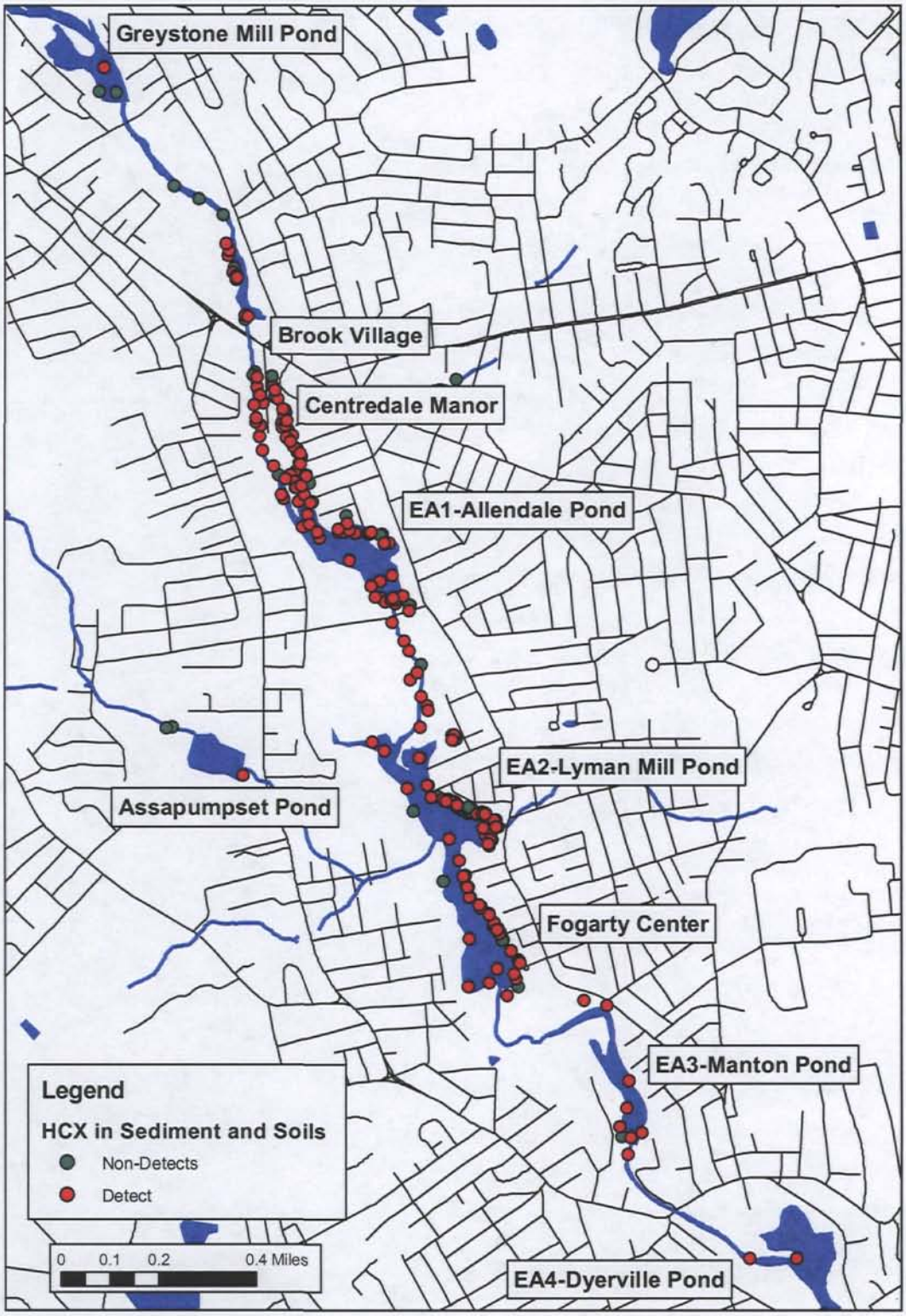


Figure 21. Pattern of HCX Detections