RISK ASSESSMENT OF POLYCHLORINATED BIPHENYLS
AT HAZARDOUS WASTE SITES

Brian Davis and Michael Wade

Department of Toxic Substances Control

Cal EPA, Sacramento, CA

March, 2003

Professional affiliations are listed for contact purposes only. Analysis and conclusions contained herein are solely those of the authors, and do not represent official policy of the Department of Toxic Substances Control.
ABSTRACT

We recently reported a hazardous waste site in which polychlorinated biphenyls (PCBs) were at such high concentrations in soil that significant levels were found in indoor residential air. This paper uses the waste site to illustrate the complexities in exposure assessment and toxicity assessment of PCBs. Aroclors are commercial mixtures of different arrays of the 209 individual PCB congeners. What is actually found at a given site will be a complex mixture of individual congeners, the exact composition depending on the makeup of the original material released at the site and the subsequent environmental fate and transport of released material. Recently, individual congener analysis has begun supplementing the reporting of Aroclors. Unfortunately, long term toxicity studies, and hence toxicity criteria, are only available for Aroclors rather than individual congeners. The PCB mixtures at a waste site may be quite different than the mixture on which toxicity criteria are based. U.S. EPA currently provides upper bound cancer slope factors of 2.0 for High Risk and Persistence PCBs, 0.4 for Low Risk and Persistence PCBs, and 0.07 for Lowest Risk and Persistence PCBs, based on bioassays of Aroclors 1260, 1254, 1242, and 1016. Although the soil contaminant at our hazardous waste site was identified as Aroclor 1260, 98% of the PCBs detected in indoor air were monochlorinated biphenyls. A further complication in toxicity assessment is that noncancer toxicity can result from relatively low dose levels. For example, all doses of Aroclor 1016 predicting a cancer risk above 5E-6 are also associated with an unacceptable noncancer hazard. Another consideration for toxicity assessment is the dioxin-like activity of co-planar PCBs.

Exposure assessment of PCBs is complicated by the bioaccumulative properties. This necessitates consideration of the potential for indirect exposure to PCBs from the waste site through food pathways, including fish, poultry and livestock, and breast milk. Another exposure assessment consideration is the near ubiquitous ambient low level presence of PCBs in environmental media including air, soil, and food.

INTRODUCTION

While undertaking risk assessment at a hazardous waste site where multifamily housing was constructed over soil contaminated with polychlorinated biphenyls (PCBs), we became aware of a number of complex issues in PCB risk assessment including contaminant identification, fate and transport, ambient levels, exposure pathways and regulatory criteria. The path one follows in undertaking PCB risk assessment is complex and ambiguous. This poster identifies issues, complexities and data gaps that make PCB risk assessment challenging. We hope it serves to facilitate thought and discussion, if not illumination, concerning this topic.

FATE AND TRANSPORT OF PCBs

1. Variability in the physical and chemical properties of different PCB congeners results in variable behavior in the environment.
2. Volatility and mobility in the atmosphere increase with decreasing chlorination. Atmospheric transport is an important mechanism for worldwide dispersion.
3. PCBs enter water bodies from water channels and atmospheric deposition. PCBs leave water bodies by volatilization. PCBs are exchanged between the water column and sediments.
4. PCBs strongly sorb to soils, limiting mobility.
5. Rates of photochemical degradation in the atmosphere decrease with increasing chlorination. Half-lives of PCB congeners in soils and sediments are on the order of months and years.
7. Bioaccumulation is discussed in the panel on breast milk and fish.

CASE STUDY OF SUBCHRONIC EXPOSURE IN RESIDENTIAL SETTINGS

Example: A family housing area in southern California had low-level, widespread PCB contamination with values as high as 8 mg per kg of soil. The responsible party based its risk assessment on a four year residency. Since the excess cancer risk level to residents (using the high risk and persistence cancer slope factor) fell within the 1E-6 to 1E-4 range, it was proposed that no action was needed. However, assessment of potential, noncancer toxicity showed that a concentration of 1 mg/kg produced a hazard quotient of one. This finding was a major factor in the decision to remediate the site. It was especially important at this site since young families were present. Fetuses and children make up sensitive populations. Breast feeding is also of concern in residential areas (see the panel on breast feeding).

Conclusion: Because non-cancer toxicity can occur at relatively low levels of PCBs, it must always be considered.
For many hazardous chemicals, considerations of carcinogenicity far outweigh noncancer toxicity. Evaluation of PCBs should take into account all potential toxicity. The only noncancer toxicity criteria for PCBs listed on U.S. EPA Integrated Risk Information System are oral reference doses for Aroclor 1016 and Aroclor 1254. This table shows dose estimates which generate cancer risk estimates in the range of 1E-6 to 1E-4 may have a significant potential for other toxicity. The range in the hazard quotients results from applying the central tendency and upper bound cancer slope factors.

**CASE STUDY OF “WEATHERED” AROCLORS**

Weathered Aroclors may have a markedly different congener makeup than the original Aroclor products used in the bioassays on which toxicity criteria are based.

Example: We have previously reported (Davis et al., 2002) a residential area in California with widespread PCB contamination. Prior to a removal action, concentrations of Aroclor 1260 were as high as 160,000 mg per kg of soil. The levels and distribution of the contamination suggested the possibility that high levels of PCBs might underlie the slab foundation of at least one of the multi-unit buildings. Indoor air concentrations of PCBs in different units were correlated with concentrations found in soil around the building. The highest concentrations were 96 to 234 ng of total PCBs per m³ of air. Monochlorinated congeners were by far the most prevalent in all of the units (95-98%). In spite of this, congeners with more than four chlorines exceeded 0.5% of the total. Therefore, the indoor air failed the criterion for applying the cancer slope factor for lowest risk and persistence (see the panel on cancer slope factors). Soil samples obtained at depth contained up to 38% monochlorinated PCBs. These samples were taken after the earlier soil removal and are clearly not Aroclor 1260.

**Congener Composition of Site PCBs and Aroclors**

<table>
<thead>
<tr>
<th>Congener Composition of Site PCBs and Aroclors</th>
<th>Indoor Air</th>
<th>Deep Soil</th>
<th>Aroclor 1016</th>
<th>Aroclor 1260</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monochlorinated</td>
<td>95-98%</td>
<td>38%</td>
<td>&lt; 1%</td>
<td>&lt; 0.1%</td>
</tr>
<tr>
<td>5 or more chlorines</td>
<td>1-3%</td>
<td>1%</td>
<td>0.6%</td>
<td>&gt;99%</td>
</tr>
</tbody>
</table>
Conclusions: Indoor air contamination should be evaluated at sites with high concentrations of PCBs in soil or ground water. Application of cancer slope factors, derived from Aroclor bioassays, to PCB mixtures in the environment entails a great deal of uncertainty.

<table>
<thead>
<tr>
<th>Population Studied</th>
<th>Maternal Blood (wet basis)</th>
<th>Breast Milk (lipid basis)</th>
<th>Health Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mich nonfish-eaters</td>
<td>4.1 ppb</td>
<td>622 ppb</td>
<td>Birth weight, head circumference, gestational length, neurobehavior</td>
</tr>
<tr>
<td>Mich fish-eaters</td>
<td>6.1 ppb</td>
<td>866 ppb</td>
<td>Neurobehavior</td>
</tr>
<tr>
<td>North Carolina</td>
<td>9.1 ppb</td>
<td>1,800 ppb</td>
<td>Neurobehavior</td>
</tr>
</tbody>
</table>

These selected examples from ATSDR (2000) illustrate PCB bioaccumulation. (1) The 50% increase in blood levels of PCBs with fish consumption demonstrates the significance of food, particularly fish, as a source of PCB. Bioconcentration of PCB congeners from water to aquatic organisms can be as high as $2 \times 10^6$. (2) Concentrations of PCBs in breast milk are even higher because of the high lipid content. (3) There is a strong relationship between increasing PCB levels in maternal blood and increasing PCB levels in breast milk.

<table>
<thead>
<tr>
<th>Population studied</th>
<th>Dose [mg/(kg x day)]</th>
<th>Hazard Quotient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Michigan nonfish-eaters</td>
<td>0.09</td>
<td>1200-4300</td>
</tr>
<tr>
<td>Michigan fish-eaters</td>
<td>0.12</td>
<td>1700-6000</td>
</tr>
<tr>
<td>North Carolina</td>
<td>0.25</td>
<td>3600-12,500</td>
</tr>
</tbody>
</table>

Based on the PCB concentrations in breast milk, doses to infants were estimated using the equation and assumptions from CAPCOA (1993). We assumed one year of exclusive, daily breast feeding. Hazard quotients were based on oral reference doses of $7 \times 10^{-5}$ mg/(kg x day) for Aroclor 1016 and $2 \times 10^{-5}$ mg/(kg x day) for Aroclor 1254. These Aroclors are unlikely to be representative of the PCBs in breast milk, demonstrating the need for toxicity data for PCB congeners. Furthermore, the reference doses are based on chronic exposures. On the other hand, infants from birth to one year are likely to have far greater sensitivity than the general population. These results should not be interpreted as an argument against breast feeding.

<table>
<thead>
<tr>
<th>Population studied</th>
<th>Dose [mg/(kg x day)]</th>
<th>Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Michigan nonfish-eaters</td>
<td>0.001</td>
<td>9E-5 to 3E-3</td>
</tr>
<tr>
<td>Michigan fish-eaters</td>
<td>0.002</td>
<td>1E-4 to 3E-3</td>
</tr>
<tr>
<td>North Carolina</td>
<td>0.004</td>
<td>3E-4 to 7E-3</td>
</tr>
</tbody>
</table>

This table shows lifetime doses, derived from the previous table by applying an Averaging Time of 70 years. The resulting risk estimates are based solely on PCBs from one year of nursing and do not account for additional exposure during the remainder of the lifetime. The risk range is based on the lowest and highest U.S. EPA cancer slope factors, 0.07 and 2 [mg/(kg x day)]$^{-1}$. The Aroclors on which the slope factors are based are unlikely to be representative of the PCBs in breast milk, demonstrating the need for toxicity data for PCB congeners. These results should not be interpreted as an argument against breast feeding.

CHEMISTRY AND USES OF PCBs

Polychlorinated biphenyls (PCBs) are biphenyl molecules linked by a carbon-carbon bond at the 1-1' position. The other 10 positions on the phenyl rings are substituted with 1-10 chlorine atoms, resulting in 10 isomer families (mono-, di-, tri-, etc.). There are 209 individual PCB congeners. Aroclors are industrial mixtures of PCBs, differing in level of chlorine substitution. Aroclor 1260 contains 60 percent chlorine by weight.
PCB manufacture began about 1930. Because of their heat stability, low reactivity and lubricating properties, PCBs had a variety of industrial uses (electrical transformers and capacitors, hydraulic fluid, paints, inks, adhesives, paper products, etc.). PCBs were banned in the U.S. in 1977, because of their bioaccumulation, persistence in the environment, and toxicity. Current contamination at many hazardous waste sites demonstrates the persistence of PCBs.

![Chemical structures of PCBs](image)

**ANALYTICAL CHEMISTRY**

1. Aroclor-based methods:
   a. Extracts of environmental samples are analyzed by gas chromatography with electron capture detection (GC/ECD). Aroclor category determined by the best match of peaks to Aroclor reference standards, i.e. Aroclor 1260, 1254, etc.
   b. Complexities in interpretation of environmental samples:
      i. More than one Aroclor may be present.
      ii. Each Aroclor batch is unique in its exact congener composition.
      iii. Weathering with individual congeners being selectively degraded, metabolized, or transported elsewhere.
2. Congener analysis: Individual congeners are quantitated using either newer GC/ECD methods or combined gas chromatography/mass spectrometry (GC/MS).
3. Quantitation limits for Aroclor method about 36 ug/kg; congener analysis by GC/MS as low as 0.005 ug/kg.

**DIOXIN-LIKE PCBs**

PCBs lacking two chlorines in the ortho position are called “coplanar” congeners. Compare coplanar PCB126 and non-coplanar PCB99 in the panel on chemical structure. Coplanar PCBs stereochemically resemble dioxins and bind to the Ah receptor with a relatively high affinity. In general, coplanar congeners exhibit enzyme induction and certain other toxic effects indicative of Ah receptor-binding. In the World Health Organization 1998 list of Dioxin Toxicity Equivalency Quotients (TEQs), PCB congener TEQs range from 0.00001 to 0.1, compared to the maximum TEQ of 1.0 assigned to 2,3,7,8 TCDD. In fish samples recently obtained from San Francisco Bay, approximately 80% of the total dioxin TEQ was due to PCBs, with about half of that from one congener, PCB126 (see the panel on chemistry).

How should dioxin-like PCBs be evaluated in risk assessment? Congener analysis for PCBs is lacking at most hazardous waste sites and PCB risks are assessed using the assigned Aroclor slope factors and reference doses. In the absence of a full understanding of the mechanism or mechanisms of PCB carcinogenicity, it is unclear whether this is the best way to evaluate cancer risks.
AMBIENT LEVELS

1. Although production of PCBs halted 25 years ago, low levels are still found in air, water, sediment, soil and food.
2. Typical air concentrations range from 0.1 to 3 ng/m$^3$. Levels are declining. Urban areas have higher levels. Indoor levels may be higher than outdoors.
3. Despite low solubility, PCBs are widespread in water bodies. Typical levels of PCBs in the ocean and the Great Lakes are 0.02 to 0.6 ng/L.
4. Storm water runoff and rainwater are commonly contaminated with PCBs. Rainwater concentrations from 1980-1990 were in the range of 0.1 to 20 ng/L.
5. PCB levels in rivers, lakes and ocean sediments vary widely but are highest in areas receiving runoff from contaminated sites.
6. PCBs have been reported in many foods, most notably fish. A recent study in San Francisco Bay reported median values in seven fish species ranging between 13-306 ng/g of wet tissue, with one species non-detect.

INTERACTIONS WITH OTHER CHEMICALS

Since Aroclors are mixtures and since Aroclors have several different biological activities, they are expected to have toxic interactions with other chemicals. Indeed, Aroclors are routinely used to increase the sensitivity of a variety of bioassays by inducing hepatic enzymes which alter the metabolism of other chemicals and hence alter their toxicity.

**Documented PCB Interactions:**
- Other PCB congeners
- Chlorinated Dibenzo-p-Dioxins and Furans
- Polybrominated Diphenyl Ethers
- Methylmercury
- Pesticides
- Metals
- Viral infections

**Mechanisms of Toxicity:**
- Ah-receptor mediated
- Ah-receptor independent
- Involving the Ah-receptor and other mechanisms
- Estrogen receptor mediated
- Thyroid levels in fetus/infant

CONCLUSIONS

1. Declining but widespread, low-level PCB contamination is present throughout the environment.
2. Higher levels of PCB contamination are associated with many former manufacturing and industrial sites.
3. Complications and inconsistencies in PCB risk assessment may arise:
   a. PCBs are congener mixtures that vary from batch to batch and “weather” at different rates. Congener makeup of environmental samples may be very different than the mixture for which toxicity criteria were determined.
   b. PCBs have several different toxicity mechanisms with different risk assessment methodologies, i.e. “dioxin-like” activity as compared to Aroclor carcinogenicity and toxicity.
   c. The traditional focus on carcinogenicity may result in overlooking potential noncarcinogenic PCB toxicity. Noncarcinogenic toxicity can be significant at doses within the “risk range” for carcinogenicity.
   d. Bioaccumulation of PCBs can lead to significant exposure via pathways such as breast milk, which may be overlooked in conventional risk assessment paradigms.
4. Congener analysis is informative, but appropriate risk assessment for congeners remains uncertain.
5. The potential for intrusion of PCBs into indoor air should be considered.
6. Residual, ambient levels of PCBs, particularly in urban areas, are confounding factors.
7. A fresh approach is needed for PCB risk assessment. We suggest an inclusive, overarching framework with attention to such elements as congener makeup, consideration of all relevant exposure pathways, and accounting for all modes of toxicity.
8. Such a framework may require development of new information such as:
   a. New sampling and analytical protocols.
   b. Toxicity data for selected individual congeners and representative weathered PCB fractions.
   c. More information on non-standard exposure pathways such as breast milk.
   d. Additional information on body burden and “ambient” exposure to PCBs from all sources.
   e. Interaction of PCBs with other chemicals.
REFERENCES


DISCLAIMER

The opinions and findings in this paper are those of the authors. They do not represent guidance or policy of the California Department of Toxic Substances Control or California Environmental Protection Agency.