Assessment of human health risks posed by consumption of fish from the Lower Passaic River, New Jersey

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Abstract

The Lower Passaic River (LPR) in New Jersey has been impacted by a variety of human activities over the course of the last two centuries. In this risk assessment, we assessed potential human health risks associated with consumption of fish from the LPR, the human exposure pathway of greatest concern when addressing contaminated sediments. Our risk assessment incorporates fish consumption information gathered during a year-long, intercept-style creel angler survey and representative fish tissue concentrations for 156 chemicals of potential concern (COPCs) obtained from USEPA’s public database (OurPassaic website: http://www.ourpassaic.org/sizeprojects/premis/public/index.cfm?Fuseaction=contaminants). Due to the large number of COPCs investigated, this risk assessment was divided into two phases: (1) identification of COPCs that contribute to the majority of overall cancer risk and hazard estimates using deterministic and probabilistic methods, and (2) probabilistic characterization of risk using distributions of chemical concentration and cooking loss for those compounds identified in Phase 1. Phase 1 relied on point estimates of COPC concentrations and demonstrated that PCDD/Fs and PCBs (dioxin-like and non-dioxin-like) are the greatest contributors to cancer risk, while non-dioxin-like PCBs are the primary contributors to non-cancer hazard estimates. Total excess cancer risks for adult and child receptors estimated in Phase 1 were within USEPA’s acceptable cancer risk range, with the exception of RME child (3.0 × 10−4 and 1.3 × 10−4 for deterministic and probabilistic approaches, respectively). Phase 2 focused on PCDD, PCDF, and PCBs and used distributions of chemical concentrations in fish. The results showed that all excess cancer risk estimates were within the acceptable risk range, although non-cancer hazard estimates for PCBs slightly exceeded a Hazard Index of 1. This HHRA of LPR fish ingestion represents the most comprehensive evaluation conducted to date, and demonstrates that measured concentrations of COPCs are not likely to pose a health risk to people currently consume fish from the LPR.

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1. Introduction

Proximity to waterways has had a substantial impact on growth and development in cities across the United States. There may be no other area where the effects of such growth and development are more pronounced than along a 6- to 7-mile stretch of the Lower Passaic River (LPR) in New Jersey (Iannuzzi et al., 2002). This stretch of the LPR flows past several municipalities including Harrison, Kearny, Newark, and East Newark, NJ to the river’s mouth at Newark Bay. The LPR is a seminal example of a waterway that has been instrumental to the economic advancement of a region at the expense of the water body itself. Since the mid-1800s, this stretch of the Passaic River has experienced substantial urbanization and industrialization (Ludwig and Iannuzzi, 2005). There are a number of activities that have occurred, and numerous contamination sources that have existed, over this period of time that have impacted the environmental and ecological conditions of the LPR (Iannuzzi et al., 2002). Such activities include the reshaping of the tidal estuary (e.g., through dredging activities, bulkhead placement, etc.), urban development, and industrialization of the river shoreline (Crawford et al., 1994). The historical and ongoing impacts of these activities include a number of environmental stressors, such as organic- and metals-based chemical waste, pathogens (bacterial, viral, and protozoan), and other physical strains (e.g., dissolved oxygen deficiency, excessive nutrients), all of which have contributed to degradation of the river (Crawford et al., 1995; Donovan et al., 2008a,b; Iannuzzi and Ludwig, 2004; Iannuzzi et al., 2008).

One of the primary concerns about water bodies in close proximity to urban/industrial environments is the potential cancer and non-cancer health risks posed to people consuming fish, shellfish, crabs, and other biological species from the water body (Bienenfeld et al., 2003; Fry et al., 2008). The introduction of non-volatile, lipophilic, and metabolism-resistant contaminants to surface waters and sediments can result in the bioaccumulation of these contaminants in fish and other biological species commonly consumed by humans (Clarkson,
1995; Thomann, 1995). The potential significance of bioaccumulation within an aquatic ecosystem is highlighted by the National Study of Chemical Residues in Fish, which has shown that the levels of certain contaminants in fish exceed their respective water column concentrations by factors of up to 1,000,000 (USEPA, 1992a,b). Many states monitor contaminant levels in fish and shellfish. Such monitoring efforts are used to provide information about water and sediment quality, as well as the need for fish advisories (USEPA, 2000). The USEPA recently reported that a total of 3852 fish consumption advisories had been issued in 48 of 50 states, two of four territories, and five Indian tribe locations (USEPA, 2007a). In fact, the State of New Jersey has issued consumption advisories for all species of fish, shellfish, and crabs originating from the LPR, and has prohibited the harvest and sale of any such species collected from this part of the river (NJDEP, 2006–2007). These advisories were established as a result of measuring elevated levels of polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), polychlorinated biphenyls (PCBs), and mercury in fish, shellfish, and crabs from the LPR. Additionally, shellfish harvesting has been prohibited in the LPR since 1970 as a result of health concerns associated with sewage-related pathogen contamination (NJDEP, 1990).

The mere presence of a contaminant in and of itself does not pose a human health risk. There must be a complete exposure pathway for individuals to potentially be at risk. The specific activities and habits of a population targeted in a human health risk assessment are crucial elements to enhancing the underlying accuracy of that risk assessment. However, conducting surveys aimed at elucidating a targeted population’s activities and habits can be time consuming and costly. For these reasons, risk assessors often rely upon default (or generic) exposure factors established by the USEPA. In 1997, the USEPA released an updated version of the Exposure Factors Handbook, in which default values for all exposure factors, including fish ingestion rates, are presented and discussed. The default ingestion rates for freshwater fish derived by the USEPA are based on several mail surveys conducted between the late 1980s and mid-1990s (USEPA, 1997a,b). Furthermore, the target populations in these mail surveys were licensed anglers in Maine, Michigan, and regions of Lake Ontario. Because of the inherent limitations of these mail surveys, they may not represent the characteristics of many angler populations. Examples of some of the characteristics that can differentiate one water body from another include (1) urban vs. rural location, (2) open vs. restricted access, (3) fresh water vs. estuarine environment, and (4) high crime vs. low crime region.

The LPR presents unique characteristics that should be taken into account when evaluating potential cancer and non-cancer human health risks posed by chemicals and other pollutants present in biota found in the river. For example, the lowest 6 miles of the LPR flows through a highly industrial and urbanized area, is part of a tidal estuary, and therefore is comprised of brackish water rather than solely fresh water. Also, there are only a limited number of access points along this stretch of the LPR. Given these characteristics, it is obvious that the lowest 6 miles of the LPR bears little resemblance to the locations that were the subject of angler surveys from which the fish consumption exposure factors outlined in the USEPA’s Exposure Factors Handbook were derived (USEPA, 1997a; Kinnell et al., 2007). Clearly, angler populations, access to waterways, fish species present, and fish species actually consumed are characteristics of a water body that can be highly variable from one region to another, which highlights the importance of utilizing available region-specific data for evaluating risk (Ebert et al., 1993; Moya, 2004).

To address these important exposure factors, a comprehensive creel angler survey (CAS) was conducted in 2000–2001 along the lower 6 miles of the LPR to collect site-specific fish consumption data for use in assessing potential cancer and non-cancer human health risks, thereby eliminating the need for reliance upon default exposure factors that are of little relevance to the LPR (Kinell et al., 2007; Ray et al., 2007a,b). The LPR CAS was a year-long, intercept survey designed to characterize the angler population, as well as to determine site-specific exposure factors such as fishing and consumption frequency and duration, the species caught and consumed, actual meal preparation methods used, and fish consumption rates across this population (Finley et al., 2003; Kinell et al., 2007). Oversight was provided by an independent expert panel, which was commissioned to evaluate both the need for such a study as well as the adequacy of the CAS design for characterizing fish consumption endpoints. The CAS demonstrated that the LPR angler population was relatively small and consisted primarily of recreational, rather than subsistence, anglers. Fish consumption rates determined based on the CAS for the LPR were an order of magnitude lower than the default values published in the USEPA Exposure Factors Handbook (Ray et al., 2007b; USEPA, 1997a). Also of interest was the observation that none of the blue crabs caught by intercepted anglers were intended for consumption; rather, the anglers reportedly either caught them accidentally and did not keep them, or indicated that they were using them as bait (Ray et al., 2007b). The panel concluded that default exposure parameters listed in the USEPA’s Exposure Factors Handbook for fish ingestion were inadequate for the purposes of conducting a comprehensive risk assessment of the LPR given the site-specific conditions and characteristics of the local angler population (Finley et al., 2003). Furthermore, survey design and analysis recommendations were provided by the expert panel and incorporated into the CAS, reinforcing it as a powerful and relevant tool for evaluating cancer and non-cancer health risks from fish ingestion.

There are several COPCs to which individuals who consume fish, crabs, and other aquatic organisms can be potentially exposed. The primary COPCs that are typically the focus when evaluating food chain exposures are those known to bioaccumulate in the food chain, such as mercury, PCBs, select organochlorine pesticides, PCDDs, and PCDFs. These compounds are just a few of the hundreds of chemicals listed on the Hazardous Substance List (HSL) under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA (40 CFR Part 302)). More recently, there have been several non-HSL chemicals identified in various waterways across the United States that could present potential cancer and/or non-cancer health risks to populations that come into contact with contaminated waters, sediments, and biota. This broad group of compounds includes pharmaceuticals (and their metabolites) and personal care products (PPCPs), hormones, steroids, as well as chemicals contained in everyday household products such as detergents, disinfectants, plasticizers, flame-retardants [e.g., polybrominated diphenylethers (PBDEs)], and insecticides (Kolpin et al., 2002; Schwab et al., 2005).

The objective of this assessment was to determine the potential cancer and non-cancer human health risks posed by the ingestion of fish collected from the lower 6 miles of the LPR. For the purposes of this risk assessment, excess cancer risk and non-carcinogenic hazard were evaluated using both deterministic and probabilistic methods of analysis, as well as relevant exposure parameters based on the site-specific information from the LPR CAS as reported by Ray et al. (2007b).

2. Methods
2.1. Overview

USEPA has collected data that represent those fish caught and analyzed over a time period spanning three decades and has made this analytical data available online (www.ourpassaic.org). For the purposes of this risk assessment, fish tissue data from the USEPA’s database, representing four sampling events spanning the time period from 1995 to 2001, have been analyzed to characterize contaminant concentrations among fish known to be ingested by humans based on results of the LPR CAS (i.e., catfish, white perch, striped bass, American

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eel, and carp; as explained below, although carp were identified in the LPR CAS, there were no suitable analytical analyses of carp tissue in the OurPassaic.org database). There were 156 COPCs analyzed in the tissues of the former four LPR fish species that had usable data. Of the 156 COPCs identified, cancer slope factors have been established for 69 chemicals and non-cancer reference dose values have been reported for 72 chemicals. Combined, a total of 121 of the 156 COPCs have one or both type(s) of toxicity value(s), meaning that 38 of the COPCs analyzed were not evaluated in our risk assessment. The approach used to extract and process the fish tissue data available on the OurPassaic.org website, as well as the method used to calculate representative fish tissue concentrations in the species of interest, is described briefly in the Data reduction section, and in more detail in a companion paper (Tachovsky et al., submitted for publication).

The extent to which a risk assessment represents an exposed population is dependent upon the exposure assumptions incorporated into the assessment. Based on the results of the LPR CAS and the conclusions published by the expert panel charged with overseeing the LPR CAS, one of the goals of this risk assessment was to incorporate relevant, site-specific exposure parameters derived from the CAS conducted along the lower 6 miles of the LPR (Finley et al., 2003; Kinnell et al., 2007; Ray et al., 2007a,b). USEPA default exposure parameters were used only in cases where the site-specific information was either unavailable or deficient. The exposure assessment incorporated exposure scenarios for two age groups – adults (ages 18 years and older) and children (ages 1–6 years) – to demonstrate a range of estimates for exposure.

Due to the fact that valid analytical data were present in the database for 156 COPCs for the fish samples evaluated in this assessment, it was recognized that performing a probabilistic risk assessment (PRA) using concentration distributions for all chemicals would have been a cumbersome and inefficient process. Therefore, this human health risk assessment was divided into the following two phases: 1) Phase 1 – the goal of which was to evaluate human health risks and hazards associated with all COPCs measured in LPR fish species known to be consumed by the local angler population, as well as to identify those COPCs that were the greatest contributors to the health risk estimates; and 2) Phase 2 – the goal of which was to conduct a more refined risk assessment for the COPCs identified as the major risk contributors through the use of refined PRA methods. A flow chart that illustrates our approach is outlined in Fig. 1. Phase 1 itself is comprised of two types of analyses: 1) a deterministic risk assessment in which central tendency (CT) and upper bound (RME – reasonable maximum exposure) point estimates were used for all variables in the risk equation; and 2) a PRA which relied upon the same COPC fish tissue concentration point estimates derived for the deterministic assessment in combination with distributions of key exposure variables (i.e., fish ingestion rate, exposure duration, and body weight). Tissue concentrations were not distributed, however, nor was cooking loss factored into either Phase 1 assessment. The

Fig. 1. LPR fish data reduction and risk assessment flow chart.
Phase 1 PRA was based upon Monte Carlo analyses and was conducted using Crystal Ball version 7.3.1 (Decisioneering, 2008).

In Phase 2 of our assessment, those chemicals determined to be the greatest contributors to cancer or non-cancer health risks (>70% of total cancer risk or health hazard) in Phase 1 were evaluated further in a more refined probabilistic framework. In addition to utilizing distributions for ingestion rates, body weights, and exposure durations as was done in Phase 1, the concentration term also was expressed as a distribution. Distributions were developed for each chemical/congener (30 total) and each fish species (4 total) evaluated. This yielded a total of 120 concentration term distributions that were evaluated in the refined PRA framework. Each concentration term distribution was developed using two different approaches: (i) as a continuous uniform distribution (with equal probability of occurrence for any value over the range from minimum to maximum measured tissue concentration), and (ii) as an empirical distribution (in which any measured value could be selected, all measured values possessing equal probability of occurrence). Since the PRA results generated using either distribution of select COPCs were found to be nearly identical, only the results of the empirical distribution (measured values) are presented in this paper. Cooking loss was also included in this refined assessment for PCBs and PCDD/Fs (see Applicable equations and exposure parameters section below).

PCDD/Fs and dioxin-like PCBs were evaluated using the 2006 World Health Organization (WHO) Toxic Equivalency Factors (TEFs) (Van den Berg et al., 2006) for the deterministic analyses conducted during Phase 1 of the assessment. For the probabilistic analyses conducted in both Phases 1 and 2 of the assessment, these compounds were evaluated using the 2006 WHO TEFs and the distributions of the relative potency (REP) values used to derive the 2006 WHO TEFs as presented in Haws et al. (2006a). These REP custom discrete distributions were developed using the weighting framework described in Haws et al. (2006b).

2.2. Data reduction

A critical component of this risk assessment was the compilation, organization, and reduction of all pertinent historical sampling data available in the online OurPassaic.org database (database available as a Microsoft Access download at www.ourpassaic.org). This task alone was large in scope and has been presented in a step-by-step manner in a companion paper (Tachovsky et al., submitted for publication). A brief summary of the data reduction process and results is provided below.

The OurPassaic.org database contains the results of chemical analyses of biological tissue samples collected by state and federal agencies and non-governmental entities over the last three decades. During this time period, over 25 studies, consisting of 32 surveys, were conducted along and around the LPR. The biological data collected during these studies and surveys include samples from more than 35 aquatic species and consist of 712 discrete samples totaling more than 62,000 analyte records. Although the OurPassaic.org database purports to contain data for the Passaic River, not all of these samples originated from the Passaic River. Complicating the task of data analysis was the fact that sample sets were found to have analyte lists, data quality indicators, and detection/quantitation limits that were unique to each survey/study. Furthermore, in some cases duplicate samples were listed as field data, whereas in other cases, samples of unknown quality control (QC) status were included in the database. To ensure the most accurate and valid site-specific risk assessment of the LPR, the only data used in this analysis were from samples identified as being derived from the LPR and having known QC status. In cases where an analyte had a measured value less than the sample quantitation limit (SQL), but greater than the method detection limit (MDL), an estimated concentration was provided in the OurPassaic.org database. In cases where the chemical not detected above the Method Detection Limit (MDL), the USEPA provided the SQL in the Ourpassaic.org database.

A recent CAS demonstrated that only five fish species are consumed by the local LPR angler population: catfish, white perch, striped bass, carp, and American eel (Ray et al., 2007b). Analytical chemistry profiles were available for four of the five species in the OurPassaic.org database (no LPR carp data are present in the database). These four species (i.e., catfish, white perch, striped bass, and American eel) were selected for development of concentration terms, resulting in a total of 63 fish tissue samples associated with 14,060 analyte records. For the purpose of this assessment, it was assumed that whole fish, including head and viscera, were not consumed. Rather, in keeping with the USEPA’s Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories, fillet data were used in this assessment to estimate potential cancer and non-cancer human health risks associated with consuming fish from the LPR (USEPA, 2000). This approach is supported by data from the CAS, which indicated that more than 95% of the angler population intercepted for the study that reported consuming their catch (less than 9% of all anglers interviewed) ate only fish fillets (skin on or off) (Ray et al., 2007b).

LPR fish samples from the OurPassaic.org database were composite samples in which multiple fish samples were homogenized to generate enough tissue mass to accommodate the number of analyses reported. Composite collection stations were distributed throughout the lower 6 miles of the LPR. Composite samples were not standardized with regard to the number of fish in the composite or the mass contribution of each fish to the composite. Accordingly, each composite measurement was considered a realization from the sampling distribution of the means. The 50th and 95th percentiles of the composite samples were utilized as point values for CT and RME estimates, respectively, in the deterministic analyses, as well as in the Phase 1 PRA. Percentile calculations were performed using the empirical cumulative distribution function (CDF) method in SYSTAT version 11 (SYSTAT, 2008). A representative fish concentration was derived for use with the total fish consumption rate reported by Ray et al. (2007b). This value was calculated using the species analyte concentrations reported in the database and the proportion of each fish species consumed annually from the LPR reported by Ray et al. (2007b). Since there were no analytical data available for LPR carp, carp were assumed to have the same concentrations of contaminants as catfish.

2.3. Applicable equations and exposure parameters

Care was taken to use all available site-specific information when developing exposure parameters in order to present the most relevant and accurate evaluation of potential cancer and non-cancer health risks associated with consumption of fish from the LPR. Theoretical excess cancer risk and hazard associated with consumption of LPR fish were determined using Eqs. (1) and (2), below, respectively. The specific values for each of the exposure parameters and phases of the risk assessment are provided in Table 1 and are described below.

\[
\text{Risk} = \left[ \text{CT} \times \text{IRf} \times \text{EF} \times \text{FL} \times (1 - \text{Loss}) \times \text{ED} \times \text{CF} \times \text{CSF} \right] / \left[ \text{BW} \times \text{AT} \times C \right] \\
\text{Hazard} = \left[ \text{CT} \times \text{IRf} \times \text{EF} \times \text{FL} \times (1 - \text{Loss}) \times \text{ED} \times \text{CF} \right] / \left[ \text{RF.D} \times \text{BW} \times \text{AT} \right] 
\]

(1)

(2)

Where:

- \( \text{CT} \): analyte concentration (ng/g fish tissue);
- \( \text{IRf} \): fish ingestion rate (g/day);
- \( \text{EF} \): exposure frequency (days/year).
2.3.1. Analyte concentration (Ct)

The concentration term for each chemical is represented by CT and RME estimates of the representative fish, as described above, for the deterministic and probabilistic analysis in Phase 1. For the PRA analyses of Phase 2, the exposure concentration for each chemical and each fish species is specified using continuous uniform or empirical distributions. The representative fish concentration for each analyte is then re-calculated during each iteration of the PRA depending on the proportion of each species consumed and the concentration of analyte in the respective species.

2.3.2. Fish ingestion rate (IRf)

Fish ingestion rates for adult and child receptors determined in the CAS for the lower portion of the LPR (Ray et al., 2007b) were utilized in this risk assessment. The CAS for the LRP yielded a mean and 95th percentile ingestion rate of 0.42 and 1.8 g/day, respectively, for adults. In the deterministic analysis, it was assumed, for the sake of conservatism, that both adult and child receptors consumed LPR fish at equivalent ingestion rates for both CT and RME scenarios. Values from a gamma distribution of the CAS ingestion rates based on the consumption information provided by Ray et al. (2007b) were used in all PRA analyses. As Ray et al. (2007b) reported, the ingestion rate information is highly skewed since greater than 91% of the anglers interviewed indicated that they did not consume the fish they caught. Thus, a gamma distribution was used because of its flexibility and capacity to compensate for skewed data (Decisioneering, 2008). Fish ingestion rates determined based on the CAS for the LPR were an order of magnitude less than the default values published in USEPA's Exposure Factors Handbook (EFH) (Ray et al., 2007a,b; USEPA, 1997a). For an adult recreational angler, the EFH recommends CT and RME fish ingestion rates of 8 g/day (mean value) and 24 g/day (95th percentile), respectively, while for a child, the EFH recommends...
distribution based on exposure information reported by Ray et al. (1993, 2004; USEPA, 1997b). In the present case, use of the default values would clearly inflate actual fish consumption and associated health risk estimates (Finley et al., 2003; Kinnell et al., 2007).

### 2.3.3. Fraction ingestion (FI)

The chemical concentrations were derived from fish tissue samples prepared as fillets. It was assumed that consumers of LPR fish are consuming the whole fillet, and therefore the FI was given a value of 1.

### 2.3.4. Exposure frequency (EF)

An exposure frequency for consumption of LPR fish is assumed to be 365 days per year based on USEPA Risk Assessment Guidance for Superfund (USEPA, 1989). This value is selected because the ingestion rates specified in the CAS were derived as daily rates based on total annual mass of fish consumed divided by 365 days per year.

### 2.3.5. Cooking loss (Loss)

Cooking loss was not incorporated into the analyses of the Phase 1 risk assessment, but was evaluated in the probabilistic framework of Phase 2. Since measurements for the 69 and 72 analytes detected in the fish composite samples used in the cancer and non-cancer risk assessments, respectively, had been reported in the OurPassaic.org database, and given the incomplete nature of the information available on how each of the analytes can be affected by the cooking process, it was determined that Phase 1 should take the most conservative approach for evaluating health risk and assume no cooking loss for all contaminants (USEPA, 1997b). This approach is in agreement with USEPA’s Guidance on Fish Advisories. This guidance recommends that cooking loss be considered only when data are available on how methods of preparation impact the measured concentrations for specific contaminants (USEPA, 2000).

Cooking loss was factored into the more refined assessment conducted in the Phase 2 PRA for PCDD/Fs and PCBs. Information on cooking loss presented in Table C-1 of Appendix C of USEPA Guide on Fish Advisories was used to develop a distribution of cooking loss data (USEPA, 2000). Only those chemical reduction values associated with an actual cooking activity were incorporated into this distribution (e.g., skinning and trimming chemical reduction values were ignored). There were a total of 62 different cooking loss values for total PCBs and 7 values for TCDD. Distributions of these values were applied to all PCB and PCDD/F congeners, respectively, and distributed for all fish species combined since there was not enough data per fish species to distribute cooking loss on a species-specific basis.

### 2.3.6. Exposure duration (ED)

The mean and 95th percentiles for exposure duration (i.e., LPR fishing careers) determined by Ray et al. (2007b) for LPR anglers were used to represent CT and RME exposure scenarios, respectively, in the deterministic analyses conducted in Phase 1. The CT and RME values determined in Ray et al. (2007b) are 1.5 and 4.8 years respectively. These site-specific exposure durations are six times less than the recommended values published in the USEPA’s Risk Assessment Guidance for Superfund (USEPA, 1989). For the PRA analyses in Phases 1 and 2, this parameter was distributed according to a gamma distribution based on exposure information reported by Ray et al. (2007b). As with the ingestion rate information, the use of a gamma distribution approach was warranted given that these data were also skewed, as evidenced by the fact that the median LPR fishing career (0.9 years) was less than the mean (Ray et al., 2007b).

### 2.3.7. Body weight (BW)

For the deterministic risk assessment, the body weights for adult and child anglers were assumed to be 71.8 and 15 kg, respectively, based on the EPA’s EFH (USEPA, 1997b). This parameter was normally distributed for both receptors for the PRA analyses in Phases 1 and 2.

### 2.3.8. Averaging time (AT)

Since the excess cancer risk estimates reflect an excess lifetime cancer risk, the averaging time for both deterministic and probabilistic analyses is assumed to be a lifetime and is the product of 70 years and 365 days (25,550 days). Since the non-cancer hazard estimates are determined for the period of exposure, the averaging time reflects the estimated duration (CT and RME of 1.5 and 4.8 years, respectively) and frequency of exposure (356 days/year). Thus for Phase 1 deterministic analyses, the CT and RME estimates for non-cancer AT are 547.5 and 1752 days, respectively. The non-cancer AT values for Phases 1 and 2 probabilistic analyses, however, were dependent upon the ED value selected for each iteration.

### 2.3.9. Toxicity values (CSF and RfD)

Cancer Slope Factors (CSF) and Oral Reference Dose (RfD) values were collected for as many of the chemical analytes measured in the fish as were available. CSFs are upper bound estimates of carcinogenic potency, while an RfD reflects the highest daily exposure estimated to be without appreciable risk for non-carcinogenic adverse health effects over a lifetime. Based on the level of acceptance and peer review, the following hierarchy of sources was used to identify relevant toxicity criteria: values from USEPA’s Integrated Risk Information System (IRIS) were given the highest priority, followed by USEPA Region 9 values, California EPA values, and Oak Ridge National Laboratory values. For a complete listing of all COPCs detected in LPR fish and the toxicity values used in this assessment, see Table S1 in the Supplementary data section. There were certain analytes for which no toxicity values could be identified and these chemicals were not included in this risk assessment.

For every fish sample, the concentration values of each PCDD/F and dioxin-like PCB were multiplied by either their respective 2006 WHO TEF (for all analytes) or by weighted REP values (PRA analyses only) to determine a TCDD toxic equivalent (TEQ). Since there are no RDs available for dioxin-like compounds, and the development of such remain controversial, these analytes were considered only in the excess cancer risk analyses. Both excess cancer risk estimates and non-cancer hazard estimates were determined for the non-dioxin-like PCBs. In the cancer risk analyses, total PCBs were determined based on the sum of PCB homolog groups with the concentration of dioxin-like PCBs removed from this sum (Connor et al., 2005). For non-dioxin-like PCBs, a CSF of 1 mg/kg day was used when evaluating the CT PCB concentration, and a CSF of 2 mg/kg day was used when evaluating the RME PCB concentration. For the evaluation of non-carcinogenic hazard, total PCBs were assessed based on the sum of PCB homolog groups including dioxin-like PCBs. In this case, an oral RfD of $2 \times 10^{-5}$ was used, a toxicity value that was originally derived for Aroclor 1254 (USEPA, 1997b). Application of the Aroclor 1254 RfD to total PCB concentration is recommended by the 2000 EPA Fish Advisory Guidance (USEPA, 2000), and is utilized in the most recent draft of the Risk section of the USEPA’s Focused Feasibility Study of the LPR (USEPA, 2007b).

### 3. Results

#### 3.1. Phase 1

#### 3.1.1. Deterministic risk assessment

CT and RME estimates of concentrations for chemical analytes measured in the LPR fish samples, for which toxicity values were available, were used in deterministic calculations of potential human health risks posed by consumption of fish from the Lower Passaic River, New Jersey, Sci Total Environ (2009), doi:10.1016/j.scitotenv.2009.03.004
health excess cancer risk and hazard indices for adult and child receptors associated with LPR fish ingestion. Table 2 shows the results for excess cancer risk for these two receptors using point estimate CT and RME values for all exposure parameters. The total excess cancer risk for all chemicals with CT estimates is $2.7 \times 10^{-6}$ and $1.3 \times 10^{-5}$ for adult and child receptors, respectively. Excess cancer risk results are more than an order of magnitude higher when RME estimates are considered ($6.4 \times 10^{-5}$ and $3.0 \times 10^{-4}$ for adult and child receptors, respectively).

Using the conservative deterministic risk assessment approach, PCDD/Fs, PCBs (non-dioxin- and dioxin-like), other organic chemicals, and metals/PAHs were found to contribute 51%, 26%, 18%, and 5%, respectively, to the overall CT excess cancer risk in adults and children (Fig. 2; Table 6). Excess cancer risk is similarly distributed among the chemical groups when conservative deterministic RME estimates are used: 52% for PCDD/Fs, 31% for dioxin-like and non-dioxin-like PCBs, 12% for other organic contaminants, and 5% for metals and PAHs (Fig. 2; Table 6). A breakdown of excess cancer risk by chemical group shows that the excess cancer risk for all chemical groups and scenarios is within the acceptable risk range with the exception of the RME child (albeit only slightly outside of the acceptable risk range, at $1.6 \times 10^{-4}$). Evaluating the conservative deterministic risk assessment results more closely, 2,3,7,8-TCDD and PCB126 congeners were found to account for almost all of the PCDD/F and dioxin-like PCB risk, respectively (data not shown). Overall, the total excess cancer risk estimate for the RME child only slightly exceeds the acceptable risk range of $1 \times 10^{-4}$ when the most conservative site-specific approach to determining excess cancer risk is employed, including the assumption that ingestion rates between adult and child receptors are equivalent and that individuals are exposed to the maximum concentrations of these COPCs in fish.

Table 3 indicates that hazard indices for the adult RME estimates, as well as child CT and RME estimates, exceed the acceptable hazard index value of 1.0 when hazards are combined across all chemicals. The hazard index for the CT child receptor scenario is 3, while the more conservative adult and child RME scenarios are about 4 and 20, respectively. PCBs are by far the most significant contributors to the non-cancer hazards associated with exposure to LPR fish, contributing 89% and 91% to the overall hazard estimates (CT and RME, respectively) (Table 3). Other organics account for 6% and 5% of the hazard, while metals are similarly responsible for 5% and 4% of the hazard (CT and RME, respectively) (Fig. 3). The contribution of PAHs to overall hazard index estimates is negligible. As noted above, non-cancer toxicity criteria values have not been developed for PCDD/Fs and, as such, these compounds are not included in the hazard assessment.

Tables 2 and 3 also illustrate the percent contribution that non-detect values (%ND) had on the excess cancer risk and non-cancer hazard estimates. In both cases, risk based on RME scenarios is less

Table 2
Phase 1 deterministic excess cancer risk estimates.

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<thead>
<tr>
<th>Risk</th>
<th>CT</th>
<th>RME</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chemicals</td>
<td>Adult</td>
<td>Child</td>
</tr>
<tr>
<td>PCDD/Fs</td>
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<td>6.7E-06</td>
</tr>
<tr>
<td>DL PCBs*</td>
<td>4.8E-07</td>
<td>2.3E-06</td>
</tr>
<tr>
<td>(Mono PCBs)</td>
<td>4.1E-07</td>
<td>2.0E-06</td>
</tr>
<tr>
<td>(Non-DL PCBs)</td>
<td>6.8E-08</td>
<td>3.2E-07</td>
</tr>
<tr>
<td>Metals</td>
<td>2.3E-07</td>
<td>1.1E-06</td>
</tr>
<tr>
<td>PAHs</td>
<td>8.5E-08</td>
<td>4.1E-07</td>
</tr>
<tr>
<td>Organics</td>
<td>4.8E-07</td>
<td>2.3E-06</td>
</tr>
<tr>
<td>Sum</td>
<td>2.7E-06</td>
<td>1.3E-05</td>
</tr>
<tr>
<td>Non-detect</td>
<td>6.1E-07</td>
<td>2.9E-06</td>
</tr>
<tr>
<td>% Non-detect</td>
<td>23%</td>
<td>23%</td>
</tr>
</tbody>
</table>

Shaded cells indicate excess cancer risk that exceeds upper bound acceptable risk.

\*Dioxin-like PCBs are comprised of non-ortho- and mono-ortho-chlorinated PCBs.
impacted by the non-detect values than the CT scenarios. This is expected since the upper percentile of the distribution of the means is more likely to represent a measured value as opposed to a non-detect. Also, the %ND values are smaller in the hazard table (Table 3) than in the excess cancer risk table (Table 2). This can be explained by the fact that a significant majority of non-cancer hazard is related to total PCBs, which were generally measured as high values that exceeded the limits of detection.

3.1.2. Probabilistic risk assessment (cancer risk)

The mean, the 50th and 95th percentile summary statistics are presented in Table 4 for all results derived from PRA analyses. Distributions of ingestion rate, exposure duration, and body weight parameters were utilized in this analysis. However, because of the number of chemicals evaluated in this analysis, only point estimates for analyte concentrations in fish tissue were used. The results in Table 4 for PCDD/F and DL PCB are based on the 2006 WHO TEFs. The 95th percentile of total excess cancer risk for the child RME scenario (i.e., the most conservative estimate) is $1 \times 10^{-4}$, or less than half the total excess cancer risk estimated using deterministic risk analysis. As is observed with the results of the deterministic analysis, the excess cancer risk estimates for all other scenarios fall below the acceptable risk benchmark of $1 \times 10^{-4}$ (non-shaded cells in Table 4). Unlike the results from the deterministic analysis, however, none of the chemical groups independently exceeds the acceptable risk range in this most conservative scenario. The 95th percentile for child RME probabilistic risk for PCDD/Fs is still the highest among all chemical groups at $6.5 \times 10^{-5}$, or half the total risk; dioxin-like and non-dioxin-like PCBs together comprise more than one quarter of the total risk estimate. A probabilistic analysis of the proportions of contribution to total excess cancer risk for each group of COPCs was also conducted (Table 6). The results shown in Table 6 represent the distribution means for each proportion, which when combined among the COPCs add up to 100%. As with the deterministic analyses, PCDD/Fs and PCBs (dioxin-like and non-dioxin-like) are the largest contributors to total risk for both CT (47% and 26%, respectively) and RME (47% and 34%) scenarios. Other organics and metals/PAHs contribute 22% and 6%, respectively, for the CT scenario, and 13% and 5%, respectively, for the RME scenario. The reason for the disparity in the percent contribution for the other organics is that the tissue concentrations of the analytes that contribute the most to organics risk are based on detection limit values, and therefore do not vary from CT to RME.

Table 5 illustrates how these probabilistic excess cancer risk estimates are affected when the discrete 2006 WHO TEFs are replaced with weighted REP distributions. Overall, the total 95th percentile probabilistic excess cancer risk estimates for the child receptor exceed $1 \times 10^{-4}$ for both CT and RME total concentration estimates ($1.5 \times 10^{-4}$ and $2.6 \times 10^{-4}$, respectively). The reason for this becomes obvious when the dioxin-like chemical groups are assessed individually. Relative to the 2006 WHO TEFs, the REP weighted distributions increase the 95th percentile probabilistic risk estimate for dioxin-like PCBs in child receptors by almost an order of magnitude (from $1.2 \times 10^{-5}$ to $8.8 \times 10^{-5}$ for CT, and $1.9 \times 10^{-5}$ to $1.5 \times 10^{-4}$ for RME). As for PCDD/Fs, the REP weighted distributions have little to no effect on excess cancer risk estimates (Table 5). This is because the bulk of the PCDD/F TEQ contribution is from 2,3,7,8-TCDD, the basis of comparison for all dioxin-based risk estimates and which has a TEF of 1. Additionally, this analysis suggests that the human cancer risk posed by PCBs may be underestimated using the 2006 WHO TEFs. Again, PCB126 is the

---

**Table 3**

Phase 1 deterministic hazard index estimates.

<table>
<thead>
<tr>
<th>Chemical</th>
<th>CT</th>
<th>RME</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Adult</td>
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</tr>
<tr>
<td>Total PCBs</td>
<td>0.56</td>
<td>2.70</td>
</tr>
<tr>
<td>Metals</td>
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</tr>
<tr>
<td>PAHs</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Organics</td>
<td>0.04</td>
<td>0.19</td>
</tr>
<tr>
<td>Sum</td>
<td>0.64</td>
<td>3.05</td>
</tr>
<tr>
<td>% Non-detects</td>
<td>7%</td>
<td>7%</td>
</tr>
</tbody>
</table>

Shaded cells indicate excess cancer risk that exceeds acceptable hazard index. *The sum of all PCB homologues.*

---

**Fig. 3.** Non-cancer Hazard Summary for Ingestion of Fish from the Lower Passaic River. The central tendency (CT) and reasonable maximum exposure (RME) estimates represent the 50th and 95th percentiles, respectively, of the sampling distribution of the composite concentration means.
As Table 6 illustrates, PCDD/Fs and PCBs (dioxin-like and non-dioxin-like) are the largest contributors to total excess cancer risk when weighted REPs are incorporated into the risk analyses (CT scenario: 33% and 54%, respectively; RME scenario: 33% and 48%, respectively). Other organics and metals/PAHs contribute 15% and

Table 5
Phase 1 probabilistic excess cancer risk estimates using weighted REPs for PCDD/Fs and DL PCBs.

<table>
<thead>
<tr>
<th>Statistics – Adult</th>
<th>Total (weighted)</th>
<th>PCDD/Fs (weighted)a</th>
<th>DL PCBs (weighted)b</th>
<th>non-DL PCBsb</th>
<th>Metals</th>
<th>PAHs</th>
<th>Organics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>9.6E–06</td>
<td>1.7E–06</td>
<td>6.7E–06</td>
<td>3.1E–07</td>
<td>8.3E–08</td>
<td>1.3E–07</td>
<td>7.3E–07</td>
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<tr>
<td>50%</td>
<td>1.8E–06</td>
<td>5.1E–07</td>
<td>5.8E–07</td>
<td>9.6E–08</td>
<td>2.6E–08</td>
<td>2.3E–08</td>
<td>2.3E–07</td>
</tr>
<tr>
<td>95%</td>
<td>3.3E–05</td>
<td>7.3E–06</td>
<td>1.9E–05</td>
<td>1.3E–06</td>
<td>3.5E–07</td>
<td>5.7E–07</td>
<td>3.1E–06</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Statistics – Child</th>
<th>Total (weighted)</th>
<th>PCDD/Fs (weighted)a</th>
<th>DL PCBs (weighted)b</th>
<th>non-DL PCBs</th>
<th>Metals</th>
<th>PAHs</th>
<th>Organics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>4.5E–05</td>
<td>8.1E–06</td>
<td>3.1E–05</td>
<td>1.4E–06</td>
<td>3.8E–07</td>
<td>5.8E–07</td>
<td>3.4E–06</td>
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<tr>
<td>50%</td>
<td>8.4E–06</td>
<td>2.4E–06</td>
<td>2.7E–06</td>
<td>4.5E–07</td>
<td>1.3E–07</td>
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<tr>
<td>95%</td>
<td>1.5E–04</td>
<td>3.5E–05</td>
<td>8.8E–05</td>
<td>5.9E–06</td>
<td>1.6E–06</td>
<td>2.6E–06</td>
<td>1.4E–05</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Statistics – Child</th>
<th>Total (weighted)</th>
<th>PCDD/Fs (weighted)a</th>
<th>DL PCBs (weighted)b</th>
<th>non-DL PCBs</th>
<th>Metals</th>
<th>PAHs</th>
<th>Organics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.7E–05</td>
<td>3.3E–06</td>
<td>1.1E–05</td>
<td>1.0E–06</td>
<td>2.3E–07</td>
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<tr>
<td>50%</td>
<td>3.2E–06</td>
<td>9.0E–07</td>
<td>1.0E–06</td>
<td>3.3E–07</td>
<td>3.5E–08</td>
<td>3.1E–08</td>
<td>2.6E–07</td>
</tr>
<tr>
<td>95%</td>
<td>5.7E–05</td>
<td>1.4E–05</td>
<td>3.2E–05</td>
<td>4.4E–06</td>
<td>5.2E–07</td>
<td>1.1E–06</td>
<td>3.5E–06</td>
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Shaded cells indicate excess cancer risk that exceeds upper bound acceptable risk.

Table 4
Phase 1 probabilistic excess cancer risk estimates using 2006 WHO TEFs for PCDD/Fs and DL PCBs.

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<th></th>
<th></th>
<th></th>
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<th></th>
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<tr>
<td>Mean</td>
<td>3.6E–06</td>
<td>1.8E–06</td>
<td>5.9E–07</td>
<td>3.1E–07</td>
<td>8.3E–08</td>
<td>1.3E–07</td>
<td>7.3E–07</td>
</tr>
<tr>
<td>50%</td>
<td>1.1E–06</td>
<td>5.1E–07</td>
<td>1.8E–07</td>
<td>9.6E–08</td>
<td>2.6E–08</td>
<td>2.3E–08</td>
<td>2.3E–07</td>
</tr>
<tr>
<td>95%</td>
<td>1.5E–05</td>
<td>7.5E–06</td>
<td>2.5E–06</td>
<td>1.3E–06</td>
<td>3.5E–07</td>
<td>5.7E–07</td>
<td>3.1E–06</td>
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<table>
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</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.7E–05</td>
<td>8.2E–06</td>
<td>2.8E–06</td>
<td>1.4E–06</td>
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<td>5.8E–07</td>
<td>3.4E–06</td>
</tr>
<tr>
<td>50%</td>
<td>5.4E–06</td>
<td>2.4E–06</td>
<td>8.8E–07</td>
<td>4.5E–07</td>
<td>1.3E–07</td>
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<td>1.1E–06</td>
</tr>
<tr>
<td>95%</td>
<td>7.0E–05</td>
<td>3.5E–05</td>
<td>1.2E–05</td>
<td>5.9E–06</td>
<td>1.6E–06</td>
<td>2.5E–06</td>
<td>1.4E–05</td>
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</thead>
<tbody>
<tr>
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<td>6.5E–06</td>
<td>3.3E–06</td>
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<td>2.2E–07</td>
<td>8.5E–07</td>
</tr>
<tr>
<td>50%</td>
<td>2.0E–06</td>
<td>9.1E–07</td>
<td>3.1E–07</td>
<td>3.3E–07</td>
<td>3.5E–08</td>
<td>3.1E–08</td>
<td>2.6E–07</td>
</tr>
<tr>
<td>95%</td>
<td>2.8E–05</td>
<td>1.4E–05</td>
<td>4.1E–06</td>
<td>4.4E–06</td>
<td>5.2E–07</td>
<td>1.1E–06</td>
<td>3.5E–06</td>
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</tbody>
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<table>
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<tr>
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<th></th>
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</thead>
<tbody>
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<td>Mean</td>
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<td>1.5E–05</td>
<td>4.5E–06</td>
<td>4.9E–06</td>
<td>5.6E–07</td>
<td>1.0E–06</td>
<td>3.9E–06</td>
</tr>
<tr>
<td>50%</td>
<td>9.5E–06</td>
<td>4.3E–06</td>
<td>1.5E–06</td>
<td>1.6E–06</td>
<td>1.7E–07</td>
<td>1.4E–07</td>
<td>1.2E–06</td>
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<tr>
<td>95%</td>
<td>1.3E–04</td>
<td>6.5E–05</td>
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<td>2.3E–06</td>
<td>4.9E–06</td>
<td>1.7E–05</td>
</tr>
</tbody>
</table>

Shaded cell indicates excess cancer risk that exceeds upper bound acceptable risk.

**primary risk contributor among dioxin-like PCBs (data not shown).**

As Table 6 illustrates, PCDD/Fs and PCBs (dioxin-like and non-dioxin-like) are the largest contributors to total excess cancer risk when weighted REPs are incorporated into the risk analyses (CT scenario: 33% and 54%, respectively; RME scenario: 33% and 48%, respectively). Other organics and metals/PAHs contribute 15% and 28% to the total excess cancer risk.
4%, respectively, for the CT scenario, and 9% and 3%, respectively, for the RME scenario.

3.1.3. Probabilistic risk assessment (hazard)

Hazard indices for most of the chemical groups also were estimated using the PRA approach. As with the PRA excess cancer risk estimates, the CT and RME point estimates for fish tissue concentrations were utilized, along with distributions for the three exposure parameters mentioned above. Overall, the 95th percentile hazard index estimates exceed 1.0 for both adult and child receptors using both CT and RME values (Table 7; Fig. 3). In fact, for both child CT and RME scenarios, the 95th percentile hazard indices exceed the acceptable hazard index (1.0) by more than an order of magnitude (approximately 13 and 21, respectively). What is most readily apparent is that non-dioxin-like PCBs are largely responsible for these large 95th percentile hazard estimates, with indices of approximately 12 and 19, respectively. For the child RME scenario, other organics also slightly exceed the acceptable hazard benchmark (1.1 vs. 1.0), while the hazard associated with metals is approximately 1.0. The hazard indices for PAHs suggest these compounds present a negligible non-carcinogenic health risk. The 95th percentile values for these probabilistic hazard indices compare well with those derived in the deterministic analyses (Table 3).

3.2. Phase 2

Based on the results from both of the Phase 1 analyses, it is clear that, of all the chemical groups measured in the LPR fish tissues, PCDD/Fs, dioxin-like PCBs, and non-dioxin-like PCBs contribute the bulk of the excess cancer risk associated with fish ingestion (>70% total cancer risk or non-cancer hazard regardless of the Phase 1 assessment; Table 6). As such, these three groups of compounds were further evaluated in a more detailed PRA in which distributions for the concentrations of each congener in each fish species were assessed.

Table 7
Phase 1 probabilistic hazard estimates.

<table>
<thead>
<tr>
<th>Scenario</th>
<th>PCDD/Fs (%)</th>
<th>DL PCBs (%)</th>
<th>Non-DL PCBs (%)</th>
<th>Metals (%)</th>
<th>PAHs (%)</th>
<th>Sum DL PCBs and non-DL PCBs (%)</th>
<th>Sum PCDD/Fs, DL PCBs, and non-DL PCBs (%)</th>
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<tbody>
<tr>
<td><strong>CT estimates</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Adult</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Mean</td>
<td>0.90</td>
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<td>3.6e-05</td>
<td>0.06</td>
<td></td>
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<tr>
<td>50%</td>
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<td>0.49</td>
<td>0.03</td>
<td>1.9e-05</td>
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<tr>
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<td>2.84</td>
<td>2.51</td>
<td>0.15</td>
<td>1.3e-04</td>
<td>0.20</td>
<td></td>
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<tr>
<td>Child</td>
<td></td>
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</tr>
<tr>
<td>Mean</td>
<td>4.19</td>
<td>3.68</td>
<td>0.22</td>
<td>1.7e-04</td>
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<tr>
<td>50%</td>
<td>2.73</td>
<td>2.37</td>
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<td>9.1e-05</td>
<td>0.19</td>
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<tr>
<td>95%</td>
<td>13.09</td>
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<td>0.68</td>
<td>5.9e-04</td>
<td>0.90</td>
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<tr>
<td><strong>RME estimates</strong></td>
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</tr>
<tr>
<td>Mean</td>
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<tr>
<td>50%</td>
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<td>0.85</td>
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<td>0.36</td>
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<tr>
<td>50%</td>
<td>4.55</td>
<td>4.07</td>
<td>0.22</td>
<td>1.5e-04</td>
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<td>21.31</td>
<td>19.27</td>
<td>1.01</td>
<td>1.0e-03</td>
<td>1.13</td>
<td></td>
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</table>

Shaded cells indicate excess cancer risk that exceeds acceptable hazard index.

Please cite this article as: Urban JD, et al, Assessment of human health risks posed by consumption of fish from the Lower Passaic River, New Jersey, Sci Total Environ (2009), doi:10.1016/j.scitotenv.2009.03.004
along with the distributions for the exposure parameters described in the Phase 1 PRA. Although the contribution to total excess cancer risk by the other organics ranged from 9% to 22% depending upon the analysis and scenario, it was determined that much of the excess cancer risk associated with this COPC group was being driven by non-detect analytes, and therefore they were not included for Phase 2 analysis. Additionally, cooking loss was accounted for in this analysis. Both uniform (continuous) and empirical distributions were developed for each congener; however, the distribution methods produced very similar results, so the results of only the empirical distribution are presented here (Tables 8 and 9; Fig. 2).

Table 8 lists the mean, the 50th and 95th percentile summary statistics for excess cancer risk associated with LPR fish ingestion using this PRA design. Neither adult nor child excess cancer risk exceeds the acceptable risk levels when either the 2006 WHO TEFs or weighted REPs are applied to the congener concentration distributions. Similar to the results of Phase 1 PRA, application of the weighted REPs results in an increased excess cancer risk in both adult and child scenarios relative to the excess cancer risk estimates based on the 2006 WHO TEFs. Indeed, the 95th percentile probabilistic risk estimates for adult and child are close to three-fold greater for the weighted REPs than the 2006 WHO TEFs.

Table 9 shows the breakdown of the total excess cancer risk using probabilistic methods for LPR fish ingestion by chemical group. Since there is some debate surrounding the relative potency of the mono-ortho dioxin-like PCB TEFS (Van den Berg et al., 2006), the dioxin-like PCBs were divided further into mono-ortho and non-ortho PCBs. Based on the 2006 WHO TEFs, PCDD/Fs are the greatest contributors to total excess cancer risk, although dioxin-like PCBs are also significant contributors, with the 95th percentile excess cancer risk estimates only about two-fold less than those determined for PCDD/Fs. Non-dioxin-like PCBs account for almost four-fold and three-fold less risk than PCDD/Fs for adult and child 95th percentile risk estimates, respectively. The sum of excess cancer risk estimates for dioxin-like and non-dioxin-like PCBs is approximately equal to that estimated for PCDD/Fs (Table 10). Weighted REPs have little effect on excess cancer risk associated with PCDD/F but have a profound impact on dioxin-like PCB risk contribution. The PCDD/F 95th percentile risk for a child receptor is equivalent regardless of whether the 2006 WHO TEFs for weighted distributions of REPs are used, whereas weighted REP distributions increase dioxin-like PCB-related risk by almost eight-fold. Expectedly, this has the effect of decreasing the PCDD/F contribution to total excess cancer risk (from 52% using 2006 WHO TEFs to 33% using weighted REPs for both adult and child) while concomitantly increasing the proportion of total excess cancer risk associated with all PCBs (from 48% using 2006 WHO TEFs to 67% using weighted REPs for both adult and child).

To further investigate this observation, subclasses of dioxin-like PCBs were individually evaluated. The results of comparing the excess cancer risk associated with mono-ortho vs. non-ortho PCBs when weighted REPs are applied to this analysis illustrate the high degree of variability surrounding the mono-ortho PCB REPs. Indeed, the 95th percentile child risk for mono-ortho PCBs is more than 25-fold greater using weighted REPs than the 2006 WHO TEFs, whereas the difference is only about three-fold for the non-ortho PCBs (Table 9). Even given this variability, what remains clear with the weighted REP analysis is that non-ortho PCBs are a significant contributor to total excess cancer risk.

As already indicated in the discussion of the Phase 1 assessment, because there are no non-cancer toxicity benchmarks available for the dioxin-like compounds, the hazard assessment for Phase 2 PRA includes only non-dioxin-like PCBs. The results, illustrated in Table 10 and Fig. 3, further support the results of the probabilistic and...
deterministic hazard assessments of Phase 1; that is, non-dioxin-like PCBs only). These values are approximately three-fold and two-fold lower than analogous non-dioxin-like PCB hazard quotients in the Phase 1 PRA (Fig. 3). Cooking loss, which was not factored into the Phase 1 PRA analysis, represents the majority of the difference in hazard indices between the two PRA analyses.

4. Discussion

Various point and non-point source pollutants have affected the LPR adversely for more than a century. Although fishing advisories have been established for this area over the last three decades, there remains concern over the potential cancer and non-cancer health risks posed by consumption of fish caught from this river. Recently, a comprehensive CAS was performed to characterize the population of anglers along the lowest 6 miles of the LPR (Finley et al., 2003; Kinnell et al., 2007; Ray et al., 2007b). The CAS demonstrated that very few anglers frequent this section of the LPR (385 or less annually), and all were recreational fishermen. No subsistence fishermen were identified during the course of the CAS (Ray et al., 2007b). Fish consumption rates were determined to be significantly lower than the typical defaults recommended for recreational anglers in the USEPA’s Exposure Factors Handbook (USEPA, 1997a).

The objective of this risk assessment was to estimate the excess cancer risk and non-cancer health hazard posed to consumers of LPR fish from the lowest 6 miles of the LPR based on exposure parameters derived from the LPR CAS and Representative Fish concentrations for contaminants based upon data contained in the OurPassaic.org database (Tachovsky et al., submitted for publication; www.ourpassaic.org). To conduct the current risk assessment, a two-phased approach was designed to assess the potential excess cancer risk and non-cancer health associated with ingestion of fish from the LPR for both adult and child receptors. First, deterministic and probabilistic methods were employed to identify which groups of compounds contributed most to overall excess cancer risk estimates. The deterministic method employed, though useful as an initial step in assessing risk due to its relative time effectiveness, is a highly conservative screening-level type approach for assessing risk since it is dependent upon upper bound point estimates while failing to account for the variability and uncertainty associated with exposure variables. Use of a PRA methodology in Phase 1 allowed us to further probe health risks presented by the combined COPC concentrations and to assess the degree to which the distributions of the exposure factors affect estimates of risk. The process of first conducting a deterministic risk assessment and then conducting a probabilistic risk assessment to understand variability and uncertainty is consistent with USEPA probabilistic risk assessment guidance (USEPA, 2001).

In the first phase of this analysis, PCDD/Fs, dioxin-like PCBs, and total PCBs are identified as the chemical groups that contribute most to the excess cancer health risk. Additionally, the excess cancer risk calculated for the adult receptor scenarios is within the acceptable risk range. However, the sum of all compounds does slightly exceed the $1 \times 10^{-4}$ benchmark for the RME child receptor, as does the PCDD/Fs category alone. The RME excess cancer risk estimates are representative of the most conservative exposure assumptions: i.e., ingestion of fish samples containing the highest levels of all the compounds measured [RME values are equivalent to the maximum analyte concentration reported due to the small number of relevant fish samples (Tachovsky et al., submitted)], no cooking loss, and equivalent fish ingestion rates for both adult and child receptor scenarios.

For the second phase of this risk assessment, the use of site-specific exposure parameters and the 2006 WHO TEFs demonstrates that the 95th percentile excess cancer risk associated with the consumption of fish caught in the lowest 6 miles of the LPR for an adult or child is decidedly below USEPA’s unacceptable cancer risk benchmark (adult 95th percentile = $6.7 \times 10^{-6}$, child 95th percentile = $3.1 \times 10^{-5}$). Upon further examination of the cancer risk contributions of each chemical group, it is apparent that PCDD/Fs and the combined PCBs chemical groups are each responsible for roughly half of the total risk associated with fish consumption for this area of the LPR (Table 9). Cooking loss had a significant impact on the probabilistic cancer risks calculated in the Phase 2 PRA. Excess cancer risk associated with PCDD/Fs decreased by more than two-fold for adult and child receptors at the 95th percentile, whereas total PCB cancer risk was reduced by approximately 30% when cooking loss distributions were factored into the Phase 2 PRA.

These analyses demonstrate that there is a great deal of variability/uncertainty associated with the mono-ortho-PCB REPs. However, the same is not true for the non-ortho PCBs. Indeed, regardless of whether the 2006 WHO TEFs or the weighted REPs were applied to the PCB concentrations in fish tissue, non-ortho PCBs in general (and PCB126 in particular) contribute(s) to the bulk of overall PCB cancer risk.

The current study also examined the potential non-cancer hazards associated with ingesting fish from the LPR. The non-cancer hazard assessment employed both deterministic and probabilistic analytical methods to calculate hazard indices. PCDD/Fs were not factored into these hazard assessments since no oral RfD (nor analogous non-cancer toxicity criteria) exists for these chemicals. Total PCB homologues were evaluated in this hazard assessment. Hazard indices for all chemicals combined (Phase 1) were found to exceed 1.0 by approximately 20-fold when the most conservative scenario (child RME) was considered. The majority of the overall health hazard for adult and child in the Phase 1 assessment was due to PCBs. Taking cooking loss into account (Phase 2) results in a drop in PCB hazard, although it still exceeds 1.0 by more than six-fold. Average cooking loss accounts for an approximate one-third reduction in hazard associated with PCBs (compare Tables 7 and 11). PCB cooking loss estimates were based on a distribution of cooking loss values that represent various cooking techniques and fish species (USEPA, 2000). These cooking loss estimates for PCBs are similar to what has been reported previously in the literature (Sherer and Price, 1993). The RfD established for Aroclor-1254 by the USEPA’s IRIS program was used for total PCB hazard assessment. It is possible that the application of an Aroclor-derived RfD to total homologue PCB tissue concentrations inflated hazard values to a certain extent. However, use of the Aroclor-based RfD is supported by USEPA, which currently employs the same RfD for assessing PCB hazard associated with fish and crab ingestion (USEPA, 2007b).

4.1. Risk characterization

The probabilistic risk assessments indicate that the excess cancer risk estimates for adult as well as child receptors are generally within the acceptable risk range established by the USEPA. There is only a slight exceedance of the acceptable excess cancer risk level for the RME child at the 95th percentile of the risk distribution ($1.3 \times 10^{-4}$ vs. $1.0 \times 10^{-4}$), and this exceedance is not supported by the results of the more refined Phase 2 PRA. Moreover, when the deterministic child...
RME excess cancer risk estimate is compared to the Phase I PRA risk distributions, the deterministic child RME excess cancer risk is greater than the 100th percentile using WHO TEFs and slightly larger than the 95th percentile using weighted TEFs (data not shown); either metric suggests that the deterministic child RME excess cancer risk estimate is a conservative estimate of excess cancer risk. Additionally, Ray et al. (2007b) estimated the annual angling population along the lower 6 miles of the LPR to consist of no more than 385 individuals. Even at the highest hypothetical excess cancer risk of $3.0 \times 10^{-4}$ calculated in this risk assessment (Phase 1, deterministic, child, RME), the population cancer risk associated with this site is 0.12 ($3.0 \times 10^{-4}$ excess cancer risk/hypothetical angler $\times 385$ hypothetical anglers $= 0.12$). Leigh and Hoskin (1999) have conducted similar calculations for evaluating risks to residents residing near hazardous waste sites. This, however, is an unrealistically conservative estimate since the LPR CAS found the vast majority of LPR anglers were adults, with all of those interviewed indicating that their catch was not going to be consumed by children or pregnant females. There was not sufficient information available to Ray et al. (2007b) to estimate the number of annual LPR child anglers, but it was clear that such a population would be minute given that only 1 of 177 potential interviewees was described as “under the age of 15,” and less than 0.5% of the boat based counts tallied throughout the year of study identified individuals that appeared younger than 15. Therefore, a more pragmatic approach to estimating the population risk incorporates the highest adult excess cancer risk estimate (Phase 1, deterministic, adult, RME) with the resulting number of excess cancers associated with this site to be an order of magnitude lower than the Phase 1 child RME cited above (using the most $6.4 \times 10^{-7}$ excess cancer risk/hypothetical angler $\times 385$ hypothetical anglers $= 0.025$). Clearly, the excess cancer risk posed by ingestion of fish from this site is not significant given that not even one excess cancer would be expected from ingestion of fish under current usage conditions.

To put these cancer risk levels into context, they can be compared to the cancer risk from average background exposures to dioxins and dioxin-like compounds. There have been several estimates of background dioxin exposures derived over the past two decades (Furst et al., 1990; Gilman and Newhook, 1991; Travis and Hattemer-Frey, 1991; Henry et al., 1992; Schrey et al., 1995; Schuhmacher et al., 1997; Jacobs and Mobbs, 1997; Liem et al., 2000). However, since environmental levels of these compounds have steadily decreased over time (Aylward and Hays, 2002; Lorber, 2002), only the most recent estimates should be utilized for the purposes of current risk characterization. Lorber et al. (2008) recently calculated a total daily intake of 41 pg TEQ/day, an estimate based upon typical dietary levels (the primary exposure route) as well as other pathways (air, water, soil, and vegetable oil). This is equivalent to a dose of 0.58 pg TEQ/kg day for a 70 kg adult, which corresponds to $9 \times 10^{-5}$ excess cancer risk given a cancer slope of 156,000 (mg/kg day)$^{-1}$. Though this is within USEPA’s acceptable risk range, it is clear that excess cancer risk from background dioxin exposure on the national level is greater than that estimated for a child in the 95th percentile who consumes cooked LPR fish at the LPR consumption rate.

Another comparison can be made to put these cancer risk levels into context. The cancer risk estimates developed in this assessment can also be compared to the dose received by a nursing infant. This is an important comparison given that, despite levels of dioxin-like compounds measured in breast milk, medical professionals continue to recommend breast-feeding. Clearly this would not be the case if such levels were believed to pose an imminent or substantial health threat. In fact, studies have demonstrated that these lipophilic compounds are typically present in breast milk, and exposures are relatively high early in life. Lorber and Phillips (2002) estimated that breastfed infants are exposed to approximately 240 pg TEQ/kg day at birth, and about 170 pg TEQ/kg day by one month. Their work illustrates that exposures are expected to decrease as the child grows and feeding habits change, such that the typical child at one year of age is only receiving a dose of approximately 18 pg TEQ/kg day. Fig. 4 compares the estimates of TEQ intake for breastfed infants and toddlers derived by Lorber and Phillips (2002) with estimated TEQ dose intakes from LPR fish calculated for adult and child receptors in the 50th and 95th percentiles. From this figure it is clear that a one-year old breast fed child is still receiving a greater TEQ dose than that estimated for a child in the 95th percentile risk category who consumes cooked LPR fish (12.1 pg TEQ/kg day), and a more than eight-fold larger dose than the LPR fish-consuming child of the 50th percentile as derived in the PRA of Phase 2 of this risk assessment. Fig. 4 also illustrates the tolerable daily intake (TDI) range for dioxin-like compounds, 1–4 pg TEQ/kg day, established by the WHO (WHO, 2000), and draws attention to the fact that infant exposure to dioxin over two years of breastfeeding greatly exceeds this mark. Conversely,
of the adult and child scenarios evaluated in the Phase 2 PRA of this human health risk assessment, only the child in the highest percentile of cancer risk is exposed to levels of dioxin-like compounds from LPR fish consumption that exceed the TDI (Fig. 4).

4.2. Uncertainties

There are several uncertainties associated with this risk assessment. An important source of uncertainty involves the fish tissue samples utilized in the current study. These samples were collected in 1995–2001 and thus are 7–13 years old and may not represent current conditions in the LPR. On-going site investigation work should result in additional fish tissue chemistry becoming available. Additionally, the CAS was conducted in 2001 and both access and usage of the LPR may have changed.

Another source of uncertainty is related to the characteristics of the LPR angler population. Of all the LPR anglers interviewed along the lower 6 miles of the river, two anglers (or less than 5% of all interviewees), claimed to eat the fish head along with the fish fillet (Ray et al., 2007b). The current risk assessments assumed consumption of the fillet only. Given the extremely small size of the angler population who claimed to eat fish heads, and the lack of relevant fish-head contaminant concentration data on hand, it was not deemed appropriate to attempt to quantitatively assess the “extra” cancer and non-cancer risk imparted to the fish head eaters. It is reasonable to assume that the fish head eaters are confined to adult anglers, as there was no indication that this type of consumption activity could be attributed to children. Furthermore, based on the relatively low 95th percentile excess cancer risk values for adults, it also is reasonable to expect upper risk percentiles for fish head eaters also to be at an acceptable risk level.

Further uncertainty associated with the LPR angler population involves the effect that fishing advisories might have had on the size of the population reported by Ray et al. (2007b). The authors of the LPR CAS asked anglers if they were aware of the fishing advisories during the course of their interviews and determined that only 40% were aware that the advisories existed, and less than 10% indicated that the advisories influenced their fishing habits and/or eating habits (Ray et al., 2007b). Pflugh et al. (1999) conducted a study that evaluated the impact of fishing advisories on angling and consumption activities in nearby areas in Newark Bay and obtained similar results. They reported that although 60% of the anglers were aware of the advisories, these anglers did not appear to be concerned with the health effects presented on the advisories. Of course, neither study was designed to determine the extent to which actual fishing activity was suppressed, as there were no mechanisms in place for determining how many potential anglers decided not to fish based upon the advisories. However, the authors' personal observations in the area of interest are, generally, that the fish advisory signs are not well identified in the field. Moreover, the investigation described herein was designed to address current potential risk to human health due to LPR fish ingestion, whereas the suppression of fishing activities by advisories is a different issue — one that may impact the design and conduct of risk assessments to address future potential exposures.

Indeed, future potential exposures were not addressed in this risk assessment and represent another source of uncertainty associated with assessing potential human cancer and non-cancer health risks posed by ingestion of fish from the LPR. Estimating future cancer and non-cancer health risks needs to take into account, among other factors, future concentrations of chemicals in fish tissue, which can be greatly influenced by such things as sediment remediation activities, on-going discharges to the LPR (including from up-river areas, storm water outfalls, permitted industrial outfalls, and, especially, from combined sewer overflows), and the decline over time of PCDD/Fs, PCBs, and/or other contaminants in environmental matrices. Also, restoration activities conducted under the auspices of the USEPA or the New Jersey Department of Environmental Protection could serve to increase access for anglers to the portion of the LPR addressed by this risk assessment, which, in turn, may impact human health cancer and non-cancer risk estimates.

Finally, the cancer slope factor for 2,3,7,8-TCDD is an important and controversial source of uncertainty that must be discussed in any human health risk assessment where dioxin and dioxin-like compounds are identified as significant contributors to excess cancer risk. The current slope factor of 156,000 (mg/kg day)^−1 was first derived in the 1980s by the USEPA using a linear extrapolation approach based on the presence of hepatocellular carcinomas and adenomas identified on pathology slides from a chronic rat toxicity assay conducted by Dow Chemical (Kociba et al., 1978; USEPA, 1985). Subsequent to its derivation, however, histopathological criteria for proliferative lesions were modified, and reevaluation of these data by an independent panel of pathologists found significantly fewer tumors associated with exposure to 2,3,7,8-TCDD (Goodman and Sauer, 1992). Based on these updated data, Keenan et al. (1991) derived an upper bound cancer slope factor that was 16-fold lower than the previous value. More recently, the National Toxicology Program (NTP) conducted a two-year cancer bioassay for 2,3,7,8-TCDD in rats and reported results similar to those of the pathology reevaluation of the original Dow study (Walker et al., 2006). The State of California has recently proposed a cancer slope factor for 2,3,7,8-TCDD of 26,000 (mg/kg day)^−1 based on the data reported in Walker et al. (2006) and using a linear dose response modeling approach (CAL EPA, 2007). This cancer slope factor was not utilized in this risk assessment as it was draft at the time of preparation of the assessment; however, utilization of the CAL EPA (2007) or Keenan et al. (1991) cancer slope factor would have decreased excess cancer risks by a factor 6 or 7 in this assessment.

Furthermore, the authors of the NTP study point out that the dose response curves are indicative of a nonlinear response for all neoplasms investigated. This is in agreement with the recent recommendations made by the National Academy of Sciences (NAS) expert panel charged with reviewing the USEPA's Dioxin Reassessment. In 2003 the USEPA released a draft version of a report on dioxins that represented a decade's worth of effort to comprehensively describe and summarize the vast literature on exposure, toxicology, and cancer and non-cancer health risks related to dioxins and dioxin-like compounds. Upon reviewing the draft report, the NAS expert panel released its own critique of the Dioxin Reassessment, in which it issued several recommendations for improving the report. Among the recommendations was a call for the USEPA to reevaluate the dioxin cancer slope factor using a nonlinear approach (NAS, 2006). Thus, until this toxicity value has been re-calculated by the regulatory community, human health risk assessments that investigate the carcinogenic health impact of dioxin-like compounds and employ the current 2,3,7,8-TCDD slope factor incorporate a considerable degree of conservatism based upon this factor alone.

The results of this risk assessment of human ingestion of LPR fish demonstrate that PCDD/Fs and PCBs are each responsible for approximately half of the total cancer risk associated with fish consumption for this area of the LPR. Overall, however, we conclude that, under current conditions, ingestion of LPR fish is not resulting in an imminent threat to human health. These results are in contrast to what might be suggested otherwise by use of USEPA’s default exposure assumptions, some of which were derived from waterways that bear little resemblance to the LPR.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.scitotenv.2009.03.004.

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