Accumulation of persistent organic pollutants in consumers of eel from polluted rivers compared to marketable eel

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Abstract
Globally, many river sediments are seriously contaminated with persistent organic pollutants (POPs) known to accumulate in aquatic food. In the Netherlands, toxicological risks of human exposure to dioxins and dioxin-like compounds led to a ban on eel fishing in the Rhine-Meuse delta. The aim of this study is to investigate differences in serum POP levels in consumers of eel from high-polluted areas and consumers of eel from low-polluted areas or aquaculture. In total 80 Dutch men were included, aged 40–70 years, with a habitual eel consumption of at least one portion (150 g) per month. Total levels of dioxins and dioxin-like compounds were measured in serum of all participants with the DR CALUX bioassay, validated with GC-MS. For a subgroup of 38 participants extensive POP measurements were performed. We revealed that consumption of eel from polluted rivers resulted in 2.5 and up to 10 times increased levels of dioxins and polychlorinated biphenyls (PCBs) respectively compared to controls. The highest PCB levels were detected for PCB 153, with a median level of 896 ng/g lipid and a maximum level of 5000 ng/g lipid in the high-exposed group. Furthermore, hydroxylated PCB metabolites (OH-PCBs: sum of 4-OH-CB107, 4-OH-CB146, 4-OH-CB172, and 4-OH-CB187) were 8 times higher in men who consumed eel from polluted areas, and detected at levels (median 4.5 ng/g ww) reported to cause adverse health effects. Also, the majority of the perfluoroalkyl substances (PFASs) were significantly higher in consumers of eel from polluted areas. In conclusion, this study is the first to reveal that (past) consumption of eel from polluted areas resulted in high body burdens of dioxins, PCBs, OH-PCBs and PFASs. We confirmed the predictions made in a former risk assessment, and the high levels of dioxins and dioxin-like compounds as well as the OH-PCBs are of health concern.

1. Introduction
Persistent organic pollutants (POPs) are a group of mainly lipophilic compounds that are resistant to degradation and accumulate in the environment and the food chain. POPs include dioxins, polychlorinated biphenyls (PCBs), organochlorine (OC) pesticides, brominated flame retardants, and perfluoroalkyl substances (PFASs). These chemicals are known for their potency to cause various adverse health effects, including endocrine disorders, cancer, and neurodevelopmental problems (Li et al., 2006). Many POPs are regulated by the Stockholm Convention, and POP levels in the environment have been decreasing over the past decades (Muir and de Wit, 2010). However, their occurrence in the environment is still a major concern. A high degree of urbanization and industrialization along European rivers have caused the sediments of the main Dutch rivers and estuaries to be highly polluted (den Besten...
Over 90% of human exposure to POPs comes from food consumption (Liem et al., 2000), with high contributions from fish in general (Bilau et al., 2008; De Mul et al., 2008). Certain fish species dwelling in contaminated areas may contribute even more. In particular eel has potential for high accumulation of contaminants due to eco-physiological features such as bottom dwelling, being a long-living predator, and having a high lipid content (de Boer et al., 2010; Guhl et al., 2014; Kwadijk et al., 2010).

Dioxins (polychlorinated dibenzo-p-dioxins/dibenzo furans (PCDD/Fs)) and dioxin-like (DL-) PCBs in food have received considerable attention from the European Commission. PCDD/Fs are highly toxic, although with different toxic potencies, as expressed in a range of toxic equivalency factors (TEFs) (Van den Berg et al., 2006). DL-PCBs, including non-ortho (NO) and mono-ortho (MO) PCBs, have properties similar to PCDD/Fs and therefore also assigned TEF values. The NO-PCBs have a higher dioxin-like potency than the MO-PCBs (Van den Berg et al., 2006). To reduce levels in food and hence human exposure, maximum levels (MLs) for PCDD/Fs and DL-PCBs in various food items have been established using toxic equivalents (TEQs) to sum up the different TEF values. In 2012, the ML for the sum of PCDD/Fs and DL-PCBs in wild eel was revised to 10 pg TEQ/g wet weight (ww), while farmed eel and other seafood items have to comply to 6.5 pg TEQ/g ww (EC, 2011). Several studies showed that the majority of eel from the Rhine-Meuse delta exceeded the ML (Guhl et al., 2014; van Leeuwen et al., 2007). Following this discovery, a risk assessment was performed, including the potential impact on the body burden. Long-term consumption of one portion eel (150 g) per month from these high-polluted areas (average level of 29 pg TEQ/g eel) was estimated to result in a body burden of 7.5 ng TEQ/kg body weight (bw), as compared to 3.0 ng TEQ/kg bw for fish eaters not consuming eel (Hoogenboom et al., 2007). Even such moderate eel consumers were therefore expected to reach POP levels above the safe body burden of 4 ng TEQ/kg bw (extrapolated from rat studies), implying that adverse health effects could not be excluded (EU-SCF, 2001; Malarvannan et al., 2014). After a documentary was broadcasted raising awareness, the Dutch government decided on a complete ban on eel fishing from 2011 onwards in the seriously polluted fishing areas, where the majority of the eel exceeded the European maximal levels. Accumulated POPs are very persistent and it takes years before body burdens decrease (half-lives approximately 10—15 years for PCBs; Ritter et al., 2011). Therefore, it is expected that people who consumed this eel regularly in the past will have elevated levels of PCDD/Fs, PCBs and other POPs in their body years after the ban. As the necessity of the prohibited eel fishing is debated, we decided to study the actual body burdens in eel consumers to verify the expectations expressed in the former risk assessment. The aim of this study was to compare blood POP levels in men who consumed eel from high-polluted areas (now closed for eel fishing) with men consuming eel from aquaculture and relatively low-polluted areas (areas open for eel fisheries).

2. Methods

2.1. Study population

The overall setup of the study is depicted in Fig. 1. Eligible participants were all Dutch men aged from 40 to 70, with a long-term habitual eel consumption (in their adult life) of at least one portion (150 g) a month (at least until the implemented ban on eel fishing in 2011). Age range was based on Hoogenboom et al. (2007), who showed that body burden increases due to eel consumption, but remains relatively stable from 40 to 70 years of age. Men were invited to participate through professional and recreational fishermen associations, because these men are more likely to know the origin of the eel, and through advertisements in local newspapers and webpages. A total of 80 men were included, all between February and June 2015, after checking whether they met the inclusion criteria. This study was approved by the Medical Ethical Committee of Wageningen University, and written informed consent was obtained from all participants before inclusion in the study.

Participants filled out a questionnaire at home about their fish consumption habits, including the origin of the eel. The eel was considered to be low-polluted when it came from either aquaculture or areas still open for eel fishery where the eel complies with the European maximum level. The eel was considered to be high-polluted when it came from areas where there is a current ban on eel fishing, as the majority of the eel caught here does not comply with the European maximum level (Rijksoverheid, 2011).

Height and weight of participants were measured in light clothes using standard methods. Blood samples were obtained by venipuncture after overnight fasting, and serum for the persistent organic pollutant (POP) analyses was separated by centrifugation within 6 h after collection. Samples were stored at –80 °C until further analyses. Total cholesterol and triglycerides levels were measured in plasma in a clinical chemistry laboratory (Hospital Gelderse Vallei, Ede, The Netherlands).

2.2. DR CALUX bioassay

Total levels of dioxins and dioxin-like compounds were measured for all 80 subjects using the DR CALUX bioassay at RIKILT Wageningen UR, which was validated for food and feed, as described previously (Bovee et al., 1998). In short, aliquots of 5 mL serum were extracted twice with hexane and purified on a column containing 10 g acid silica (33% H2SO4) with 1 g dried Na2SO4 on top. The extracts were dried in a SpeedVac with 10 μL DMSO as a keeper, which was later mixed with cell culture medium. Control samples of butter fat with different levels of a mix of PCDD/Fs and DL-PCBs (0.5; 17; 39; and 88 pg TEQ/g lipid), and with a similar absolute amount of fat, were included and extracted in the same way. p-GudLuc transfected H4IE-cells were obtained from Wageningen University (Merk et al., 1997) and are similar to those sold by Biodeetection Systems (BDS, Amsterdam, The Netherlands). Cells were cultured in a 96-wells plate and exposed to the extract (1% DMSO) in quadruplicate for 24 h. The cells were lysed and the luciferase content was measured in a Luminoskan (Labsystems). Total levels of dioxins and dioxin-like compounds were estimated from a calibration curve of the reference butter fat samples included in each clean-up series. These estimated levels were expressed in bioanalytical equivalents (BEQs) to acknowledge the fact that they were determined with a bioassay and not with gas chromatography high resolution mass spectrometry (GC-HRMS). In the bioassay also other compounds that pass the selective clean-up may contribute to the response. BEQ levels were adjusted for lipid content, calculated based on triglycerides and total cholesterol levels as described by Phillips et al. 1989 and recommended by AMAP 2009, which correlates well to gravimetric determination (Bergonzi et al., 2009; Bernert et al., 2007).

2.3. Validation bioassay with GC-HRMS and congener patterns

Eight pooled samples as well as two individual samples with the highest estimated BEQ levels (>100 pg BEQ/g lipid) were measured with GC-HRMS at RIKILT Wageningen UR, which is ISO/IEC 17025 accredited for the analysis of PCDD/F and PCB containing extracts (LO14). GC-HRMS was first of all performed to validate the bioassay results, and secondly to compare congener patterns. Pooled serum included samples from all low- and high-exposed men (n = 34 and...
n = 21, respectively) and the subgroup for which the extensive POP measurements were performed (n = 14 for low- and n = 13 for high-exposed men). Furthermore, samples were pooled based on individual BEQ levels to get sub-groups for the low- and high-exposed group with the same average BEQ levels (20 and 45 pg BEQ/g lipid). Levels of PCDD/Fs and DL-PCBs were determined in 3–12 mL serum. Prior to extraction, the samples were spiked with 13C- isotope labelled internal standard for each congener. Extraction of lipids was performed as previously described (Smedes, 1999). After evaporation of the solvents, the residues (lipids) were dissolved in hexane and the crude extract was purified using an automated clean-up (PowerPrep system, Fluid Management Systems, Waltham, USA). Extracts were purified on an acid silica column, a neutral silica column, a basic alumina column and an activated carbon/Celite column. For the elution from the columns, custom made solvents and mixtures were used, respectively being hexane/dichloromethane (1:1, v/v), ethyl acetate/toluene (1:1, v/v) and toluene. The volume of the final extract was reduced using an automated evaporation system with fixed endpoint of 0.5 ml. The recovery standards, 13C-labelled 1,2,3,4-TCDD and 2,3,4,6,7,8-HxCDF, were added and the volume of the extract was again reduced to 0.5 ml. PCDD/F and PCB analyses were performed by GC-HRMS using an Agilent (Wilmington, USA) 6890 Series gas chromatograph and an AutoSpec Ultima high resolution mass spectrometer (Waters, Milford, USA) operated at a resolution of 10,000 (10% peak valley). The GC column was a DB5 MS (60 m, 0.25 mm i.d., 0.25 μm; J&W, Folsom, USA). The mass spectrometer was operated in electron impact ionization mode, using selected-ion monitoring. Further GC-HRMS details can be found elsewhere (Tuinstra et al., 1994). A large volume injector (LVI) capable to inject 100 μl was used to inject the sample on the GC. Data were converted to TEQ levels using the TEFs of 2005 (Van den Berg et al., 2006) and expressed per gram lipid using the calculated lipid content.

2.4. Extensive POP analyses

Further analyses for individual POPs were performed for 38 participants. Samples were selected based on age and BEQ values. Participants were divided in three age groups (40–49, 50–59, 60–70) to ensure age differences between groups would not affect POP results as body burden is related to age (Hardell et al., 2010). Based on bioassay results, participants were divided in having a low body burden (median around 13 pg BEQ/g lipid), medium body burden (median 2 × higher than low body burden), and high body burden (median 4 × higher than low body burden, detailed information in Table S4). After this stratified sampling, we used a simple random sampling for each stratum to choose the 38 participants.

The individual POP analyses were performed at the Laboratory of Environmental Toxicology at the Norwegian University of Life
Sciences (NMBU), which is accredited by the Norwegian Accreditation for testing of PCBs, organochlorine (OC) pesticides, and brominated flame retardants (BFRs) in biological material according to the requirements of the ISO/IEC 17025 (TEST 137) (Norwegian Standard-European Committee for Standardization/The International Electrotechnical Commission, 2005). The extraction method for most POPs, excluding perfluoroalkyl substances (PFASs), was performed with 4 mL of serum and is based on a liquid/liquid lipid extraction with acetone and cyclohexane (3:2), sodium chloride (NaCl) and 10 mL 1 M sulphuric acid (H₂SO₄) which is described by Polder et al. (2008). An additional extraction with potassium hydroxide (1 M) was performed to extract the hydroxylated metabolites (Løken et al., 2006). The analyses were performed with GC, followed by Electron-Capture Detection (ECD) for analyzing PCBs and OC pesticides, and by low resolution MS (LRMS) quadrupole detector for OH-PCBs, BFRs and for confirmation of certain PCBs and OC pesticides. The limit of detection (LOD) was set at three times the noise levels and results were reportable if the contaminant data were normalized to lipid content of individual samples (Krishnamoorthy et al., 2009; U.S. EPA, 2000). All POP levels were log₁₀-transformed for statistical and graphical purposes. After log-transformation not all POPs were normally distributed as verified with the Kolmogorov-Smirnov test. Therefore, non-parametric tests were used. The Mann-Whitney U test was performed to compare the low-exposed to the high-exposed group and the Spearman rank correlation was used to test the statistical significance for the correlation between the BEQ levels and age. Differences were assumed to be statistically significant when the p-value is below 0.05. To convert the well-accepted safe body burden of 4 ng TEQ/kg bw to levels in serum (15.1 pg TEQ/g lipid) for comparison reasons, a 26.5% body fat content was used (Deurenberg et al., 1997; Gurrici et al., 1998).

3. Results

3.1. Study characteristics

In total 80 eligible men responded to our research invitation and eventually participated in this study. Of these men, 34 consumed eel exclusively from areas open for eel fishery or aquaculture (low-exposed group) and 21 consumed eel exclusively from high-polluted areas where the ban on eel fishing is now implemented due to exceedance of the MLs (high-exposed group). Other participants consumed eel from both areas or were not aware of the origin of the eel. The majority of the men consumed eel once or twice per month, and 20% consumed eel at least five times per month. Most men also consumed other fish. Age and BMI were comparable between the low- and high-exposed groups (Table 1).

3.2. Dioxins and dioxin-like compounds

Total levels of dioxins and dioxin-like compounds are expressed in bioanalytical equivalent (BEQ) when measured with the

<table>
<thead>
<tr>
<th>Number of participants</th>
<th>Total</th>
<th>Low-exposed</th>
<th>High-exposed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>Med (min-max)</td>
<td>80 (40–70)</td>
<td>34 (41–70)</td>
</tr>
<tr>
<td>Fishermen</td>
<td>n (%)</td>
<td>26 (33)</td>
<td>10 (29)</td>
</tr>
<tr>
<td>n (%)</td>
<td>Sport</td>
<td>27 (34)</td>
<td>9 (26)</td>
</tr>
<tr>
<td>Frequency eel consumption</td>
<td>1–2× per month</td>
<td>51 (64)</td>
<td>23 (68)</td>
</tr>
<tr>
<td>n (%)</td>
<td>3–4× per month</td>
<td>13 (16)</td>
<td>2 (6)</td>
</tr>
<tr>
<td>Frequency consumption other fatty fish</td>
<td>&lt;1× per week</td>
<td>44 (55)</td>
<td>17 (50)</td>
</tr>
<tr>
<td>n (%)</td>
<td>1–2× per week</td>
<td>34 (43)</td>
<td>16 (47)</td>
</tr>
<tr>
<td>Frequency consumption of semi-fatty fish</td>
<td>&lt;1× per week</td>
<td>33 (41)</td>
<td>12 (35)</td>
</tr>
<tr>
<td>n (%)</td>
<td>1–2× per week</td>
<td>44 (55)</td>
<td>20 (59)</td>
</tr>
<tr>
<td>Frequency consumption of lean fish</td>
<td>&lt;1× per week</td>
<td>44 (55)</td>
<td>21 (62)</td>
</tr>
<tr>
<td>n (%)</td>
<td>1–2× per week</td>
<td>34 (43)</td>
<td>13 (38)</td>
</tr>
</tbody>
</table>

* The low-exposed group ate eel exclusively from aquaculture and/or eel complying with fish quality standards. The high-exposed group ate eel exclusively from areas now banned for eel fishing. The other participants consumed eel from both areas or were not aware of the origin of the eel.
bioassay, and in toxic equivalent (TEQ) when analyzed with GC-HRMS. These values correlated well (Spearman \( r = 0.95; \) BEQ = 0.95*TEQ + 4.0), based on ten samples analyzed with both techniques. The BEQ values followed a normal distribution (Fig. 2A) and were in general increased in the high-exposed men (Fig. 2B), with levels up to 145 pg BEQ/g lipid (164 TEQ/g lipid based on GC-HRMS). Based on the pooled samples from the low- and high-exposed men, the high-exposed men had on average 2.5 times higher TEQ levels. In the low-exposed group especially the dioxins contributed to the TEQ level (Fig. 2C). For the high-exposed group the majority of the TEQ came from DL-PCBs, more specifically the NO-PCBs (Table S1). The MO-PCBs, however, showed a higher relative increase, namely 2.3-times compared to 1.5-times for the NO-PCBs comparing percentages of high- to low-exposed. Also other PCBs were present at relatively higher levels as shown by the ratio of PCB153 and TEQ (Fig. S1).

3.3. Individual persistent organic pollutants (POPs)

Of the 48 POPs measured in the subgroup of 38 men (14 low-exposed, 13 high-exposed, and 11 with unknown origin of the majority of the consumed eel), 30 POPs had levels above the LOD in at least 60% of the samples (Table S2). Levels of the OC pesticides: \( \beta \)-HCH, mirex, oxychlordane, and trans-nonachlor, did not differ between the two groups (Fig. S2). Levels of HCB and DDT, but not DDE, were significantly elevated in the high-exposed men (Fig. 3). Furthermore, all PCBs (except for PCB 52) were significantly higher in the high-exposed men, with fold differences ranging up to 10.6 (Fig. 3). Concomitant increases of the internal OH-PCB metabolites were observed, amounting to an eight-time increase for the sum of the four measured OH-PCBs (Fig. 4).

Two PBDEs, BDE-47 and BDE-153, were detectable in at least 60% of the men but levels were not significantly different between the groups (Fig. S3). Furthermore, two perfluorinated alkylsulfates and six perfluorinated alkylacids were detectable (Fig. 5). The median levels of most PFASs were significantly elevated, up to 3.5 times in the high-exposed men. Detailed information for all 30 POPs can be found in Table S4.

4. Discussion

Our research is the first to reveal that men who consumed eel from high-polluted areas in the Netherlands have, up to ten times, higher body burdens of dioxins, (OH-)PCBs and PFASs compared to men consuming eel from relatively clean areas or aquaculture. Although risk assessment and management mainly focus on dioxins and PCBs, we show that also the body burden of hydroxylated PCBs might be of health concern.

The levels of dioxins and dioxin-like compounds observed in the

![Fig. 2.](image)

![Fig. 3.](image)
low-exposed men are comparable to background levels observed in other European studies (Consonni et al., 2012) (orange block, Fig 2A). The high-exposed men had on average 2.5 times higher TEQ levels, which is in agreement with the predictions made in a previous risk assessment on eel consumption (Hoogenboom et al., 2007). Total levels of dioxins and dioxin-like compounds, however, were lower than those reported in fishermen consuming fish from the highly polluted Baltic Sea (Kiviranta et al., 2002). The Baltic Sea herring contains in general lower TEQ levels than eel from polluted rivers (Karl et al., 2010), but is consumed more frequently, explaining the even higher levels in consumers.

The median BEQ levels of both groups in our study (18 and 39 pg BEQ/g lipid, for the low- and high-exposed respectively), are above the estimated safe level of 15 pg TEQ/g lipid (EU-SCF, 2001). Although fish consumption is considered healthy because of its nutritional value, frequent consumption of fish with levels above the ML is expected to result in TEQ body burdens that exceed the safe level (Baars et al., 2004; Hoogenboom et al., 2007). The safe level is derived from reproduction studies with rats and aims to protect humans from accumulating dioxins and DL-PCBs to body burdens that may result in adverse effects on especially unborn children, and is therefore based on a long-term exposure (EU-SCF, 2001).

Men in this study consumed eel for decades, and given the long half-life, the body burden is assumed to include POPs from (possibly even higher) contaminated eel consumed in the past and from other food. Interestingly, in the low-exposed group the BEQ levels are positively correlated with age, but not in the high-exposed group (Fig 5). This could be explained by recent intake of high-polluted eel, while the low-exposed group might have benefited more from the generally decreased POP levels in food products.

Levels of PFAS in the low-exposed group are comparable to levels measured in the USA (Olson et al., 2003), while the high-exposed men had PFOS levels well above general population levels (Zeng et al., 2015). Recently, the National Institute for Public Health and the Environment in the Netherlands has estimated a safe level of 89 ng PFOA/mL serum (RIVM, 2016). As PFOS and PFOA have a similar toxicity profile (ATSDR, 2015), this means that the high-exposed men (median level 82 ng/g, maximum level 260 ng/g) are close to this safe level by PFOS alone.

Brominated flame retardant levels did not significantly differ between the groups, which is in accordance with earlier reports that regular eel consumption does not cause health risks from PBDEs and HBCD (Malarvannan et al., 2014). Besides food, significant exposure to PBDEs occurs from house dust due to e.g. electronics or carpets releasing flame retardants (Buttke et al., 2013), while PCB exposure is almost fully limited to the food chain (Liem et al., 2000).

The highest PCB-exposed populations are found around the arctic, where traditional foods include marine mammals, such as seal and whale blubber (Fängström et al., 2002; Sandanger et al., 2003; Sandau et al., 2000). Interestingly, levels of PCBs and their OH-metabolites were higher in Dutch men consuming eel from high-polluted areas than in men consuming marine mammals, and were among the highest levels measured in the 21st century (orange blocks, Fig 4) (Montano et al., 2013). This could be related to the higher PCB biotransformation capacity in mammals compared to fish (Murk et al., 1994), resulting in higher PCB levels in eel due to a lack of elimination of the higher chlorinated PCBs (de Boer et al., 1994). The PCBs will be metabolized by the eel consumers who subsequently retain the OH-metabolites in their body.

OH-PCBs have been associated with several adverse health effects in animal studies, especially related to development, growth and behavior (Montano et al., 2013). OH-PCBs can mimic thyroid hormones and have a high affinity for thyroid hormone transport proteins (Marchesini et al., 2008). In all men the sum of the OH-metabolites are expected to give adverse health effects based on in vitro studies, and in the majority of the men based on animal studies (Fig. 4). Furthermore, the (OH-)PCB levels in the high-exposed eel consumers were slightly higher than those reported for Inuit from Canada, where negative effects on thyroid hormone parameters were identified, especially a reduction of triiodothyronines (Dallaire et al., 2009).

To confirm that eel is the actual source of the elevated POP levels, we compared the POP profiles in men and eel. In general, TEQ exposure from aquatic species mainly originates from DL-PCBs,
while in terrestrial sources (such as meat and dairy) PCDDs and DL-PCBs have an equal contribution (Focant et al., 2002). Indeed, for the average Dutch consumer the main sources contributing to PCDDs (Baars et al., 2004). In our study the major contribution to the TEQ levels in the high-exposed men came from DL-PCBs, indicating fish as a likely source, while in low-exposed men the PCDD/Fs contributed most to the TEQ. Additional indication for eel as the source of the higher TEQ levels is the relatively high contribution of MO-PCBs within the DL-PCBs. In farmed eel and other fish, NO-PCBs predominate, but wild eel has relatively high levels of MO-PCBs (van Leeuwen et al., 2007). The PCB profiles in the high-exposed men followed the eel profiles, while the PCDD/F profiles were different between men and eel (Fig. 6). This, together with the increasing PCB153 to TEQ ratio in the high-exposed men (Fig. S1), indicates that eel is the most likely source of the elevated TEQ and PCB levels.

Our results show that eel from high-polluted areas is not safe for consumption, and therefore our results support the arguments from the previous performed risk assessment justifying the ban (Hoogenboom et al., 2007). Several neighboring countries (e.g. Belgium and Germany), however, still do not have a ban on catching eel from high-polluted areas (LANUV, 2012; Ministerie van de Vlaamse Gemeenschap, 2005). Indications exist that recreational fishermen are still catching high-polluted eel for consumption, and we recommend more strict regulation to prevent consumption of these eels. Most eel sold in the Netherlands nowadays comes from aquaculture and to a limited extent from low-polluted areas. Our results reveal that consumption of this eel indeed does not lead to a high increase of background POP levels, as was predicted before (Hoogenboom et al., 2007). Because of its potential beneficial effects, weekly fish consumption is advised by the WHO (WHO, 2016), but contaminant levels should comply with safe legal limits. Appropriate measures should be taken to prevent consumption of fish from high-polluted areas world-wide.

Conflict of interest

All authors declare no financial competing interests.

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**Fig. 6.** Profiles of (A) PCDD/Fs that occurred in levels higher than 5 pg/g lipid in high-exposed men and (B) the seven indicator PCBs in both eel and men. Eel data are derived from 360 (pooled) samples, collected between 2001 and 2012. The average levels in eel are calculated for both low- (dark green bars) and high-polluted areas (dark red bars) (partly unpublished data from monitoring program, based on de Boer et al., 2010; Van Leeuwen et al., 2013). Human data are determined in pooled samples for low- (n = 34, bright green bars) and high-polluted men (n = 21, bright red bars) (this study). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
UR. The support by DUPAN (Dutch sustainable eel foundation) enabling more elaborate chemical analyses is highly appreciated. The funding sources had no involvement in conducting the research or in preparing the article. The data presented in this paper are available in the Supplementary Materials.

Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.envpol.2016.09.019

References


110, 355–361.