Comments
on
Housatonic River Corrective Measures Study
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prepared by
ESC, LLC
Dr. Peter L. deFur
Henrico, VA
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On behalf of
Housatonic River Initiative

Document Summary

This Corrective Measures Study (CMS) is a revised and changed draft of the CMS submitted by GE to EPA in 2008. The CMS presents options and alternatives for cleaning up the PCBs and other chemical contaminants in the Housatonic River that were released by GE from the Pittsfield plant for more than 70 years. The current CMS was prepared with input from EPA in terms of assumptions and conditions.

The CMS presents cleanup options in three categories: 1) the river sediments; 2) the floodplain/riverbank areas and; 3) treatment or disposal of sediments removed from the river. These various specific options are combined to give a series of complete options addressing contamination in the river and floodplain and addressing how to handle the contaminated sediments. The combinations are limited to fit a pre-determined range of options that range from no removal (no action) to partial removal and finally, removal of a larger volume of contamination. Removing less sediment from the river is combined with removing less soil in the floodplain and applying comparable criteria to focus on the more contaminated locations for removal. One option provides for minimizing the physical disturbance of the river, riverbed and riverbank with the idea of reducing the physical disturbance to the river during remediation.

The CMS presents, but does not select, several possible ways to handle the contaminated sediments and soils after removal. The CMS discusses some options that were not selected for further consideration:

- A confined disposal facility (CDF) in water in an unspecified location
- Sediment/soil washing of dredged sediment
- Thermal desorption of dredged sediment

The CMS does not consider various other options for treating contaminated soils/sediments, including:

- In place treatment of PCB

- In place isolation, including activated carbon
- Other processes for removing PCBs from contaminated sediments in addition to Biogenesis soil washing

Generally, the alternatives rely to a great extent on sediment removal “in the wet” and use more mechanical dredging than hydraulic dredging. Hydraulic dredging tends to be generally less damaging when conducted properly. More hydraulic dredging and use of smaller pieces of equipment (e.g. hand-held) will reduce the footprint of the remediation activities. The disadvantage to GE and the remediation in using smaller pieces of equipment is that the process tends to take longer and progress slower because of a lower rate of sediment removal.

The CMS is supposed to satisfy the legal and regulatory conditions set out in CERCLA that are included in the Consent Decree. One of these requirements is to meet applicable or relevant and appropriate requirements (ARARs). One such ARAR is the water quality standard for PCBs. The CMS observes that this ARAR will not be met under several options and that EPA will need to grant a waiver based on the impracticality of meeting the water quality standard for PCBs.

In the end, GE presents an option that is supposed to satisfy EPA requirements but which GE does not agree to implement because of their continued denial of scientific facts about PCB toxicity and EPA requirements and assumptions.

General Comments

- The Executive Summary sets the tone for the entire document by presenting arguments about why GE should not undertake any further actions to actively clean up the PCBs and other chemicals that GE has released for more than 70 years into the Housatonic. The style and tone are more like that of an opinion piece rather than a technical document, and the tone is one of denial that there is a problem or that GE should clean up the river. GE continues to assert that PCB toxicity is not a problem.

- GE continues to deny the scientific evidence that PCBs cause cancer and other adverse health effects in humans, or that PCBs cause any harm to ecosystems and ecological receptors. The CMS flies in the face of scientific evidence and ignores the independent scientific research, citing instead the work of their own scientists or scientists funded by GE. The international scientific community long ago recognized the dangers and toxicity of PCBs;GE’s continued denial simply undermines the credibility and scientific veracity of the CMS.

- The waiver criteria and process need to be explained in the CMS much more effectively. In several places, the CMS notes that ARARs will not be achieved because it is too difficult or that, due to GE’s continued insistence that PCBs are not toxic, the PCBs do not need to be removed. If EPA allows a cleanup remedy that still presents conditions not meeting standards, and cleaning up the contamination is simply not practical, then EPA can determine that full cleanup is "impractical" and seek a waiver. This outcome is referred to as an "impracticality waiver" and needs thorough coverage in the CMS.

- The CMS does nothing to address PCB levels in the Connecticut portion of the river. The CMS simply indicates that time will heal this contamination wound by covering or washing the PCBs. The formal term for not taking any active measurements is "natural recovery" and if the plan calls for taking samples to measure improvements, then the process is called "monitored natural recovery." As proof that MNR is an effective remediation technique, the CMS points to decreases in PCB levels in Connecticut fish.

- The CMS presents the cleanup options and information on the general framework for a cleanup plan as set out in CERCLA.

- The CMS seems to be written as a document intended to support a legal challenge by GE to any EPA decision for further cleanup action.

- Section 2 contains a great deal of language about why GE does not have to comply with EPA directives. Section 2 also states GE's position that the river does not need to have PCBs removed. GE ignores or discounts PCB toxicology and the growing literature on the impacts of PCB contaminated sites on the surrounding human and ecological populations.

- The selection of alternatives is based on a series of criteria that were approved by EPA. One of these criteria is to meet IMPGs for sediment. EPA agreed with GE that there is no need to set or achieve IMPGs for either surface water or air. The problem with this decision and approach is that the air pathway will not be addressed nor included as a quantitative factor in the cleanup.

- The air pathway has been discounted for this site and other PCB contaminated sites, in spite of evidence that indicate the significance of the atmospheric transport pathway. The section on PCB toxicity and health below provides literature citations that indicate proximity to PCB contaminated sites is associated with increased health effects and greater risk for several diseases.

- The CMS does make some attempt to discuss what may occur during flooding by including extreme weather events from the past (section 3.2.2.1, March 1936 and August 1990). This effort, seemingly included at EPA's direction, begins to account for flood events, but does not completely address the likely consequences of sustained higher rainfall, flood, and drought sequences that are possible or probable in the future.

- The CMS states that PCB levels are decreasing as proof that MNR will work to clean the river. In the same explanation, GE indicates that inputs of PCBs to the river are being reduced by actions taken in Pittsfield. This explanation is neither satisfactory nor entirely correct. Unless GE intends to completely eliminate all PCB inputs to the Housatonic River from storm water, leaching, and site sources (which should be done), then the Pittsfield plant will continue PCB inputs to the river for the foreseeable future.

- Other PCB remediation techniques should be considered. Other, more applicable techniques, may include activated carbon treatment and streambank restoration efforts. Activated carbon and streambank restoration are discussed later.

PCB toxicity

GE erroneously sites reductions in PCB levels in the water column as evidence of successful remediation; it is well understood, though, that measuring PCB levels in sediment is a more appropriate measure. PCBs do not remain within the water column but adhere to sediments, indicating that a water-based measurement of PCBs would be misleading to the actual volume of PCBs present in the system. Also, the modeling of PCB levels only runs 52 years into the future, or just 30 years after remediation. These estimates are short-sighted and do little to promote the long-term health of the Housatonic River.

Scientists have continued to research the detrimental toxic effects caused by PCBs. In the last two years, further research has been published supporting that PCBs are hazardous to plants, fish, animals, and humans. PCBs are known to bioaccumulate within the food chain and effect gene expression, cytochrome P450 and P-glycoprotein expressions, proteins, kidney tubules, and cause embryotoxicity, cell death, oxidative stress, and more detrimental health effects. There is no evidence to support the notion that PCBs are safe and not a health threat to people or the environment. EPA has tested and demonstrated that PCBs are, at the very least, a probable carcinogen and need to be taken very seriously.

A literature search on PCB toxicology during the last two years provides an abundance of papers on the toxic effects of PCBs, the results of which are provided in Appendix B.

Natural Recovery and Monitored Natural Recovery

There is no evidence in the literature or in government reports that natural recovery is a highly effective, long term means of cleaning up PCBs in a fast flowing cold-water river such as the Housatonic River. In addition, the CMS has a number of problems, indicated here:

- The CMS lacks a site-specific description of the processes involved in the monitored natural recovery option. What natural processes does the monitored natural recovery option rely on? What are the “naturally occurring processes” that are referenced in the MNR definition on page 1-17? Will the option affect PCB bioavailability or toxicity or both? Overall, the MNR method is unclear and obviously not fully developed.

- Misleading language, such as, “MNR would be applied,” leads a reader to believe that MNR is an active option. MNR is a passive option and its success is conditional on a variety of factors such as sedimentation rate, contaminant type, soil type, types of microbes present, the amount and types of contaminants. In the case of PCBs, MNR means exclusively the sedimentation rate exceeds the rate of erosion and scour.

- MNR relies on natural capping and “transformation or removal of contaminants via contaminant weathering” (Brenner 2004). The success of natural capping, which occurs when clean sediment deposits on top of contaminated sediments, depends on the rate of clean sediment deposition in relation to the rate of sediment resuspension (Brenner 2004). River discharge and hydrology affect these factors and must be considered in a discussion of MNR as a viable option. MNR is not effective in all instances.

- The CMS states, on page 6-23, that the combination of MNR and source controls “has been demonstrated to be effective in reducing contaminant levels in sediment and biota” (GE 2010). Similar past projects are listed on the same page. The actual results of these projects are not included or cited; it is the Record of Decision, however, that is cited for these projects. It is not possible to comment on the effectiveness of a project by consulting the ROD only. A study conducted at one of the listed sites, the Sangamo-Weston/ Twelvemile Creek/Lake Hartwell Superfund Site in Pickens, SC is in non-flowing warm waters, a lake, and has no similarities with the Housatonic. At Sangamo, ten years after remediation implementation, the remedial alternative based on MNR and source control did not bring PCB concentrations in largemouth bass and hybrid bass below the FDA’s fish tolerance level of 2.0mg/kg (Brenner 2004). These findings do not support the statement on page 6-23, claiming that MNR effectively reduces contaminant levels in biota.

- Commencement Bay Nearshore/Tideflats (Sitcum Waterway), WA is another site listed in the CMS as a “similar” site (GE 2010, p. 6-23). The site, however, is not actually included in the literature cited. Additionally, the literature cited is a review of Enhanced Monitored Natural Recovery (EMNR) case studies (Merrit 2009). EMNR includes additional components, such as thin-layer capping. The terms EMNR and MNR are not interchangeable.

- The CMS does note, however, that “long-term monitoring data” does not exist for these sites, and so it is not possible to make claims on the long-term effectiveness of MNR based remediation alternatives (GE 2010, p. 6-23).

- MNR will be accompanied by a set of institutional controls (GE 2010, p. 6-122). MNR may be a slow process taking "decades" (EPA 2001). How far into the future is it reasonable to expect institutional controls to remain effective?

- PCB movement in soil may occur through mechanisms of advective transport and benthic mixing. These processes may retard and/or further complicate the natural capping process on which MNR's effectiveness relies (Brenner 2004).

Other PCB Remediation Techniques
Activated Carbon

Contaminant sequestration using activated carbon amendment has been proposed as an alternative or add-on to dredging at sites contaminated with polychlorinated biphenyls (PCBs). Recently, capping materials with high chemical retention capacity have come into focus, such as active carbon and zeolite minerals, a practice commonly termed ''capping with active barriers." The supposed effect of such active materials is that adsorption of contaminants to the active material will significantly reduce the contaminant transport from the capped sediment.

Activated carbon can reduce the bioavailability of hydrophobic organics such as PCBs and heavy metals present in sediments. To test the efficacy of activated carbon amendment to reduce the bioavailability of polychlorinated biphenyls (PCBs), clams (*Macoma balthica*) were exposed to different levels of field-contaminated sediment (McLeod 2007). Clams exposed to the sediment at various doses of activated carbon resulted in significant differences between their tissue PCB concentrations (Figure 1). Efficacy of activated carbon treatment was found to increase with both increasing carbon dose and decreasing carbon particle size. Average reductions in bioaccumulation of 22%, 64% and 84% relative to untreated Hunters Point sediment were observed for carbon amendments of 0.34%, 1.7% and 3.4%, respectively. Average bioaccumulation reductions of 41%, 73% and 89% were observed for amendments (dose = 1.7% dry wt) with carbon particles of 180 to 250 µm, 75 to 180 µm, and 25 to 75 µm, respectively, in diameter. The present results demonstrate that adding activated carbon to sediment from Hunters Point can reduce PCB uptake in *M. balthica* by almost one order of magnitude. Furthermore, the biodynamic modeling exercise strongly suggests that this reduction is accomplished through the mass transfer of PCBs from native sediment particles to the activated carbon (McLeod 2007).

McLeod et al. (2008) compared activated carbon capping with aqueous equilibrium PCB concentration for untreated Grasse River sediment (1.03 ± 0.08 μg/L) near Massena, NY. Total PCB concentrations in pore-water, semi-permeable membrane device (SPMD), and clams versus carbon dose in Grasse River sediment are shown in Figure 2. The general trend of decreasing concentration with carbon dose observed in physicochemical tests with Grasse River sediment are similar to those reported for Hunters Point (Zimmerman et al., 2004; Zimmerman et al., 2005) and Lake Hartwell (Werner et al., 2005). Average percent reductions in aqueous equilibrium PCB concentration were 82%, 94%, and 97% for carbon dry weight doses of 0.7, 1.3, and 2.5

respectively. PCB uptake into semi-permeable membrane devices (SPMDs) from the untreated Grasse River sediment was 476 ± 61 μg/g. Average percent reductions in SPMD uptake were 54%, 83%, and 92% for carbon dry weight doses of 0.7, 1.3, and 2.5%, respectively. Consistent with results from previous studies (Zimmerman et al. 2004; Zimmerman et al. 2005; Werner et al. 2005), treatment efficacy was higher for lower-chlorinated congeners (McLeod et al.. .2007). Average percent reductions in clam tissue PCBs were 67, 86, and 95% for activated carbon doses of 0.7, 1.3, and 2.5% (dry wt), respectively (McLeod et al. 2008).

The cost of activated is relatively cheap. 5% AC by dry wt in the top 4" equals six lb/sq. yd or 30,000 lb/acre. This amount is equivalent to 2 mm sedimentation. The material cost for AC is currently at $1/lb. Therefore one acre of AC will cost $30,000. The application cost will depend on the method utilized. Activated carbon is plentiful and a 100 acre site requiring a consumption of 3 M lb of AC is less than one percent of the U.S. annual production (Ghosh 2010). Also use of activated biochars from agriculture residue can provide additional opportunities for carbon sequestration. Additional application of small increments over multiple years to incorporate into new annually deposited sediments will help ensure further efficiency for long term remediation. A comparison of remedial alternatives and cost estimates is presented in the 2008 Final FS report for Parcel F at Hunters Point Navy Shipyard (Table 1). Seven remedial alternatives were compared (Figure 3) and range from $2-30 million (ESTCP, 2008). The use of activated carbon reduces the amount of offsite disposal. Approximately 57,850 cubic yards would be treated, requiring approximately 1,610,000 lb of activated carbon. The cost of activated carbon is $1.04/lb, which is based on the quote for Calgon Carbsorb 50x200 for the Grasse River, NY, study (ESTCP, 2008).

Overall, activated carbon appears to be a high-quality remedial alternative that is also cost effective. The use of activated carbon has shown to significantly reduce the bioavailability of PCBs, which is one of the primary concerns of PCBs. Dredging followed by backfilling with activated carbon mixed in with clean sediment would a most effective strategy because it would remove the sediment with the highest concentrations as well as adsorb any PCBs not removed during dredging.

984 *Environ. Toxicol. Chem.* 26, 2007 P.B. McLeod et al.

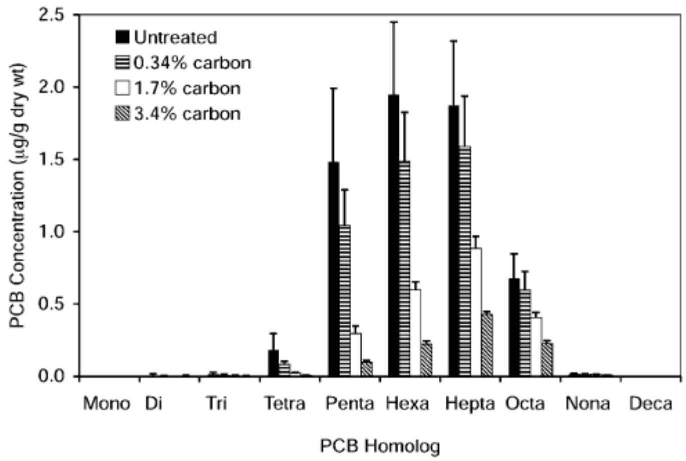

Figure 1. Clam tissue PCB homologue concentrations from clams exposed to Hunters Point (San Francisco Bay, CA) sediment amended with varying doses of activated carbon. Error bars represent one standard deviation (n=3-5 samples with 3-4 clams/sample). (McLeod 2007)

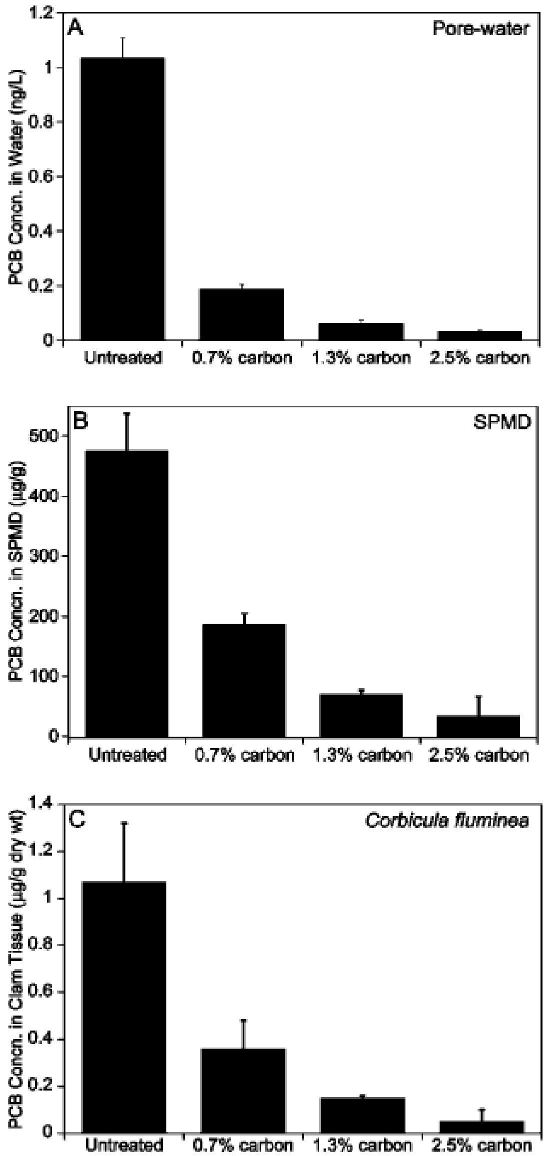

Figure 2. PCB concentrations in pore-water (A), SPMDs (B), and clams (C) in untreated and amended Grass River sediment. Error bars represent one standard deviation (n=3-5) (McLeod et al. 2008).

Cost Element	Data Tracked during the Demonstration	Costs	
Treatability study	• Detailed assessment required • Personnel required and associated labor • Materials • Analytical laboratory costs	Lab technician, 80 h	$2000
		Materials	$3000
		Analytical laboratory	$7200
Baseline characterization	• For 20 monitoring locations • Detailed field/laboratory assessments required • Field assessment costs • Analytical laboratory costs • Personnel required and associated labor • Materials	Field technician, 5*20 h Lab technician, 3*160 h Materials	$2500 $12,000 $18,000
		Analytical laboratory	$26,400
Site preparation	• No cost tracking	NA	
Activated carbon amendment	• For 700 ft^2 treatment by one of mixing options • Activated carbon • Mobilization/demobilization of AEI Aquamog • Mobilization/demobilization of CEI injector system • Personnel required and associated labor	Field technician, 5*20 h Materials	$2500 $3000
		Activated carbon (TOG), 350 lb / 100 ft^2	$ 7000
		AEI Aquamog Labor & rental, 2 days	$10,000
		CEI Injector Labor & rental, 2 days	$10,000
Operation and maintenance costs (periodic monitoring)	• For 20 monitoring locations • Detailed field/laboratory assessments required • Field assessment costs • Analytical laboratory costs • Personnel required and associated labor • Materials	Field technician, 5*20 h Lab technician, 3*320 h	$2500 $24,000
		Materials	$18,000
		Analytical laboratory	$26,400
		Reporting (per year)	$10,000
Decontamination and residual waste management	• Standard practice, no cost tracking	NA	
Public education program	• No cost tracking	NA	
Operation and maintenance costs	• No unique requirements recorded	NA	
Long-term monitoring	• No cost tracking	NA	

Table 1. Cost model for in situ stabilization by activated carbon mixing (ESTCP 2008).

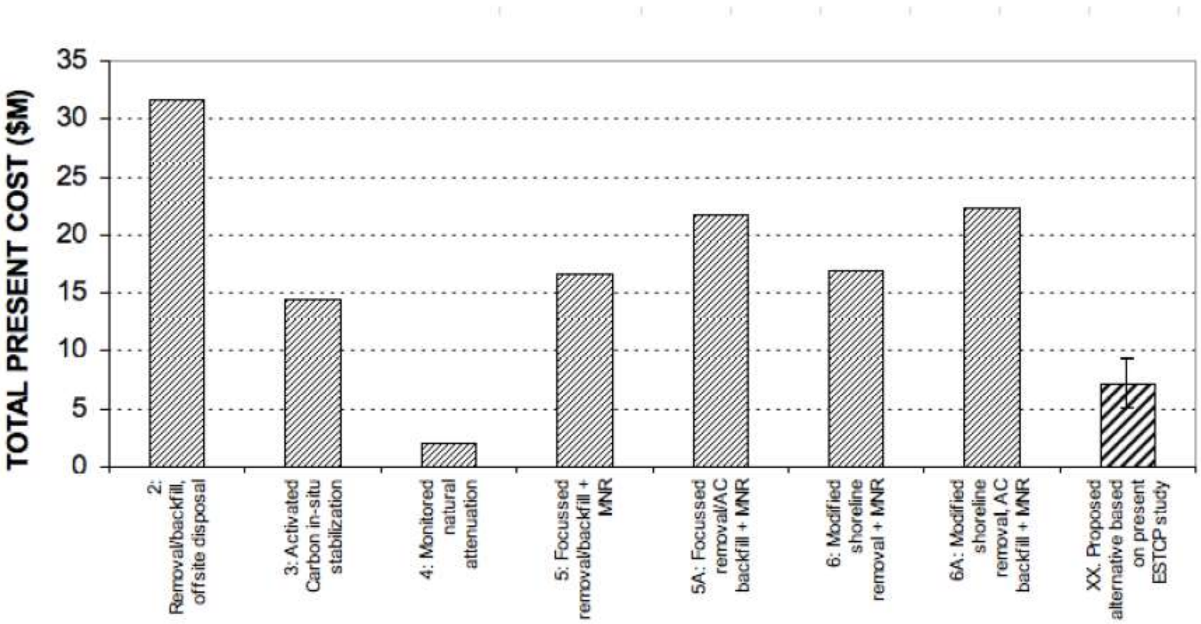

Figure 3. Present value cost comparison of different remedial alternatives for Hunters Point Navy Shipyard South Basin area (Area IX/X) (ESTCP 2008).

Stream Bank Restoration

The CMS completely discounts the restoration efforts used in the U.S. to remediate a variety of conditions, including PCB contamination.

Stream bank erosion increases the movement and deposition of sediments and, therefore, is a large source of pollution in many riverine environments (Dorava 1999). Some stream bank erosion naturally occurs as water flows. Human development and activities, however, have greatly increased natural erosion processes and necessitated the implementation of stream bank erosion controls (Zaimes 2006). Because some stream bank erosion is naturally occurring, the erosion can never be completely eliminated. There are, though, a variety of methods that may be used to control and restore stream banks. A variety of factors, such as vegetation cover, topography, bank material, weather cycles, river morphology, channel stage, and watershed area should be considered during stream bank restoration efforts (Zaimes 2006).

Housatonic River channel stabilization could reduce pollutant loading into urban tributary streams and also balance the impact on the ecological system. Many traditional hard stream bank erosion control techniques rely on large quantities of riprap and/or concrete and steel structures (Dorava 1999). Although hard methods have proven effective in mitigating erosion, the unnatural materials used under these methods do not provide suitable habitats for fish and other riverine species (Dorava 1999). Other bank stabilization/restoration practices provide more natural solutions for combating stream bank erosion and sedimentation than harder methods.

Different bioengineered stabilization treatments may be used depending on the amount of erosion occurring in a particular section of a bank. Planting vegetation along the bank will usually suffice in areas that need less stabilization. Bendway Weirs, stream barbs, rock riffles, and stone toe protection are methods commonly used in areas that require more bank stabilization to reduce erosion. Bendway Weirs are low rock structures placed in a stream to direct water away from the bank to lessen stream impact on the bank (UIUC 2010). This method has been highly effective and widely used by the US Army Corps of Engineers (USACE 2001). Stream barbs are similar to Bendway Weirs, but stream barbs are placed at a more severe upstream angle to more aggressively direct water away from the bank. Rock riffles reduce bank and stream bed erosion by creating a step-like structure which slows stream water velocity. Stone toe protection involves placing stones parallel to the bank. The stones provide protection to bank-stabilizing vegetation growth. Several treatment methods are commonly combined to provide the most effective treatment for stabilization (UIUC 2010).

Bioengineered methods are often less expensive than hard methods. Riprap and concrete or steel structures generally cost between 50 to 300 dollars per foot, while bioengineered bank stabilization methods cost approximately 15 to 25 dollars per linear foot (UIUC 2010). Generally, bioengineered methods also provide a more natural functioning ecosystem and a more aesthetically-pleasing landscape (Sneeringer 2001). Vegetation, which is almost always planted streamside as part of bioengineered erosion control treatments, provides shade, rebuilds a riparian buffer, regenerates a natural habitat, and is much more visually pleasing than large concrete structures (Sneeringer 2001).

These more natural and innovative methods are being implemented with great success across the country and have been embraced by the Army Corps (USACE 2010). Cost-effective bioengineered methods should be considered to reduce erosion and to return areas to more natural habitats in and around the Housatonic River.

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Appendix A:
PCB Exposures in the Vicinity of PCB contaminated sites

Summary

Epidemiologic studies suggest an association between environmental exposure to polychlorinated biphenyl (PCBs) and higher tissue levels of PCBs in residents of the surrounding communities. PCBs are toxic chemicals classified as endocrine disruptors and known to activate the aryl hydrocarbon receptor (Ahr). The major routes of exposure to these compounds are ingestion of fish (especially sport fish caught in polluted lakes or rivers), meat and dairy products (Dellinger et al. 1996, Falk et al. 1999), and inhalation of contaminated air near hazardous waste sites (DeCaprio et al. 2005, Kouznetsova et al. 2007). Research has focused on quantifying the release, distribution and subsequent human uptake of PCBs around contaminated areas in Lake Michigan, Massachusetts, Russia, Canada, Spain, and elsewhere. Population blood samples have been measured in many areas to determine whether or not correlations exist between PCB exposures and PCB levels found in the blood or tissues. Scientific evidence indicates fetuses are most sensitive to exposure and an increased uterine exposurt to PCBs may reduce fetal and postnatal growth, psychoneurological development, and cognitive ability (Jacobson and Jacobson 1996).

Introduction

Polychlorinated biphenyls (PCBs) are a formerly used industrial chemical that is widely detected in people, wildlife, and the environment due to its persistent and bioaccumulating tendencies. It is estimated that more than 1.5 million tons of PCBs have been manufactured worldwide (Faroon et al. 2003, Pieper and Seeger 2008), where a significant amount has been released into the environment and accumulated in soils and sediments (Nogales et al. 1999, Sericano et al. 1995, Pieper and Seeger 2008). Between 1930 and 1979, over 600 million kilograms of PCBs were used in North America alone (Laukers 1986), 15% of which entered the environment through legal and illegal use and disposal (Cookson Jr. 1995) and accidental releases (Erickson 1997).

Polychlorinated biphenyls (PCBs) are a class of compounds which have been used since 1929 for various industrial and commercial purposes such as dielectric, heat transfer, hydraulic fluids, plasticizers and fire retardants. Although adverse health effects were first recorded in the 1930s (Drinker et al. 1937), PCBs continued to be used for decades. More recently, PCBs have been shown to cause cancer (Mayes et al. 1998) and a number of serious effects on the immune, reproductive, nervous and endocrine system (Aoki 2001, ATSDR 2000, Faroon et al. 2001, Pieper and Seeger 2008). Presently about 750 thousand tons of PCBs are still used, mainly in closed systems; approximately the same amount is present in the biosphere (Abraham et al. 2002, De et al. 2006, Vasilyeva and Strijakova 2007).

Despite longstanding prohibitions on their manufacture and use, PCBs are still pervasive in environmental media, wildlife, food, and humans. PCB production in many countries has been banned and PCB application has been drastically restricted. Presently, these PCB compounds are considered among the most hazardous pollutants in the world (Borja et al. 2005, Ross 2004, Vasilyeva and Strijakova 2007). The concern about environmental pollution with persistent organic pollutants is increasing due to the compounds' toxicity, bioaccumulation, extensive distribution, recalcitrance, and lipophilicity, or their ability to dissolve in animal fat or lipid tissue.

The largest amount of PCBs in the environment is localized in soil and in water sediments close to the places of their former production and application (Fedorov 1993, Mackova et al. 2006). Although PCBs have been linked to a number of human and ecological health problems, performing a cleanup is expensive. According to calculations performed by the US Environmental Protection Agency (US EPA 2009), elimination of 525 million tons of PCB-containing waste requires about 400 billion dollars (Vasilyeva and Strijakova 2007). The expense of such a cleanup undoubtedly influences PCB remediation efforts, although PCB toxicity persists.

Exposure of the general population to PCBs is evident from data on body accumulation of non-occupationally exposed populations. People are exposed to PCBs via four pathways: inhalation, ingestion of food or fluids, absorption through the skin and fetal/lactational exposure (CDC). Some exposures can be controlled, such as coming into contact with contaminated soil and consumption of fish, crops, dairy, and meat. Other routes of exposure, including breathing contaminated air, and in utero exposure, are more difficult to limit. It is important for people living near PCB contaminated sites to take precautionary measures to protect themselves from exposures to contaminated air, water, and soil. After accounting for controllable exposure pathways, it is imperative to determine the amount of exposure that remains. Investigations indicate that living in the vicinity of a PCB-contaminated site or known source is associated with higher tissue levels of PCBs.

Human samples such as serum/plasma, human milk, adipose tissue, and breast milk have been used as biomarkers to assess the extent of human exposure to lipophilic contaminants, such as PBDEs, PCDD/Fs, and PCBs. ATSDR (2010) says that the average adult has between 0.9 and 1.5 parts per billion (ppb) in our blood. Research indicates that more is worse, but that even the level now commonly found in the average person increases risk of disease. In the end, there is no “safe” level of PCBs. This is why it is so important to reduce PCB exposure to the greatest degree possible without excessive disruption of life style.

Community Exposures

Several investigations in recent years have examined PCB levels in communities in the immediate vicinity of PCB sources, including St. Lawrence River in New York, Ontario, and Quebec, Chapaevsk, Russia, New Bedford Harbor, Massachusetts, Korea and Catalonia, Spain. Residents of communities located near PCB contaminated sites show higher PCB levels in their tissues.

Fitzgerald et al. (2007) addressed the question of how fish consumption, occupation, and outdoor air affected serum PCB concentrations and congener patterns among 139 Native American men (Mohawks) living near three hazardous waste sites. Akwesasne is a Native American community of more than 10,000 persons located along the St. Lawrence River in New York, Ontario, and Quebec. Less than 100 feet to the west of Akwesasne is the General Motors Central Foundry Division Superfund hazardous waste site. Serum total PCBs was 4.9 ppb (wet weight), in the 139 Akwesasne men. 13% of the subjects had a concentration above 10.0 ppb, and the maximum concentration was 31.7 ppb. In comparison, the National Health and Nutrition Examination Survey (NHANES), conducted by the Center of Disease Control and Prevention (CDC), found the average PCB concentration in blood serum for the United States in 1999-2000 to be 2.7 ppb. Serum total PCB concentrations of the Akwesasne increased with age ($P<0.001$) and cumulative lifetime exposure to total PCBs from local fish consumption ($P=0.011$). Exposure estimates included fish consumption as part of total dietary intake; however, cumulative lifetime exposure to PCBs from local fish consumption was strongly correlated with intake alone ($r=0.94$), suggesting that intake was the primary factor driving the accumulation. These results indicate that there is a correlation between PCB exposure through fish consumption and elevated serum PCB levels in humans.

In another study, Burns et al. (2009) investigated predictors of serum PCB concentrations of 8 to 9 year old boys living in the vicinity of a chemical manufacturing factory in Chapaevsk, Russia. Increasing age, longer breast feeding duration, closer location to the factory, and eating locally grown foods were associated with significantly higher serum PCBs (Table 1). Boys' whose mothers once worked at the factory were more likely to have higher serum PCBs as well. Boys who were breast fed for 26 weeks had a 28% greater serum Toxic Equivalents (TEQ) of dioxins and dioxin-like PCBs compared with boys who were not breast fed. Most importantly, there was a strong negative correlation between distance from residences to the factory and serum PCB concentration of the boys. Lowest PCB levels were reported for boys living furthest from the factory. Boys who lived less than 2 km from the workshop had adjusted mean serum 2005 TEQs of 30.6 (95% CI, 26.8-35.0) compared to those lived 2-5 km (adjusted mean=22.2; 95% CI, 20.7-23.8) and greater than 5 km (adjusted mean serum= 18.8; 95% CI, 17.2-20.6). Boys within 2 km had PCB serum levels 63% higher than boys who grew up greater than 5 km from the factory.

The study suggests that industrial contamination of the local environment may be an important source of exposure for the Chapaevsk boys. Boys who lived closest to the plant had significantly higher serum dioxin and PCB concentrations. The environmental (soil, house dust) and human (breast milk, serum) samples were also higher for in areas closer to the plant (Sergeyev et al. 2007, 2008). The finding of high serum PCBs was unexpected because PCBs were not manufactured but may have been used at the Khimprom plant. The summary measures based predominantly on PCBs were also higher among boys who had lived longer in Chapaevsk, even after adjustment for age, suggesting that the local environment and foods of Chapaevsk were an important source of PCB exposure.

Mothers with gardens had sons with higher serum dioxin and PCB levels, even after adjustment for local food consumption (Burns et al. 2009). This could be due to mothers being further exposed in the gardens through inhalation and dermal contact and then transferred the PCBs through breast feeding. The higher PCB levels could also be caused by the boys playing in and around the garden area and further getting exposed through contact of contaminated soil, inhaling kicked up dust, and ingestion caused by not washing hands. Dietary consumption of local eggs, meats, poultry, dairy, and fish were also significantly associated with higher serum dioxins and PCBs among the Chapaevsk boys (Burnes et al. 2009). In a similar study, elevated blood dioxin and PCB concentrations have been linked to local food consumption in areas with environmental exposures to these compounds (Choi et al. 2006). Research in Chapaevsk suggests that local eggs and fish have significantly higher concentrations of dioxins and PCBs compared to other regions in Russia (Sergeyev et al. 2007, Shelepchikov et al. 2006).

Choi (2006) measured serum PCB levels in 720 newborns living around New Bedford Harbor, Massachusetts from 1993 to 1998. New Bedford Harbor is a Superfund site with PCB contaminated sediment. Dredging to remove PCB contaminated sediment in the harbor took place during 1994 and 1995. There was a substantial decrease in newborn PCB serum levels after dredging; there was an increase of PCBs, however, in serum levels during the two years of dredging due to disturbance in the sediment increasing volatilization (Figure 1). Maternal age and birthplace were the strongest predictors of the total PCB levels in the newborns. Consumption of meat and local dairy by the mothers were associated with higher PCB levels in newborns. No association was found between total PCB levels and residential distance from the Superfund site (Choi 2006); however, there is a decrease in PCB levels the further the residents were from the hot spots. In this case, inhalation exposure did not have a strong effect on PCB accumulation; instead diet was the main source, according to the authors. It is important, however, to realize that children born before or during dredging had higher cord serum PCB levels than children born after dredging.

These results suggest that differences in PCB availability affect exposure risks potentially associated with the site.

In a second study in the New Bedford Harbor, indoor air concentrations of PCBs were measured in 34 homes near the harbor. The study was conducted during the dredging of contaminated river sediments between April 1994 and April 1995. PCB levels in indoor air samples ranged from 7.9 to 61 ng/m^3 in homes closest to the dredging operation compared to more distant houses which had levels ranging from 5.2 to 51 ng/m^3 (Vorhees et al. 1997, ATSDR 2000). These indoor concentrations, however, exceeded outdoor concentrations by an average of 32 fold, indicating the importance of indoor air concentrations to human exposures.

Decastro et al. evaluated the estrogenicity of PCBs present in the environmental media and human tissue and assessed exposure pathways for air, soil, and dust from New Bedford, MA. The assessment of PCB-derived estrogenic potency indicate that both solid and vapor phase environmental media are potential sources. Of the three media, air (inhalation) had the highest PCB-derived estradiol-equivalent exposure by at least one order of magnitude. Estimated total vapor-phase exposure to estradiol-equivalents was $1.86\text{x}10^{-6}$ (indoor air) and $6.30\text{x}10^{-6}$ (outdoor air) nmol E2EQ/day-kg, while exposure from yard soil was $1.38\text{x}10^{-7}$ nmol E2EQ/day-kg, and that from house dust was $6.02\text{x}10^{-8}$ nmol E2EQ/day-kg. PCBs 17 and 18 contributed over 80% of estradiol-equivalent exposure in air samples. Over 60% of the estradiol-equivalents in yard soil was attributable to PCBs 18, 99, and PCB110. PCBs 17, 18, 44, 49, and 110 together contributed more than 75% of total estradiol-equivalents in dust samples (Decastro et al. 2006). Although estradiol-equivalents deriving from PCBs in New Bedford air, soil, and dust samples were estimated to be at very low picomole levels, they may, nonetheless contribute to undesirable hormonal effects through enhanced bioavailability, bioaccumulation, and combination with other xenoestrogen exposures. Furthermore, there may be critical periods in early development that are particularly sensitive to low-level exposures (Decastro et al. 2006).

Park et al. (2009) conducted a study in which they investigated the concentrations and congener patterns of PCDD/Fs and dioxin-like PCBs in 71 human serum samples: 26 incinerator workers (10 industrial waste incinerator workers and 16 municipal solid waste incinerator workers) and 45 residents in Korea (38 residents within 1 km of the MWI and 7 residents living over 10km away). The mean PCB TEQ for industrial and Municipal Waste Incinerator workers were 6.18 and 6.27 pg/g lipid, respectively. For residents living near the industrial waste incinerators and municipal incinerators, the mean PCB TEQ was 5.99 pg/g lipid and 8.34 pg/g lipid, respectively. The control group had a PCB TEQ of 2.99 pg/g. It is evident that there is little difference between working at the incinerator and living nearby when compared to the PCB TEQ value for residents living further than 10 km away. Schuhmacher (2007) measured dietary intake of PCBs and PBDEs by adult women (ages 20-34 years) from Catalonia, Spain (Table 2). The highest concentration of PCBs was found in fish and shellfish (11864 ng/kg wet weight) followed by dairy products (675 ng/kg wet weight), while for PBDEs the highest concentrations corresponded to fats and oils, followed by fish and shellfish (588 and 334 ng/kg wet weight, respectively) (Bocio et al. 2003, Llobet et al. 2003). The results indicate that seafood is the greatest PCB contributor in diets. Other parts of diet such as meat and dairy, however, are still important factors to consider when quantifying PCB accumulation in people.

PCB Health Risks

PCBs are industrial chemicals that do not occur naturally in the environment. PCBs were first produced commercially around 1930 and used mainly as industrial lubricants, insulators and solvents until the 1970s. PCBs are probable human carcinogens and can also cause non-cancer health effects, such as reduced ability to fight infections, low birth weights, and learning problems (US EPA 2009). PCBs have been classified as endocrine disruptors (EDs), which are defined as “exogenous agents that interfere with the synthesis, secretion, transport, metabolism, binding, action, or elimination of natural blood born hormones that are present in the body and that are responsible of homeostasis, reproduction, and developmental processes” (Kavlock and Ankley 1996). Endocrine disruptors have the ability to bind and perhaps irreversibly lock a specific hormone receptor, preventing naturally produced hormones from entering the cell, binding their receptor and performing their function (also known as hormone blocking) (Brevini 2005). Exposure to EDs can occur from a number of different sources. Humans and animals can be exposed involuntarily to EDs as a result of drinking contaminated water, breathing contaminated air, ingesting food, contacting contaminated soil, and exposure through womb and breast feeding (Brevini 2005).

Concerns over endocrine disrupting pollutants stem from their ability to affect the endocrine system of wildlife and humans. Endocrine disrupting pollutants also affect the offspring of the exposed generation, even when present in minute amounts such as parts per trillion. When ingested, these compounds are deposited in the fatty tissues (i.e., adipose tissue, blood lipids, and breast milk) of the mother and may be transferred to the infant during nursing. Fetuses are the most susceptible to the effects of PCBs and are exposed via consumption of breast milk. “A slightly higher than average intrauterine exposure to PCBs may cause deficient, reduced, or lowered fetal and postnatal growth, retarded psychoneurological development, and reduced cognitive ability”(Jacobson and Jacobson 1996).

Recent studies have provided information about the relationship between low-level endocrine disrupting compound exposure and neurodevelopmental effects; few studies, however, have focused on behavioral outcomes. Sagiv et al. (2010) investigated the association between prenatal polychlorinated biphenyl (PCB) and p,p’-dichlorodiphenyl dichloroethylene (p,p’-DDE) levels and behaviors associated with attention deficit hyperactivity disorder (ADHD). Six hundred seven children aged 7–11 years (median age, 8.2 years) born in 1993–1998 to mothers residing near a PCB-contaminated harbor in New Bedford, Massachusetts participated in the study. The childrens’ ADHD symptoms were measured with the Conners’ Rating Scale for Teachers (CRS-T). The median umbilical cord serum level of the sum of 4 prevalent PCB congeners (118, 138, 153, and 180) was 0.19 ng/g serum (range, 0.01– 4.41 ng/g serum). Using the CRS-T, Sagiv et al. (2010) found higher risk for ADHD-like behaviors in children with higher levels of PCBs and p,p’-DDE. An association between low-level prenatal PCB exposure and ADHD-like behaviors in childhood when the authors found higher risk of atypical behavior on the Connors’ ADHD Index for the highest quartile of the sum of 4 PCB congeners versus the lowest quartile and a similar relation for p,p’-DDE (Sagiv et al. 2010).

Two studies done in Michigan and Taiwan have linked in utero exposure to polychlorinated biphenyls to adverse effects on neural development in children. In Michigan, deficits were found in fetal and postnatal growth and poorer short-term memory in infancy and at four years of age (Jacobson et al. 1990). These findings have been corroborated in laboratory animals and in studies of more highly exposed Taiwanese children born to women who consumed rice oil contaminated with polychlorinated biphenyls and dibenzofurans (Lilienthal and Winneke 1991).

In Michigan, Jacobson and Jacobson (1996) tested 212 children that were recruited as newborns to represent infants born to women who had eaten Lake Michigan fish contaminated with polychlorinated biphenyls. A battery of IQ and achievement tests was administered when the children were 11 years of age. The study found that prenatal exposure to polychlorinated biphenyls was associated with significantly lower full-scale and verbal IQ scores. An analysis of covariance (Figure 2) indicated that the effect was primarily in the most highly exposed children with prenatal exposures equivalent to at least 1.25 mg per gram in maternal milk, 4.7 ng per milliliter in cord serum, or 9.7 ng per milliliter in maternal serum. The IQ scores of the most highly exposed group averaged 6.2 points lower than those of the other four groups, after adjustment for potential confounding variables (P=0.007). Concentrations of polychlorinated biphenyls in maternal serum and milk at delivery were slightly higher than in the general population.

Volatilization

PCBs have the ability to volatize and release into the air. Biphenyls with 0–1 chlorine atom remain in the atmosphere, those with 1–4 chlorines gradually migrate toward polar latitudes in a series of volatilization/deposition cycles, those with 4–8 chlorines remain in mid-latitudes, and those with 8–9 chlorines remain close to the source of contamination (Wania and Mackay 1996). PCBs enter the atmosphere from volatilization from both soil and water surfaces (Hansen 1999). Vapor-phase PCBs accumulate in the aerial parts of terrestrial vegetation and food crops by vapor-to-plant transfer (Bohm et al. 1999, ATSDR 2000). This can explain why local food and home gardens may be a significant source of PCB exposure.

Food contamination is primarily caused by airborne persistent organic pollutants (POPs) that settle on plants and accumulate in the food chain. They are particularly associated with fatty foods (Farland et al. 1994, Schuhmacher et al. 2007). The FDA reported that the source of PCBs in the past was the meat-fish-poultry composite (63–100% of total dietary intake) with fish being the major contributing source (Jelinek and Corneliussen 1976). This observation appears to have remained true based on the recent Total Diet Studies conducted from 1991 to 1997 where meat, fish, and poultry were still the primary sources of PCBs in the human diet with fish being the major contributing factor (Bolger 1999). PCB accumulation for given plants can be seen in Table 6-8. Lettuce had the greatest bioaccumulation factor at 6.0 (ATSDR 2000).

Though PCBs in sediment are common sources of exposure for fish, they can also influence atmospheric concentrations of PCBs. Martinez et al (2010) quantified the release of PCBs from Indiana Harbor and Ship Canal to Lake Michigan and the atmosphere. It was determined that 4 ± 0.05 kg of total PCBs were released from the sediment to the water and 7 ± 0.1 kg of total PCBs were volatilized from the water to the air annually (Martinez et al. 2010).

Typical atmospheric concentrations of PCBs have been found to be much lower in rural locations compared to urban locales. For example, the concentration of PCBs in urban Baltimore, Maryland ranged from 0.38 to 3.36 ng/m^3, while in rural Baltimore, the concentration ranged from 0.02 to 0.34 ng/m^3, 10-19 times lower (Offenberg and Baker 1999). PCB levels in more remote areas are even lower, with mean concentrations ranging from 0.025 ng/m^3 over the Norwegian Sea to 0.074 ng/m^3 over the Eastern Arctic (Harner et al. 1998, ATSDR 2000). Water monitoring studies indicate that PCB concentrations are generally higher near sites of anthropogenic input and in in-shore waters. The concentrations of PCBs in the waters of the Great Lakes (Superior, Michigan, Huron, Erie, and Ontario) typically range from 0.07 to 1.60 ng/L (Anderson et al. 1999). Concentrations of PCBs in drinking water are generally <0.1 µg/L and thus, drinking water is not considered a significant pathway for exposure.

Given the ability of PCBs to volatilize, people living or working around contaminated sites can inhale in low doses of PCBs. According to the National Institute of Occupational Safety and Health, the federal occupational health limit for PCBs in the air of workplaces is 1 µg/m^3, based on an 8-hour day, 5-days a week. 1 microgram = 1,000 ng, which translates into 0.240 µg /m^3 or 240 ng/m^3 for constant air exposure 24 hours a day, 7 days a week.

Conclusion

There is a growing body of evidence that supports the claim that people who live in the vicinity of contaminated sites have a higher PCB concentration in their tissues than the general population. The major routes of exposure to these compounds are ingestion of fish (especially sport fish caught in polluted lakes or rivers), meat and dairy products (Dellinger et al. 1996, Falk et al. 1999). Studies indicate, however, that uncontrollable factors, like inhalation of contaminated air near hazardous waste sites, are also significant exposure routes.

When compared to the general public, people living in the vicinity of a PCB-contaminated site are subject to a higher risk of PCB exposure and associated health effects. This risk increases because PCBs impact the community's air, gardens, and local food systems. In these communities, historical PCB institutional controls, like fish advisories, will not be sufficient to protect human and ecological health.

Additionally, these studies illustrate that before choosing a remediation strategy to clean up PCB-contaminated sites, the human health impacts of dredging and the possibility of volatilization, if the sediment is left in place, must be considered. The New Bedford studies show the importance of remediation efforts and the length of time required to accomplish standards that are protective of human health. Traditional cleanup methods may have a greater impact in the short and long term than originally expected. Serum PCB concentrations decreased dramatically after dredging was completed. However, serum levels rose during the time that dredging took place. The disturbance in the sediment increased the rate of volatilization therefore increasing availability of exposure.

PCBs have been well-documented as endocrine disruptors; precautionary measures need to be taken during remediation efforts to protect the public from exposure. Pregnant women are the most susceptible populations, due to accumulation and transfer to their fetuses. "A slightly higher than average intrauterine exposure to PCBs may cause deficient, reduced, or lowered fetal and postnatal growth, retarded psychoneurological development, and reduced cognitive ability"(Jacobson and Jacobson 1996). Sagiv et al. (2010) found higher risk for ADHD-like behaviors assessed with the CRS-T at higher levels of PCBs. These results support an association between low-level prenatal organochlorine exposure and ADHD-like behaviors in childhood. Infants have also shown to have neurologic anomalies at birth and developmental delays in gross motor function during infancy (Jacobson and Jacobson 1996) as well as deficits in fetal and postnatal growth and poorer short-term memory in infancy and at four years of age (Jacobson et al. 1990).

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Tables and Figures

Predictor	Adjusted regression coefficient[a,b]		
	Estimate	95% CI	*p*-Value
Age (years)	0.177	(0.135 to 0.219)	< 0.0001
BMI (kg/m^2)	–0.038	(–0.047 to –0.029)	< 0.0001
Duration of breastfeeding (weeks)	0.003	(0.002 to 0.003)	< 0.0001
Maximum parental education[c]	–0.018	(–0.054 to 0.017)	0.31
Residence in Chapaevsk (years)	0.008	(0.001 to 0.015)	0.03
Mother ever employed at Khimprom	0.101	(0.011 to 0.190)	0.05
Mother's local gardening	0.081	(0.039 to 0.124)	< 0.0001
Current residence, distance from Khimprom			
< 2 km	0.101	(0.034 to 0.167)	0.003
2–5 km	0.021	(–0.025 to 0.067)	0.38
> 5 km	Reference		
Any local eggs eaten	0.160	(0.105 to 0.214)	< 0.0001
Any local nonpoultry meat eaten	0.088	(–0.001 to 0.176)	0.05
Any local poultry eaten	0.094	(0.016 to 0.171)	0.02
Any local dairy eaten	0.049	(0.005 to 0.092)	0.03
Any local fish eaten	0.070	(0.017 to 0.122)	0.01
Highest category of local fruit/vegetable eaten[d]	0.089	(–0.022 to 0.200)	0.12

[a]Adjusted for age, BMI, breastfeeding, parental education, residence in Chapaevsk, mother's employment at Khimprom, mother's local gardening, residential distance from Khimprom. [b]Local foods separately included in multivariate model, adjusted for total consumption. [c]Ordinal: reference level = secondary education or less, with higher levels of junior college/technical training and university graduate. [d]Reference = lowest category of local consumption.

Table 1. Predictors of log total serum concentration of PCBs (ng/g lipid) among boys in the Russian Children's Study (Burns et al., 2009)

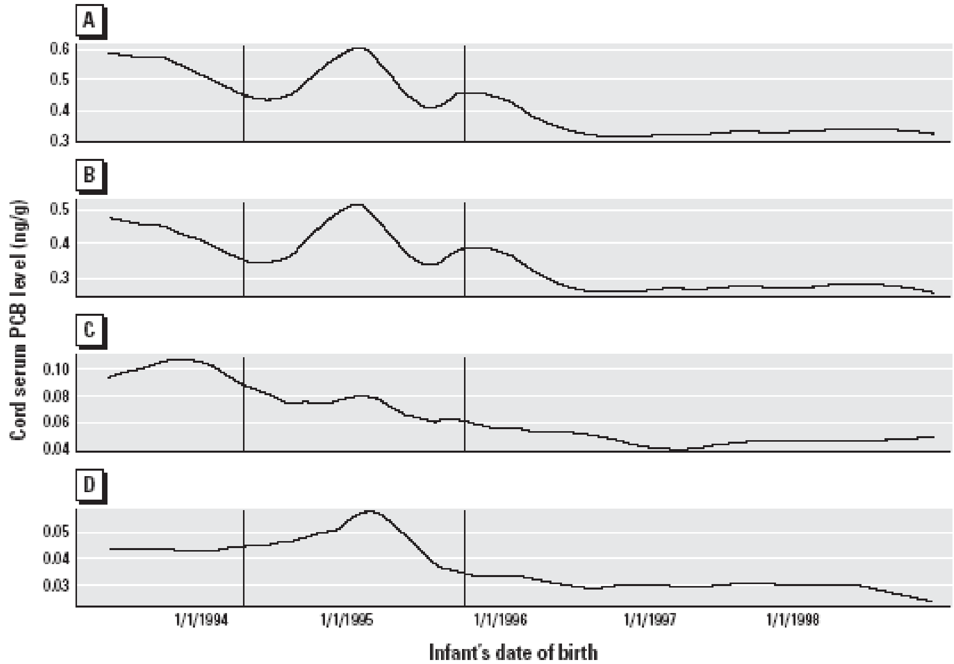

Figure 1. Covariate-adjusted smoothed plots of predicted ΣPCB (A), heavy PCB (B), light PCB (C), and PCB- 118 (D) levels versus infant's date of birth. Vertical lines denote the start and stop dates for dredging of contaminated New Bedford Harbor sediments. Plots are adjusted for child's sex, maternal age, birthplace, smoking during pregnancy, previous lactation, household income, and diet (consumption of organ meat, red meat, local dairy, and dark fish).(Choi, 2006)

Food group	Urban					Industrial				
	Daily intake (g)	PCBs (ng/day)	% of total PCB	PBDEs (ng/day)	% of total PBDE	Daily intake (g)	PCBs (ng/day)	% of total PCB	PBDEs (ng/day)	% of total PBDE
Vegetables	186	4	0.43	1.5	2.09	178	4	0.45	1.4	2.23
Pulses	9	0.1	0.01	0.1	0.14	6	0.1	0.01	0.1	0.16
Cereals	154	28	3.13	5.4	7.51	150	27	3.25	5.3	8.43
Tubercles	49	1.1	0.12	0.4	0.56	53	1.2	0.14	0.4	0.64
Fruits	197	0.9	0.10	1.2	1.67	255	1.1	0.13	1.5	2.38
Fish and shellfish	60	714	79.5	20	28.0	57	675	80.1	19	30.2
Meat and meat products	134	50	5.58	15	20.3	114	43	5.07	13	19.9
Eggs	19	9	1.0	1.2	1.67	14	7	0.77	0.9	1.43
Dairy products	90	60	6.73	4.3	5.98	85	58	6.82	4.1	6.52
Milk	236	16	1.75	4.0	5.56	262	17	2.06	4.5	7.15
Fats and oils	33	15	1.63	19	26.6	23	10	1.21	13	21.1
Total	1167	898	100	72	100	1197	843	100	63	100

Table 2. Estimated dietary intake of PCBs and PBDFs by adult women (20-34 years) according to specific place of residence (Urban or Industrial) (Schuhmacher, 2007)

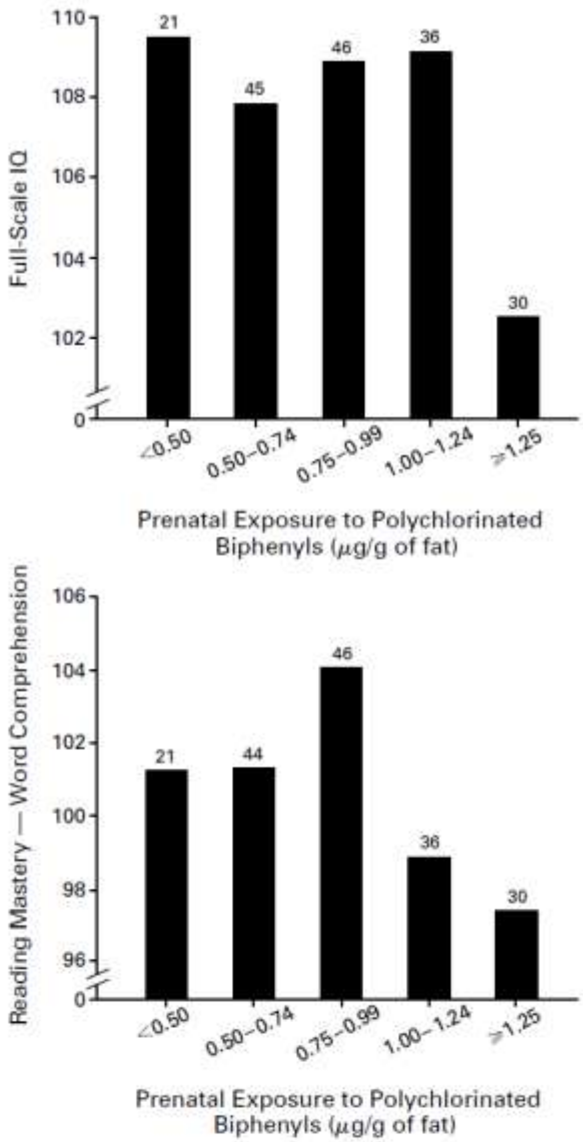

Figure 2. Scores for Full-Scale IQ Tests and Word Comprehension in Reading According to Prenatal Exposure to Polychlorinated Biphenyls (Expressed in Terms of the Fat Concentration of Maternal Milk). Scores were adjusted for the potential confounding variables Listed in the footnotes to Table 3. The number of children in each group is given above the bars. One child was not tested for reading mastery.

Crop (growth media)	Application rate	BAF[b]	Reference[a]
Carrot (soil)	Aroclor 1254 at 100 ppm mixed in top 6 inches of soil	<1 (Aroclor 1254) • 0.16 (roots)	Iwata et al. 1974
Carrot (acid soil and brown sand)	Aroclor 1254 at 0.05, 0.5, and 5 ppm (acid soil); 0.5 ppm (brown sand)	0, << 1, <1, 0.16 (roots)	Wallnöfer et al. 1975
		<1, 0.16 (roots peels)	
Carrot (soil)	PCB 4 at 1 ppm in dry soil, mixed in top 10 cm	<1 (di-PCB) 0.25 (roots) 0.25 (leaves)	Moza et al. 1976
Carrot (soil)	None	1.5 (PCB 52) 0.35 (PCB 101) 0.38 (PCB 138) 0.28 (PCB 153)	Cullen et al. 1996
Corn (field)	Aroclor 1254 and 1260 contaminated sludge (92–144 μg PCBs/L sludge)	<1	Lawrence et al. 1977
Lettuce (soil)	None	6.0 (PCB 52) 1.5 (PCB 101) 1.1 (PCB 138) 0.74 (PCB 153)	Cullen et al. 1996
Potato (soil)	None	0.29 (PCB 52) 0.01 (PCB 101) 0.17 (PCB 138) 0.28 (PCB 153)	Cullen et al. 1996
Radish (acid soil and brown sand)	Aroclor 1254 at 0.05 ppm (acid soil or brown sand), 0.5 ppm (acid soil); Aroclor 1224 at 0.2 ppm (brown sand);	0, 0, 0.02	Wallnöfer et al. 1975
	Aroclor 1254 at 5 ppm (acid soil) with moisture 40% of maximum water holding capacity	0.005	
Soybean sprouts (sandy soil)	Aroclor 1242 at 100 ppm	0.002	Suzuki et al. 1977
Sugarbeet (brown soil)	Aroclor 1254 at 0.3 ppm in soil	0.01 (leaves) to 0.5 (whole plant) 0.17 (root peels) 0.03 (peeled root)	Wallnöfer et al. 1975
Sugarbeet (field soil)	PCB 4 at 0.24 ppm in 0–10 cm soil layer and 0.17 ppm in 10–20 cm soil layer	0.07 (roots) 0.03 (leaves)	Moza et al. 1976
Tomato (soil and vermiculite)	PCB 4; PCB 7; PCB 18; PCB 52; PCB 101 (concentration not specified)	0 for all PCBs (mature plants)	Pal et al. 1980

Table 3. Plant Uptake (Bioaccumulation) of PCBs (ASTDR, 2000).

Appendix B
Results of PCB literature search for the past two years:

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