Contribution of PCB exposure from fish consumption to total dioxin-like dietary exposure

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Abstract

Polychlorinated biphenyls (PCB) are the second greatest cause of fish advisories, and are often the greatest contributors to dioxin-like toxic equivalency (TEQ) in fish and seafood. Because fish consumption is associated with both contaminant risks and health benefits, incremental health risks from PCBs in fish should be considered within the context of overall TEQ associated dietary risk to enable consumers to make informed decisions about choosing to eat fish or alternate foodstuffs. In this paper, potential TEQ exposure from PCBs in fish for adults with a variety of consumption patterns and consuming fish from a variety of sources are calculated using recent consumption and fish contaminant data from the literature and compared to total TEQ exposure from all sources. For high-level consumers and individuals eating fish from relatively contaminated sites, PCB TEQ exposure from fish consumption alone may exceed the 1 pg TEQ/kg/day average adult daily intake estimated by EPA, which itself carries an upper bound cancer risk of 1 in 1000. PCB TEQ risk for average consumers of commercial fish is expected to be far less, but is highly uncertain, since there is a dearth of congener specific PCB data for commercial fish and seafood.

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Keywords: TEQ; PCBs; Fish consumption; Risk assessment

1. Introduction

Polychlorinated biphenyls (PCBs) are the second greatest cause of US fish advisories (USEPA, 2003). PCBs are chemically stable mixtures that were manufactured and marketed domestically as “Aroclor” mixtures until the mid-1970s, primarily for use in transformers and capacitors. Their widespread use and environmental release through disposal and spillage has resulted in contamination of freshwater fish and seafood (Erickson, 1997). In epidemiological studies, PCBs have been associated with immunotoxicity (Svensson et al., 1994), neurobehavioral deficits (Jacobson and Jacobson, 1996; Patandin et al., 1999; Schantz et al., 2001), reduced birth weight (Patandin et al., 1998; Rylander et al., 2000), and cancer (Svensson et al., 1995; Loomis et al., 1997). PCBs are classified by EPA as probable human carcinogens (USEPA, 1997d). Some groups such as Native Americans, and Asian and Pacific Islanders (API) may consume 10 times as much or more fish and seafood (CRITFC, 1994; Sechena et al., 1999; Toy et al., 1996) than average US citizens (Jacobs et al., 1998; USEPA, 1997b) (Table 1). This greater consumption may mean greater exposure and risk from contaminants in fish such as PCBs.

Some PCBs may adapt a planar conformation and activate the aryl hydrocarbon (Ah) receptor. These PCBs are thought to share a common mode of toxic action with dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin, TCDD) (Van den Berg et al., 1998). Some polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are also capable of activation of the Ah receptor. Toxic equivalency factors (TEFs) provide estimates of the toxicity of dioxin-like PCBs, PCDDs, and PCDFs based on their activities relative to TCDD. PCDDs and PCDFs have been introduced into the environment from industrial activities, combustion, and as contaminants in PCB mixtures (PCDFs only) (Erickson, 1997; Hays and Aylward, 2003).
TEFs provide a system for summing exposures to dioxin-like PCBs, PCDFs, and PCDDs. The final exposure values are referred to as toxic equivalency quotients (TEQs). In this system, an exposure to a particular PCB, PCDD, or PCDF is multiplied by its TEF to convert it to an equivalent amount of TCDD exposure (based on toxicity). For example, if we have an exposure to a certain amount of a PCB congener measured in pg then we would multiply it by the appropriate TEF to express the exposure in pg TEQ. This is approximately equivalent to the amount of TCDD to cause similar toxicological effects. Alternately, if exposure is measured as pg/g fish, or pg/kg human body weight then multiplication of the compound being measured by the TEF expresses the exposure in pg TEQ/g fish, or pg TEQ/kg human body weight. From this, we can then consider exposure to all PCBs, PCDDs, or PCDFs where:

\[
\text{PCB TEQ} = \sum_i \text{PCB}_i \times \text{TEF}_i
\]

\[
\text{PCDD TEQ} = \sum_i \text{PCDD}_i \times \text{TEF}_i
\]

\[
\text{PCDF TEQ} = \sum_i \text{PCDF}_i \times \text{TEF}_i
\]

We can then define total TEQ exposure as:

\[
\text{total TEQ} = \text{PCB TEQ} + \text{PCDD TEQ} + \text{PCDF TEQ}
\]

Estimates of TEQ exposure can be used in risk equations with the same toxicity metrics (e.g., cancer slope factor) as would be used for TCDD exposure. There is particular concern about potential cancer risk from TCDD and dioxin-like chemicals. This is due in part to recent estimates of TCDD cancer slope factor (1E-3 pgTEQ/kg body weight/day) which are 10 times higher than previous estimates (USEPA, 2000). EPA’s estimate of general population total dioxin TEQ exposure (including dioxin-like PCBs, PCDFs, and PCDDs) of 1 pg/kg/day is within the range of estimates presented in other assessments (Alcock et al., 1998; Patandin et al., 1999; Schecter et al., 1994) and may carry an upper bound cancer risk of 1 in 1000 (USEPA, 2000). The tolerable daily intake (TDI), a reference value for ensuring that acceptable levels of exposure are not exceeded, determined by the World Health Organization for dioxin TEQ in 1998 is 1–4 pg/kg body weight (WHO, 1999). The USEPA has not released a reference dose (RfD) despite suggestions from the Science Advisory Board that they do so (Paustenbach, 2002). The levels of PCDDs, PCDFs, and PCBs in foodstuffs have dropped several fold (on a TEQ basis) since the 1970s (Hays and Aylward, 2003), but these chemicals are highly persistent and exposure continues. Many estimations of TEQ exposure do not include PCB TEQ exposure from fish at all (sometimes only including PCDD and PCDF TEQ exposure from fish) (Jensen et al., 2000) or do not account for the wide range of consumption patterns and sources of seafood, which may have significant effects on exposure and risk estimates (Judd et al., 2002, 2003a,b).

The exposure contribution from consumption of fish and seafood has been estimated as less than 20% of dietary total TEQ exposure for the general population (Travis and Hattemer-Frey, 1991; Schecter et al., 1994; Harrison et al., 1998). However, PCB TEQ exposure from fish may be much greater for people with above average fish and seafood consumption or consuming highly contaminated fish. Estimating TEQ exposure from fish or seafood is difficult, due to a dearth of sufficiently sensitive congener specific PCB data (Dyke and Stratford, 2002; Judd et al., 2003a). Many studies either do not test for or report the most potent dioxin-like congener, PCB 126, as not detected (Madenjian et al., 1999; Serrano et al., 2000; Ylitalo et al., 1999). The analytical limits of detection in many studies may not be low enough to assess potential health risks of concern (Judd et al., 2003a), so these non-detect data are of limited use for health risk assessment.

There remains much speculation about the appropriateness of focusing PCB risk assessments on the dioxin-like congeners (those with TCDD-TEQs), given the potential for toxicity due to non-dioxin-like PCBs (Fischer et al., 1998). However because exposure to dioxin-like PCBs raises risk concerns at lower levels than for total PCB or Aroclor exposure (the only currently

<table>
<thead>
<tr>
<th>Reference</th>
<th>Population</th>
<th>Mean</th>
<th>50th</th>
<th>90th</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sechena et al. (1999)</td>
<td>1st and 2nd generation Asian and Pacific Islanders</td>
<td>117</td>
<td>89</td>
<td>242</td>
</tr>
<tr>
<td>Jacobs et al. (1998)</td>
<td>US, contiguous 48 states</td>
<td>20a</td>
<td>NR</td>
<td>67a</td>
</tr>
<tr>
<td>CRITFC (1994)</td>
<td>Umatilla, Nez Perce, Yakama and Warm Springs-Tribes of the Columbia River region</td>
<td>59</td>
<td>30</td>
<td>171b</td>
</tr>
<tr>
<td>Connelly et al. (1996)</td>
<td>Lake Ontario Anglers</td>
<td>18</td>
<td>14</td>
<td>34</td>
</tr>
<tr>
<td>Murray and Burmaster (1994)</td>
<td>Michigan Anglers</td>
<td>45</td>
<td>33</td>
<td>81</td>
</tr>
</tbody>
</table>

NR–not reported.
a Estimate of raw consumption, assumes 22% loss in weight from cooked weight estimate.
b 95th percentile.
accepted risk assessment methods to address the toxicity of non-dioxin-like PCBs) (Williams et al., 1992; EVS, 2000; USEPA, 2002; Judd et al., 2003b), this study focuses on the consequences of this emphasis on PCB TEQ exposure from fish and seafood in the context of other TEQ exposure.

The primary aim of this study is to compare different estimates of PCB TEQ exposure for adults with various consumption rates and eating fish from different sources to average total TEQ exposure. Data on concentrations of dioxin-like PCBs in fish and seafood (Table 2) provide the opportunity to explore potential PCB TEQ exposure from fish and seafood for a range of consumption rates based on recent studies (Table 1). PCB TEQ estimates are compared to EPA’s recent estimate of overall average TEQ exposure. These considerations are important from a risk management perspective in light of the fact that PCB contamination often leads to advisories against eating an otherwise highly beneficial low fat, high protein, and often low cost food. Consumption of fish is also an important part of the cultural traditions of many groups.

2. Methods

To explore PCB TEQ exposure from fish across different consumption patterns, data on both consumption and PCB TEQ concentration in fish and seafood was identified. To use these environmental data to assess exposure, the data were evaluated to ensure that they were appropriate in terms of analytical sensitivity, sample size, and compatibility with consumption for the consumer groups considered. To provide context for the potential risk from these PCB TEQ exposure estimates, they were compared to estimates of TEQ exposure for the general population.

2.1. Considerations for estimation of PCB TEQ exposure for high seafood consuming groups

Individuals consuming high levels of contaminated fish and seafood would be expected to have greatest exposures. Recent fish and seafood consumption studies have highlighted high consuming groups including anglers, Asian and Pacific Islanders, and Tribal nations whose consumption exceeds the US average (Jacobs et al., 1998; USEPA, 1997b) (Table 1). The sources of fish consumed by these groups varies greatly, with the example angler groups eating primarily from the Great Lakes (Connelly et al., 1996; Murray and Burmaster, 1994), which are significantly contaminated with PCBs (Giesy et al., 1997). The Tribal groups in the Pacific Northwest listed in Table 1 consume much more fish, mainly salmon and shellfish caught in rivers or Puget Sound, which tend to be less contaminated than fish from the Great Lakes (CRITFC, 1994; Toy et al., 1996; USEPA, 2002). The API included in the survey eat primarily commercially caught shellfish which may come from all over the world (Sechena et al., 1999) and are subject only to FDA monitoring which has a very high limit of quantitation for PCBs (0.2 ppm) (FDA, 2001).

2.2. Calculation of PCB TEQ exposure

Exposure from consumption of fish or seafood was calculated by multiplying the consumption rate by the concentration of contaminants in the fish or seafood. Thus, a combination of the highest consumption rate and fish or seafood with highest contaminant concentration would yield the highest exposure estimate. The PCB TEQ exposure from fish and seafood from various sources (Table 2) for different consumption rates (Table 1) were calculated in units of pg TEQ/kg body weight/day (as described in Section 1). In addition to comparing potential PCB TEQ exposure from fish to estimates of general population dietary total TEQ exposure, the potential influence of non-detect values (for dioxin-like PCBs) and selection of greater and less contaminated fish on PCB TEQ exposure estimates was also investigated.

For this study, which focuses on adult exposure, body weight was always assumed to be 70 kg (USEPA, 1997a) unless otherwise specified. The exposure calculation was run using data from several fish studies. Previous modeling efforts indicate that analytical sensitivity of five parts per trillion for dioxin-like PCB congeners or better may be needed to assess an upper bound cancer risk of 1 in 10,000 (Judd et al., 2003a,b). Publicly available studies with this level of sensitivity and/or a majority of values above detection the limit for the dioxin-like congeners are limited; examples are presented in Table 2. Information was available for commercial freshwater and ocean fish (Schechter et al., 1994), for Willamette River (Oregon) fish (EVS, 2000), Columbia River Basin (Washington and Oregon) fish (USEPA, 2002), and for fish from Saginaw Bay (Michigan) (Giesy et al., 1997). PCB TEQ concentrations in fish samples had to be recalculated for the commercial (Schechter et al., 1997) and Saginaw Bay (Giesy et al., 1997) studies because consensus TEFs were established (Van den Berg et al., 1998) after their publication. For example, at the time of publication of these studies there were TEFs for congeners and 170 and 180 but today these congeners are no longer considered to contribute to TEQ. Different consensus TEFs were adopted for several other coplanar congeners as well at a WHO workshop in 1998 (Van den Berg et al., 1998). Recalculated PCB TEQ exposure estimates from fish consumption were compared to EPA’s estimate of 1 pg/kg/day total TEQ exposure (from dioxin-like PCBs, PCDFs, and PCDDs).
maximum and minimum exposure estimates were made. Ples were small and variability was high in these studies, more sensitive, but the fish sampled varied widely in the Lumbia River Basin data, analytical methods were much relatively low TEFs (Van den Berg et al., 1998). Another model for the current study focused on the total TEQ from commercial fish (Schecter et al., 1997) for various rates of fish consumption. The total TEQ was recalculated using consensus TEFs (Van den Berg et al., 1998) for all dioxin-like PCBs, PCDDs, and PCDFs (as was done to recalculate PCB TEQ and described in Section 2.2). This allowed the TEQ exposure assuming consumption of the most or least contaminated species. While this is not expected to reflect true consumption (which would likely include a variety of fish), it is useful to bracket a range of possible PCB TEQ exposures. In particular, it may help identify where even modest consumption may lead to exposures of concern. In the Columbia River Basin and Willamette River studies, EPA Method 1668A was used for analysis which is more accurate than many previous methods. Previous analyses by our group indicate this is a better method for congener analysis based on its analytical sensitivity and inclusion of key PCB congeners compared to other available methods (Judd et al., 2003a). This method does not target PCB 81 which has a low TEF (0.0001) (Van den Berg et al., 1998), so this congener is not included in the PCB TEQ estimates for these two studies (Table 2).

2.3. Calculation of total TEQ exposure

Although the contaminant studies reported primarily detectable values for dioxin-like PCBs, there were a significant number of non-detect values in the Schecter studies of two commercial fish composites: 8.3% of freshwater sample and 25% of ocean sample dioxin-like PCBs were non-detects and not all dioxin-like PCBs were included in the analysis (Table 2) (Schecter et al., 1997). To investigate the potential influence of non-detect (ND) values on PCB TEQ estimates, the exposure calculation was run using two estimates of commercial freshwater fish and seafood PCB TEQ (Table 2). In one calculation, it was assumed that concentrations of ND values of dioxin-like congeners were zero and once it was assumed that ND values were equal to the limit of detection (LOD). This was intended to span the range of possible PCB TEQ concentrations. It was not possible to account for congeners not included in the original analysis (PCB 123, 156, 157, and 189), although these are expected to be minor PCB TEQ contributors based on other studies (EVS, 2000; USEPA, 2002) and their relatively low TEFs (Van den Berg et al., 1998).

For the Willamette River, Saginaw Bay, and Columbia River Basin data, analytical methods were much more sensitive, but the fish sampled varied widely in the concentration of PCB congeners present. Because samples were small and variability was high in these studies, maximum and minimum exposure estimates were made

<table>
<thead>
<tr>
<th>Fish source (ref.)</th>
<th>Estimate</th>
<th>PCB TEQ pg/g</th>
<th>Sample notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Columbia River Basin (USEPA, 2002)</td>
<td>Maximum</td>
<td>5.3&lt;sup&gt;e&lt;/sup&gt;</td>
<td>Mountain whitefish, FS avg. (n = 13 composites), ND = LOD</td>
</tr>
<tr>
<td></td>
<td>Minimum</td>
<td>0.18&lt;sup&gt;b,d&lt;/sup&gt;</td>
<td>Walleye, FS, avg. (n = 3 composites), ND = 0</td>
</tr>
<tr>
<td>Willamette River, OR (EVS, 2000)</td>
<td>Maximum</td>
<td>4.5&lt;sup&gt;e&lt;/sup&gt;</td>
<td>Pikeminnow, WB (n = 1 composite)</td>
</tr>
<tr>
<td></td>
<td>Minimum</td>
<td>0.30&lt;sup&gt;b,f&lt;/sup&gt;</td>
<td>Sucker, FS (n = 1 composite), ND = 0</td>
</tr>
<tr>
<td>Saginaw Bay, MI (Giesy et al., 1997)</td>
<td>Maximum</td>
<td>107.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Walleye, WB (n = 1 composite)</td>
</tr>
<tr>
<td></td>
<td>Minimum</td>
<td>30.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Gizzard shad, WB (n = 1 composite)</td>
</tr>
<tr>
<td>Ocean, commercial (Schecter et al., 1997)</td>
<td>ND = LOD</td>
<td>0.25&lt;sup&gt;b,i&lt;/sup&gt;</td>
<td>Mixed seafood, fish fillets and shellfish (n = 1 composite), ND = LOD</td>
</tr>
<tr>
<td></td>
<td>ND = 0</td>
<td>0.13&lt;sup&gt;b,i&lt;/sup&gt;</td>
<td>Mixed seafood, fish fillets and shellfish (n = 1 composite), ND = 0</td>
</tr>
<tr>
<td>Freshwater, commercial (Schecter et al., 1997)</td>
<td>ND = LOD</td>
<td>0.98&lt;sup&gt;b,i&lt;/sup&gt;</td>
<td>Fish fillets (n = 1 composite), ND = LOD</td>
</tr>
<tr>
<td></td>
<td>ND = 0</td>
<td>0.31&lt;sup&gt;b,i&lt;/sup&gt;</td>
<td>Fish fillets (n = 1 composite), ND = 0</td>
</tr>
</tbody>
</table>

<sup>a</sup> LOD; limit of detection; ND, compound was not detected; WB, whole body; FS, fillet with skin.
<sup>b</sup> Does not include PCB 81. Although this compound does have a WHO TEF; it was not included in EPA Method 1668A which was used for this analysis.
<sup>c</sup> For non-detected (ND) PCB congeners, the detection limit was used. This increased the estimated PCB TEQ by less than 3% compared to counting ND = 0.
<sup>d</sup> For non-detected (ND) PCB congeners, zero was used. This decreased the estimated PCB TEQ by less than 30% compared to counting ND = LOD.
<sup>e</sup> For non-detected (ND) PCB congeners, zero was used. This decreased the estimated PCB TEQ by less than 30% compared to counting ND = LOD.
<sup>f</sup> All PCB congeners tested were detected.
<sup>g</sup> PCB 126 was not detected; zero was used for this value. This decreased the estimated PCB TEQ by less than 50% compared to counting ND = LOD.
<sup>h</sup> All PCBs with a WHO TEF were detected.
<sup>i</sup> Sample PCB TEQs were recalculated using 1998 WHO TEFs which were not available when these studies were published.
<sup>j</sup> Sample was not analyzed for PCBs 123, 156, 157, 167, or 189.

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from PCBs relative to TEQ exposure from PCDDs and PCDFs in fish to be considered. Again these estimates were made across a range of consumption values and were compared to EPA’s 1 pg/kg/day estimate of total TEQ exposure. For example, if the total TEQ estimated from fish was 0.5 pg/kg/day for high levels consumers of freshwater fish, this would be equal to half of EPA’s estimate of total TEQ exposure from all sources indicating freshwater fish as a significant exposure source for this group. This estimate could also be compared to PCB TEQ exposure alone for the same exposure group (as was calculated per the method described in Sections 1 and 2.2). This is important because some recent efforts have only analyzed samples for PCDFs and PCDDs in fish (Jensen et al., 2000), without analyzing for dioxin-like PCBs, which may contribute significantly to TEQ concentrations in the fish and therefore to TEQ exposure for consumers.

3. Results

The results of PCB TEQ exposure estimation from consumption of commercial freshwater and ocean fish across a range of consumption rates are presented in Fig. 1. As discussed in 2, exposure was calculated assuming non-detect values for PCB congeners were equal to zero or the limit of detection (Table 2). EPA’s estimate for average total TEQ exposure is also presented for comparison. Based on the limited available samples of commercial ocean and freshwater fish, the levels of potential exposure and the uncertainty (resulting from non-detect values for some PCB congeners) about PCB TEQ exposure from freshwater fish may be much greater than for ocean fish. For mean US consumption of ocean fish (14 g/day adjusted from Jacobs et al., 1998 cooked weight estimate), PCB TEQ from commercial fish is 2.6–5.0% of estimated US average total TEQ. The exposure estimates bracket a range of values by assuming non-detect data for seafood contaminants were equal to zero or the detection limit. Similarly, mean freshwater fish consumption (6.0 g/day, adjusted from Jacobs et al., 1998 cooked weight estimate) is 2.7–8.4%. However, for high-level consumers of ocean fish (105.2 g/day, API mean for seafood and average body-weight of 62 kg from Sechena et al., 1999), PCB TEQ from commercial fish is 22–42% of estimated US average total TEQ. Similarly, for high-level consumers of freshwater fish (39.3 g/day, 95th percentile adjusted uncooked weight from Jacobs et al., 1998), PCB TEQ is 17–56% of estimated US average total TEQ.

Fig. 2 presents estimates of PCB TEQ exposure from regional freshwater fish, based on maximum and minimum PCB TEQ values from available samples (Table 2), for various consumption rates. Fish from Saginaw Bay are much more contaminated than those from the Willamette and Columbia Rivers. PCB TEQ concentration estimates for maximally contaminated fish from Saginaw Bay were 20 times higher than for the maximally contaminated fish from the Willamette or Columbia Rivers (Table 2). Consuming about 0.65 g/day of the maximally contaminated fish or under 2.5 g/day of the minimally contaminated Saginaw Bay fish (Giesy et al., 1997) would lead to exposure equal to 1 pg/kg/day. This would represent occasional consumption and is many times less than may be consumed by anglers (mean 18–45 g/day, Table 1) (Falk et al., 1999). Fish sampled from the Columbia River Basin and Willamette River were less contaminated, but the level of contamination was quite variable (EVS, 2000). To achieve 1 pg/kg/day, only 15.5 and 13.2 g/day of the maximally contaminated fish or 233 and 388 g/day of the minimally contaminated fish from the Willamette River and Columbia River respectively would need to be eaten. Thus for anglers and Tribes, consumption of maximally contaminated fish (in this case mountain whitefish from the Columbia River Basin or pikeminnow from the Willamette River) may present a significant exposure, since average consumption by groups such as the Tribes included in the CRITFC study is 59 g/day (Table 1). Comparable
exposure from less contaminated fish is much less likely even for heavy consumers in these groups.

Fig. 3 shows total TEQ (from PCBs, PCDDs, and PCDFs) exposure from consumption of commercial fish. It would be necessary to consume 37–68 g/day (ND = LOD or ND = 0) of freshwater fish to achieve a TEQ exposure equal to 1 pg/kg/body weight. However, current studies have not identified groups with such high levels of commercial freshwater fish consumption, as this level of consumption would be more consistent with anglers or subsistence fishers who are catching fish from local sources (Connelly et al., 1996; Murray and Burmaster, 1994). For commercial ocean fish, consumption of 250 g/day would yield this level of exposure (115 g/day if non-detects are assumed to equal the limit of detection). Higher level API consumers may consume this amount of commercial seafood (Table 1). For average US consumers, however, total TEQ exposure from fish and seafood is expected to be minimal. Average US consumption of freshwater fish (6.0 g/day, Jacobs et al., 1998) would yield exposure of 0.088–0.16 pg/kg/day (depending on interpretation of non-detects), while average US consumption of ocean fish (14 g/day, Jacobs et al., 1998) would yield exposure of 0.056–0.09 pg/kg/day. PCDD and PCDFs in commercial freshwater and ocean fish are expected to contribute almost half of total TEQ from these sources (Schecter et al., 1997). Results from a similar exercise using total TEQ data from the regional studies were performed but are not presented. In those analyses, PCBs were much more significant contributors accounting for over two-thirds of total TEQ, while PCDDs and PCDFs accounted for less than one-third of total TEQ in the samples. This may reflect specific PCB pollution sources for the Willamette River, Columbia River, and Saginaw Bay.

4. Discussion

4.1. Exposures for average and high-level consumers

For average US consumers eating primarily commercial fish, PCB TEQ exposure from fish and seafood is a fraction of that estimated for total TEQ exposure,
with our studies showing the contribution from these sources to be five to nine percent (depending on interpretation of non-detects) of total TEQ average estimates (USEPA, 2000). Thus for the majority of the population, PCB TEQ from fish is probably not a driving contributor of dioxin-like risk. This is an important finding since there are many reasons to promote consumption of fish and seafood including health benefits such their value as low fat high protein foods and reducing risk of heart disease (Childs et al., 1990; Uauy and Valenzuela, 2000). Previous studies evaluating potential health risks from carcinogenic contaminants in fish (including dioxin and PCBs) have also indicated that the health benefits of consuming many species of fish outweigh the potential risks (Sidhu, 2003). Other high protein foods such as meat and dairy products which might be substituted for fish and seafood may be higher in fat, do not carry these same benefits, and might lead to similar or greater TEQ exposure (Jensen et al., 2000; Schecter and Li, 1997). For example, total TEQ of commercial samples of beef and ocean fish were both 0.16 pg/g, and a sample of hot dogs and bologna was 0.30 pg/g (assuming ND = 0) (Schecter et al., 2001).

Some persons consuming high levels of fish or seafood and/or eating from contaminated locations may have substantial TEQ exposure from these foods, however. This is particularly true for consumers of fish from regional freshwater sources. Even occasional consumption of fish from inland highly PCB polluted water bodies (e.g., Saginaw Bay, MI) may result in PCB TEQ exposure that is several times greater than estimates of US average total dietary TEQ exposure (Fig. 2). Regular consumption of more contaminated fish from moderately PCB polluted water bodies (e.g., Willamette and Columbia Rivers) may also lead to exposure in excess of average population exposure (Fig. 2).

4.2. Implications for fish and dietary advice

The majority of regional PCB based fish advisories currently in effect are driven by Aroclor risk projections, usually the RfD for Aroclor 1254 (a commercial PCB mixture) which is based on immunotoxicity (USEPA, 1997c). Historically, PCBs have been measured in the environment by trying to estimate the amounts of different Aroclors. However, PCBs in the environment bear little resemblance to the original Aroclor mixtures due to differential weathering of PCB congeners. Much of the recent concern about PCBs has focused on developmental immunological (Svensson et al., 1994; Weisglas-Kuperus et al., 2000) and neurological effects (Darvill et al., 2000). Some of these effects may be due to non-dioxin-like PCBs which are not considered in the current analysis. However, cancer risk is likely to be implicated at lower exposure levels than these (Judd et al., 2003a) and PCB cancer risk may be driven by the dioxin-like congeners (USEPA, 2000; Van den Berg et al., 1998).

Few data are currently available on PCB TEQ concentration in fish from regional bodies of water. However, in places where such data are available, like the Willamette River and Columbia River Basin, PCB TEQ risk calculations are much higher than those based on Aroclor risk largely due to the presence of and dioxin-like toxicity of PCB 126 (EVS, 2000; USEPA, 2002). The greater risk projections resulting from congener versus total PCB based risk assessments have previously been explored by Williams et al. (1992). So if more PCB congener data become available for regional bodies of water, there may be more and more restrictive PCB based fish advisories.

In a few cases where older congener data are available and may be used for developing advisories, more current sampling may lead to less restrictive advisories. For example, the Saginaw Bay data are from collections made over 10 years ago. Environmental PCB concentrations in the US declined dramatically in the 1970s after most manufacture discontinued. Since then levels have continued to decline, but much more slowly (Erickson, 1997). PCB concentrations in Saginaw Bay fish are likely lower now than 10 years ago, but there is a need for more current sampling and analyses to evaluate this and update fish advisories.

It is important to maintain perspective of the exposure levels in regional fish and seafood relative to those in commercial seafood, and other potential substitute foods like beef, as well as estimates of total TEQ exposure. For example, Washington state issues advisories based on PCB contamination in bodies of water with fish containing 0.2 ppm PCBs (WADOH, 2002), while FDA will not take action on commercial foodstuffs (fish or meat) unless PCBs exceed 2 ppm (FDA, 2003). For both Washington and FDA, action is based on total PCB or Aroclor risk, not PCB TEQ risk. Thus, following these advisories could lead a consumer away from one food (e.g., regional fish) to another that is equally or more contaminated (e.g., commercial fish or meat).

EPA’s TEQ adult intake estimate (1 pg/kg/day) for average US adults lends an upper bound 1 in 1000 cancer risk (USEPA, 2000). Since the contribution from PCBs in fish and seafood for most consumers of commercial fish and seafood is expected to be 1 in 10,000 (less than 9% of overall TEQ cancer risk), these sources represent a small contribution to an overall unacceptable cancer risk from combined environmental exposures which are overwhelmingly dominated by dietary exposure. This indicates that reducing exposure to these contaminants for the broader population may necessitate comprehensive approaches for exposure reduction such as environmental policy changes aimed at source release reduction and remediation, rather than simple
dietary changes. However, for persons with higher levels of fish and seafood consumption and/or those consuming more contaminated items, this dietary exposure may be very significant.

4.3. Important study assumptions, limitations, and critical data needs

In this study, PCB TEQ and total TEQ exposure from fish consumption were calculated assuming lifetime exposure since many cancers are associated with chronic exposure. Our assumptions may overestimate exposure if consumption of fish and seafood does not begin until later in life. However, for the API community (DREBWQAT, 1999; Sechena et al., 1999), and some Tribes (CRITFC, 1994; SuquamishTribe, 2000), consumption of fish may begin as infants or toddlers. The mobility of Tribal members may be less than the general population, so an assumption of lifetime exposure and residence in one area may be appropriate (Harris and Harper, 1997). For the general population, this may be an overestimate; EPA’s recommended default for average residence time is only 30 years (USEPA, 1997a). Thus exposures linked to specific contaminated sites may be overestimated, but moving may not mean moving away from contamination. Thirty-eight states currently have PCB advisories covering over 2 million lake acres and 100 thousand river miles. The number of PCB advisories has increased 155% between 1993 and 2002 (USEPA, 2003) as more environmental sampling has identified more contaminated sites, many of which have been historically contaminated (Erickson, 1997). Thus, moving and/or changing fishing location may not lead to reduced exposure.

The exposure estimates for commercial and regional fish consumption presented here are based on very small sample sizes and are not as current as would be desired. The limitations of the older Saginaw Bay data in this regard have been previously discussed. The commercial freshwater and ocean fish estimates used in these analyses are based on just a single market basket composite sample each (Schecter et al., 1997). Given the variability in total PCB levels seen between species in other commercial samples (FDA, 2000) and within species seen in regional studies (West et al., 2001), many more samples are needed to reasonably assess exposure from commercial fish. There is also great variability in individual consumer food selection, making a single composite seem inadequate. A review of samples of commercial fish from other countries reveals similar estimates to those presented here for PCB TEQ and total TEQ in mixed commercial samples (Alcock et al., 1998; Tsutsu-umi et al., 2001). TEQ for farmed salmon has been found to be considerably higher than for wild salmon from the same region (3.81–10.54 TEQ compared to 0.26–1.11 TEQ) (Easton et al., 2002). Another recent larger scale study has confirmed higher TEQs in farmed vs. wild salmon from a variety of geographic sources (Hites et al., 2004).

Our analyses reinforce our previous studies indicating that greater analytical sensitivity is needed (Judd et al., 2003a,b). A large range in PCB TEQ estimates was calculated using different interpretations of non-detect values (ND = 0 and ND = LOD). In particular, this indicates substantial uncertainty about exposure from commercial freshwater fish contamination (Fig. 1). One approach to estimate the concentration of non-detected congeners would be to assume correlation between congeners. Such assumptions were not appropriate in this study given the limited number of samples per site and species studied, the differences within and between commercial PCB mixtures used, and regional ecosystem and species level differences in weathering (Erickson, 1997; Frame, 1999). Analyzing more samples with greater analytical sensitivity will involve considerable expense, but the necessity of increasing sampling efforts is demonstrated by exposure estimates of health concern for some populations based on limited existing data.

Another major limitation of this study is the incompleteness of the PCB analysis of commercial freshwater and ocean fish. The data used in this study did not include evaluation of PCB 123, 156, 157, 167, or 189 but no more comprehensive data could be identified (Schecter et al., 1997). These omitted congeners have small TEFs, but their contribution may still be significant since they may be present at high concentrations. In some samples from the Willamette River study, PCB 156/157 contributed 30% of PCB TEQ (EV5, 2000). Because there was no information on the presence or absence of these congeners in the commercial fish data evaluated in our study (no data or detection limits) (Schecter et al., 1997, 2001), we were limited in our ability to make a reasonable estimate of their contribution. This lack of data leads to an underestimation of exposure and should be considered in interpretation of these results. This provides further indication of the need for more analyses of commercial fish and seafood to improve human health risk assessments.

Analyses were incomplete in data from regional samples as well. PCB 81 was not included in the Columbia River Basin (USEPA, 2002) and Willamette River sample analyses (EV5, 2000). Again, because there was no information on the presence or absence of this congener, its concentration was assumed to be zero. The TEF for this congener is small, however, and it contributed less than 1% of the PCB TEQ in the Saginaw Bay study samples (Giesy et al., 1997). So underestimation from this omission is expected to be small. The method used for the Willamette River and Columbia River Basin studies also did not distinguish PCBs 156 and 157. Provided the reported concentration represents

\[ \text{ND} = \text{LOD} \]
the sum of the two individual congener concentrations, this is not expected to affect TEQ estimates, since the TEF for these two congeners is the same (Van den Berg et al., 1998).

There were many more composite samples available from Saginaw Bay (n = 10), the Willamette River (n = 15), and the Columbia River Basin (n = 281) than for commercial fish (n = 1 freshwater, n = 1 ocean fish). There is great variability in PCB TEQ contamination in fish from different sites and from the same general area, indicating a need for more testing in contaminated areas. Given the great expense of PCB congener testing, efforts should be prioritized for locations where even occasional consumption may be of health concern, such as Saginaw Bay and areas with lower contamination levels that are used by high-level and subsistence fish consumers, including Tribes and recent Asian and Pacific Islanders immigrant populations.

In fish sampled from the Willamette River and Saginaw Bay, PCBs were the primary source of TEQ exposure with PCDDs and PCDFs contributing a maximum of less than one-third of total TEQ (EVs, 2000; Giesy et al., 1997). Contributions of PCDDs and PCDFs to total TEQ are less certain for commercial fish since there are so few samples, although in the study results presented here, PCDDs and PCDFs contributed the majority of TEQ (Schercter et al., 1997). Studies from other countries indicate the contribution of PCBs to TEQ may be more important. PCBs contributed the majority of TEQ in Japanese commercial fish and shellfish (Tsutsumi et al., 2001) and Norwegian farmed salmon (Lindstrom et al., 2002). A review of several international studies, reported PCB TEQ from fish and seafood to range from 32 to 92% of total TEQ with the majority over 70% (Alcock et al., 1998). Jensen and Colleagues (2000) have published extensive data on PCDDs and PCDFs in commercial fish and reported lower PCDD and PCDF TEQ levels than Schecter et al. (1997), although this may be due in part to global environmental declines over time. In the Jensen and Bolger study, PCB TEQ was not measured, so these data cannot be used to assess total TEQ exposure. To improve TEQ based risk assessment for commercial freshwater fish and seafood, more samples with greater analytical sensitivity for dioxin-like PCBs and analysis of PCDDs and PCDFs are needed.

Prioritizing improvements in exposure assessment should also be considered in the context of total risk uncertainty. When this is done, uncertainty surrounding the toxicity potency factors is very large. Toxicity metrics (e.g., cancer slope factor for TCDD) contain different safety and modifying factors. These factors, which may make the toxicity metric 1000 times lower than the dose at which effects are seen in the laboratory or in epidemiological studies, are intended to provide a margin of safety for uncertainties (animal to human extrapolations, inter-individual variability, etc.). The current draft dioxin cancer slope factor is an upper bound estimate calculated from a meta-analysis including three epidemiological studies (USEPA, 2000). Thus, while technology to increase analytical sensitivity of environmental samples is currently available, possible reductions in uncertainty about exposure may be dwarfed by toxicity uncertainty. Improving these toxicity metrics through further research is as important to improving risk assessment as improving analytical sensitivity.

5. Conclusion

For most adult consumers, fish and seafood derived PCB TEQ may contribute a small incremental risk relative to an unacceptable ambient TCDD-TEQ associated risk, indicating the need for exposure reduction options beyond simple dietary changes. However, PCB TEQ exposure for groups with high levels of commercial fish consumption, particularly freshwater fish, may be large relative to estimates of average total TEQ exposure. Consumption of even small quantities (one meal a month or less) of fish from highly PCB contaminated areas may lead to substantial TEQ exposure. This points to the need for fish advisories that allow people to self-identify based on their own consumption.

There is a lack of data in terms of number of samples and analytical sensitivity on PCB TEQ and total TEQ in commercial fish and seafood. This need must be addressed to reasonably assess exposure and risk for high-level consumers of commercial fish and seafood. The high concentration and variability in PCB TEQ in fish from known contaminated areas indicates that better characterization of fish TEQ burdens by more sensitive analytical sampling is needed at many PCB contaminated sites where recreational or subsistence fishing occurs. These data are essential for producing more accurate risk assessments and maintaining holistic health protective approaches toward issuance of fish consumption advice. PCB TEQ exposure must be considered in the context of overall TEQ exposure given that the health and cultural benefits of fish and seafood consumption are numerous and consumption of substituted foods may lead to equal or greater TEQ associated risks.

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