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PCB and organochlorine pesticide burden in eels in the lower Thames River (UK)

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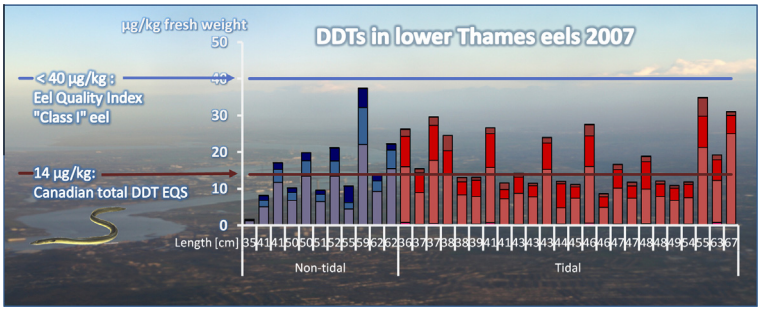
HIGHLIGHTS

- 35 Eels, caught in the Thames near London in 2007, were analysed for some POPs.
- Pesticide and PCB contamination was relatively low compared to previous studies.
- No EU food or environmental standards (EQS) were exceeded.
- However, dioxin-like PCBs and total DDT exceeded a Canadian EQS.
- Tidal eels had more lipid and fewer *A. crassus* infections than upstream ones.

GRAPHICAL ABSTRACT



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ABSTRACT

Thirty-five European eels (*Anguilla anguilla*), caught in 2007 in the river Thames upstream and downstream of both London and the tidal limit, were analysed for PCBs and organochlorine pesticides. Most chemicals were detectable in every fish, although they have been banned or severely restricted for many years. In general, the tidal eels were more contaminated than upstream ones, which was related to their higher lipid contents.

The ICES7 indicator PCB concentrations ranged overall from 4.2 to 124 µg kg^{-1} fresh weight with averages of 33 and 56 µg kg^{-1} for the upstream and tidal eels; 3.5–104 µg kg^{-1}, average 26 and 48 µg kg^{-1} of that were ICES6 PCBs. Total DDT was on average 16 µg kg^{-1} (1.7–38 µg kg^{-1}) upstream and 18 µg kg^{-1} (8.6–35 µg kg^{-1}) downstream with about half of that provided by *pp'*DDE. Lindane (γ-HCH) was found at up to 2.8 µg kg^{-1} (averages 0.58 and 1.1 µg kg^{-1} upstream and downstream) and hexachlorobenzene (HCB) was on average 1.9 and 2.5 µg kg^{-1} in the two groups with a maximum of 6.4 µg kg^{-1} in each. Therefore all individuals passed the European Environmental Quality Standard (EQS) of 10 µg kg^{-1} for HCB. PCB contamination was fairly typical for recent UK eel data, whilst DDE and lindane concentrations were lower than most previous UK eel studies, perhaps reflecting a downward trend.

Although not as highly contaminated as some eels from previous UK and European studies, the presence of so many of these chemicals, with their known health effects may represent a stress for the fish or higher predators, such as birds.

Abbreviations: EQS, environmental quality standard; EQI, eel quality index; TEF, toxic equivalency factor; TEQ, toxic equivalent concentration TEQ = $TEF_1 * conc_1 + TEF_2 * conc_2 + \ldots$; NGR, National Grid Reference.

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1. Introduction

1.1. Concern over eel numbers

The European Eel (*Anguilla anguilla*) is an important species for commercial fisheries. There is, however, much concern over sharply declining numbers from about 1980 onwards (ICES, 2011). The European eel is now on the IUCN Red List classified as a "critically endangered species" (Freyhof and Kottelat, 2010). All European Union countries where eels occur, have to produce eel management plans, with the long term aim of ensuring silver (mature) eel escapement to the sea recovers to at least 40% of what it would be if there were no anthropogenic influences (European Union, 2007). Successful recovery plans are however hindered by a lack of certainty about the main cause(s) of the decline. Climate change leading to reduced ocean productivity (Bonhommeau et al., 2008), and to variations in ocean currents (Baltazar-Soares et al., 2014), overfishing and loss of habitat perhaps particularly in coastal areas relatively near to the Sargasso Sea (Kettle et al., 2011), infections-especially with the nematode *Anguillicola crassus* (Palstra et al., 2007), barriers to migration (Chadwick et al., 2007), and pollution (Robinet and Feunteun, 2002) have all been implicated.

1.2. The eels' life cycle in relation to pollution

Since eels are benthic carnivores with a high fat content and long life span, they tend to accumulate higher amounts of persistent chemicals from water, food, and sediment than other species (Belpaire and Goemans, 2007; Jürgens et al., 2013). In other fish species the females, and to a lesser extent males, offload lipids and with them part of their contaminant burden annually during spawning, but because eels only spawn once at the end of their lives they do not have that opportunity. These characteristics, along with the fact, that during their yellow (growth) phase most eels are highly sedentary, make them ideal for monitoring chemical pollution in the water systems where they reside. However, these features may also quite literally store up problems for their own future or present a problem to their predators. During the long spawning migration, sexual maturation occurs and they do not feed but rely instead entirely on their fat reserves. Thus chemicals that were incorporated into the fat can either be remobilized, causing potential problems to the eels during this important stage of sexual maturation, or are concentrated further in the remaining fat, much of which is later incorporated into the eggs. Palstra et al. (2006) claimed to have found a link between environmental dioxin-like contamination of eels and early death during the larval development of their offspring. Developmental failure in the offspring of contaminated females has been observed in other fish species: for example Burdick et al. (1964) reported the complete loss of lake trout fry at a particular stage in development due to DDT contamination passed on to the eggs. For a detailed review of effects of chemicals on eels see Geeraerts and Belpaire (2010).

1.3. Chemicals studied

PCBs were widely used in the 50s and 60s as cooling fluids in transformers and many other uses. Their release into the environment peaked in the 1960s before concerns over human and environmental health effects led to severe restrictions from the 1970s onwards (the dates chemicals were banned are given in Table 1). PCBs have been linked to thyroid hormone disruption (Brar et al., 2010) and reduced reproductive success (Daouk et al., 2011) in fish.

Organochlorine pesticides were hailed as part of the agricultural revolution after the war, but concerns about their bio-accumulating properties led to a ban or severe restriction for most of these compounds since about the 1980s. In this study the insecticides DDT, chlordane, lindane (γ-HCH) and endosulfan and the fungicide hexachlorobenzene (HCB) as well as some of their degradation- or by-products were selected for study. Apart from endosulfan, which could be used in EU agriculture until 2007 (European Commission, 2005a), they were all banned or very severely restricted from 1981.

DDT is probably the most widely studied pesticide. Its acute toxicity to fish at high concentrations was noted early on when fish kills were observed in sprayed areas (e.g., Surber, 1946). In the 50s it was observed that the offspring from DDT contaminated female lake trout did not survive past the stage where the yolk sac is absorbed, which was explained by maternal transfer of DDT to the eggs (Burdick et al., 1964) and by the 70s effects on osmoregulation of different fish species, including eels, became known (e.g., Janicki and Kinter, 1971). Technical DDT consists of about 85% *pp*′DDT, the active insecticidal ingredient, and 15% *op*′DDT with minor contributions of *pp*′ and *op*′ DDEs and DDDs (ATSDR, 2002).

The minor component *op*′DDT along with its degradation products *op*′DDE and *op*′DDD is estrogenic and *pp*′DDE, the compound most commonly found in the environment, is an anti-androgen. These effects were initially noticed in humans and mammals but have also been shown for fish both in vitro and in vivo (Baatrup and Junge, 2001; Bayley et al., 2002; Okoumassoun et al., 2002; Uchida et al., 2010). DDT was also related to effects on thyroid function in fish (Brar et al., 2010).

The other pesticides in this study, while less intensely studied than DDT, are also all known or suspected endocrine disruptors in fish. For example, chlordane was linked to thyroid problems in wild fish (Brar et al., 2010), Lindane (γ-HCH) caused reduction in sex steroid hormones along with other effects on the reproductive axis of both sexes of catfish (Singh and Canario, 2004), endosulfan was shown in vitro to stimulate medaka estrogen receptor α (Chakraborty et al., 2011) and HCB exposure increased estradiol in females and reduced 11-keto-testosterone in males of crucian carp (Zhan et al., 2000).

1.4. Study area and aims

The river Thames is the longest river entirely in England (about 255 km from the source to the tidal limit west of London). Eel fisheries in its lower reaches have been reported as far back as the Domesday Book of 1086, but eel recruitment all but disappeared due to heavy pollution around London from the industrial revolution of the 19th century until sewage treatment improved water quality from the 1960s (DEFRA, 2010). Today, there is a relatively small commercial eel fishery in the lower reaches of the Thames, which reported catches of 7 t of yellow eels and 0.5 t silver eel in 2007 (the year of this study). Slightly smaller numbers were removed more recently (3.8 t yellow and 0.3 t silver eels in 2013).

Apart from two individual eels caught in 1995 (Yamaguchi et al., 2003) and one composite sample from the estuary (Santillo et al., 2005), we are not aware of any previous studies of persistent organic pollutants in river Thames eels. The aims of this study were therefore to examine what recent level of contamination with PCBs and organochlorine pesticides occurred in eels from the lower Thames and to review this with respect to previous UK and European studies and environmental quality standards.

Recognizing the usefulness of eels for monitoring long-term water quality as well as the consideration, that spawner *quality* is likely to be as important as *quantity* for successful eel reproduction, an eel quality database has recently been set up (Belpaire

Table 1
Summary of the main determinants in this study. All values given as mean (standard deviation, range).

Determinand	Unit	Non-tidal Thames [fresh weight]	Thames estuary [fresh weight]	Sig. diff?[a]	Non-tidal Thames [lipid weight]	Thames estuary [lipid weight]	Sig. diff?[a]	Banned in UK[b]	EQS
Fishing date		13.9.2007	1.10.2007	–					
Number	–	11	24	–					
Length	cm	51 (9.0, 35–62)	46 (7.9, 36–67)	10%					
Weight	g	228 (133, 60–482)	186 (142, 75–667)	n.s.[c]					
Age[d]	y	12 (3, 7–18)	9 (2, 6–14)	5%					
Fulton's condition factor[e]	–	0.15 (0.03, 0.12–0.20)	0.18 (0.03, 0.12–0.26)	10%					
Lipid content	%	10.0 (9.1, 1.7–29)	16.5 (8.3, 5.1–36)	5%					
Number of *A. crassus*[f]	–	2.6(2.7, 0–10)	1.0 (1.7, 0–7)	10%					
PCBs (Sum 46)[g]	µg kg^{-1}	63 (43, 7.3–166)	113 (50, 56–232)	5%	877 (540, 303–1854)	746 (239, 408–1408)	n.s.	in stages from 1972[h]	
Sum ICES7 PCBs[i]	µg kg^{-1}	33 (21, 4.2–79)	56 (24, 28–124)	5%	472 (295, 166–1007)	375 (132, 200–753)	n.s.		
Sum ICES6 PCBs[j]	µg kg^{-1}	26 (17, 3.5–63)	48 (20, 25–104)	5%	380 (235, 132–789)	325 (112, 172–630)	n.s.		
Mono-ortho PCBs as partial WHO1998 TEQ (mammals)[k,l]	ng kg^{-1}	1.6 (1.1, 0.2–4.1)	1.9 (0.9, 1.0–4.8)	n.s.	22 (14, 8.0–49)	13 (5.1, 6.5–29)	10%		Canada:0.79[m]
mono-ortho PCBs as partial WHO2005 TEQ[k,n]	ng kg^{-1}	0.32 (0.22, 0.035–0.83)	0.39 (0.19, 0.19–1.0)	n.s.	4.6 (3.0, 1.7–10)	2.6 (1.1, 1.3–6.1)	10%		EU:6.5[o]
Total DDT[p]	µg kg^{-1}	15.7(9.6, 1.7–38)	18.2 (7.8, 8.6–35)	n.s.	236 (167, 66–528)	124 (48, 57–229)	10%	1981[q]	Canada:14[r]
op′DDT	µg kg^{-1}	0.047 (0.046, 0.001–0.14)	0.059 (0.050, 0.01–0.23)	n.s.	0.57 (0.49, 0.04–1.5)	0.37 (0.23, 0.09–0.91)	n.s.		
pp′DDT	µg kg^{-1}	2.2 (1.5, 0.24–5.2)	1.5 (1.1, 0.57–4.9)	n.s.	43 (60, 6.7–217)	10 (6.3, 2.9–27)	1%		
pp′DDE	µg kg^{-1}	10.0 (5.9, 1.3–22)	10.9 (5.2, 4.4–25)	n.s.	147 (95, 41–336)	76 (35, 30–150)	1%		
α-chlordane	µg kg^{-1}	0.42 (0.32, 0.03–1.2)	0.46 (0.47, 0.08–2.0)	n.s.	5.3 (3.2, 1.8–11)	2.7 (1.8, 0.65–7.8)	0.5%	1981[q]	
γ-chlordane	µg kg^{-1}	0.13 (0.12, 0.003–0.43)	0.54 (0.31, 0.11–1.3)	0.5%	1.4 (0.78, 0.16–3.0)	3.6 (1.9, 1.1–7.0)	0.01%	1981[q]	
γ-HCH (Lindane)	µg kg^{-1}	0.58 (0.54, 0.05–1.9)	1.1 (0.71, 0.27–2.8)	1%	6.0 (1.9, 3.2–8.9)	6.4 (2.3, 3.5–14)	n.s.	2002[s]	
β-endosulfan	µg kg^{-1}	0.06 (0.06,<0.02–0.23)	0.22 (0.11, 0.09–0.50)	0.05%	0.71 (0.29, 0.33–1.1)	1.4 (0.40, 0.82–2.2)	0.01%	2007[t]	
HCB	µg kg^{-1}	1.9 (1.7, 0.05–6.4)	2.5 (1.6, 0.82–6.4)	n.s.	21 (12, 2.8–38)	15 (5.9, 7.7–29)	n.s.	1981[q]	EU:10[o]

[a] Significance level in Student's *t*-tests (for equal or unequal variance as determined with *F*-test (5% level)), on log transformed data for the chemical analysis, and on untransformed data for the other parameters.
[b] Or severely restricted (de facto ban).
[c] n.s.: not significant at 10% level.
[d] Years continental age, determined from stained otolyths. In a few cases the age could not be accurately determined and was for statistical purposes instead estimated from the linear length/age relationship of these eels.
[e] Weight[g]/(length[cm])^3 * 100.
[f] Juveniles + adults, no larval stages were found.
[g] 46 PCBs (see Section 2.1).
[h] Open uses prohibited 1972, ban in all new systems 1986, most existing equipment with > 5 L 2000 (The UK Department of the Environment, 1997; DEFRA, 2002).
[i] Commonly found congeners 28,52,101,118,138,153, and 180.
[j] ICES7 without the dioxin-like congener 118.
[k] To calculate the complete TEQ, dioxins, furans, and non-ortho-substituted PCBs would also need to be measured.
[l] Van den Berg et al. (1998).
[m] Canadian Council of Ministers of the Environment (2001) for dioxin-like PCBs.
[n] Van den Berg et al. (2006).
[o] European Union (2013) for dioxins, furans and dioxin-like PCBs.
[p] sum of pp′DDT, op′DDT, pp′DDE, op′DDE, pp′DDD, op′DDD.
[q] EEC (1978).
[r] Canadian Council of Ministers of the Environment (1999).
[s] European Commission (2000), technical HCH, which is typically dominated by the α-congener was already banned 1981 EEC (1978).
[t] European Commission (2005a).

et al., 2011a). This study can help to address the relative lack of recent UK data in that database.

2. Material and methods

2.1. Sampling sites and eel collection

Eels were caught at two locations in the lower part of the river Thames in autumn 2007 (for numbers of fish and biometrical data refer to Table 1 or the supplementary information): Both sites are in the Greater London area about 55 river km apart (Fig. 1). The stretch between Sunbury and Molesey (about 12–17 km upstream of the tidal limit, NGR TQ105681 to TQ144692) lies upstream of central London and was chosen as a non-tidal reach that is low in the catchment and therefore likely to contain sufficient numbers of eels. Eels from that reach were caught by electrofishing with a boom boat. The tidal reach is in the Thames estuary near Woolwich, downstream of Central London, about 42 river km from the tidal limit and about 50 km from the sea (NGR TQ438796). This is an area of commercial eel fishing and the eels from this site were caught by commercial fishermen using fyke nets. All eels were returned to the laboratory alive and sacrificed 2 or 5 weeks later. They were assessed for parasite infections by dissection and microscopy in a commercial laboratory (Thames Valley Aquatic Services, 2007) and sections of eel were frozen in fluoro-ethylene-propylene bags and stored at −80 °C for 16 months until analysis. Silvering stage was not determined, but most of the individuals are likely to have been in the yellow eel stage, because migrating eels use preferentially the deeper middle part of the river which is unsuitable for fyke nets and also too deep for efficient electro fishing (personal communication from Darryl Clifton-Dey, Environment Agency).

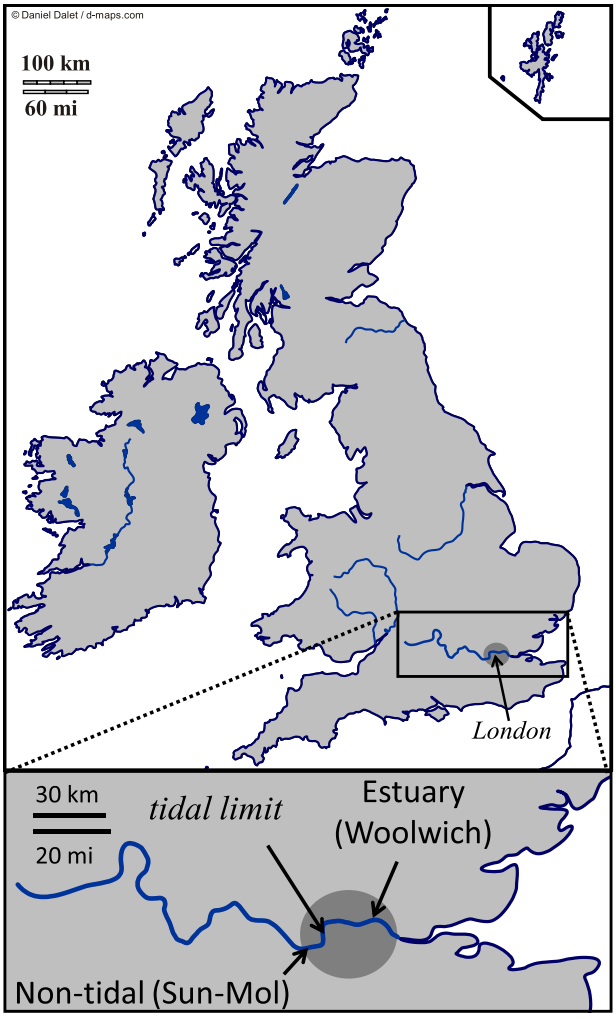

Fig. 1. Approximate locations of the eel sampling sites on the river Thames (outline © Daniel Dalet/d-maps.com).

Five of the upstream eels and 15 of the tidal ones have been analysed for otolith microchemistry (Walker et al. *in preparation*). This revealed that all had initially recruited to freshwater with those caught upstream never having returned to higher salinity. Three of the tidal eels analysed, also showed only a freshwater signal, suggesting that they had very recently arrived in the estuary from upstream, but only one of those also had the high (>20%) fat content typical of migrating silver eels. Two others had a "nomadic" signal of having moved between fresh and brackish water more than once and the rest had returned to the estuary after initially recruiting to freshwater.

2.2. Sample preparation and analysis

A portion from the central section of the eels (muscle, skin and bones) was homogenized with sodium sulphate to remove water, then $^{13}C_{12}$ –labelled ICES6 PCBs (#28, 52, 101, 138, 153, 180, Cambridge Isotope Laboratories, Andover, Massachusetts) were added as recovery standards and the sample was extracted for about 16 h with DCM in a soxhlet apparatus. Procedural blanks of sodium sulphate with internal standards were run with every batch. The DCM was solvent-exchanged to hexane which was added to a glass column with 11 g acidified silica (200 mL silica baked at 450 °C and acidified with 25 mL concentrated sulfuric acid) and eluted with hexane as a first clean up step, which removes the fats. The eluent was reduced by vacuum rotary evaporation and a subsequent cleanup was performed using gel permeation chromatography (GPC) employing a 25 mm internal diameter column containing 6 g Bio-Beads S-X3 (Bio-Rad Laboratories Ltd., Hemel Hempstead, Hertfordshire, UK) and eluting with a 1:1 v/v mixture of hexane and DCM to remove molecules outside the size range of interest. The final extract was solvent exchanged into 25 μL dodecane containing internal standards (PCB30, ^{13}C-PCB141, ^{13}C-PCB208, Wellington Laboratories Inc., Guelph, Ontario, Canada). The extracts were analysed by gas GCMS in negative chemical ionisation (NCI) mode (30 m, DB-5, 0.25 μm ID, 0.1 μm film, J&W Scientific) for HCH and endosulfan and electron impact (EI+) mode (50 m CPSil8, 0.25 mm ID, 0.12 μm film, Varian) for the other pesticides and PCBs. Target analytes were PCBs 18,22,28,31,30,41,44,49,52,54,56,60,64,70,74,87,90,101,95,99,104,105,110,114,118,123,132,138,141,149,151,154,155,156,157,158,167,170,174,180,183,187,188,189,194,199,203, *o,p′*-DDT, *p,p′*-DDT, *o,p′*-DDE, *p,p′*-DDE, *o,p′*-DDD, *p,p′*-DDD, α-endosulfan, β-endosulfan, endosulfan sulphate, α-chlordane, γ-chlordane, α-HCH, β-HCH, γ-HCH, δ-HCH and HCB (standards from Wellington Laboratoris Inc., Guelph Ontario, Canada).

Lipid content was determined by weighing the air-dried residue from a soxhlet extract of an adjacent body section to the one analysed for PCBs and pesticides.

3. Results and discussion

3.1. Parasites, condition factor, and lipid content

About half of the estuary eels and all but two of the 11 non-tidal ones were infected with adult or juvenile stages of the nematode *A. crassus*, no larval stages were found. The estuary eels tended to have a higher lipid content and a higher Fulton's condition factor (K = weight[g]/(length[cm])^3 ∗ 100) than their upstream

counterparts, which could not simply be explained by different size ranges (Table 1). These parasite and lipid results confirm findings of a German study (Marohn et al., 2013), which found higher fat content and lower *A. crassus* infections in eels from coastal or estuarine regions than in those from freshwater. Fat contents above about 20% of body weight appear to be necessary for successful migration and spawning (e.g., Belpaire et al., 2009). This makes the tidal Thames eels possibly better candidates for successful spawning, despite the fact that due to their higher lipid content they were slightly more contaminated. All chemicals measured were strongly related to lipid content of the individuals, while correlations to length or weight were much weaker. Both fresh weight and lipid-normalised data are given in Table 1 and the supplementary material available online, but we focus the discussion on fresh weight concentrations because regulatory values are framed that way.

3.2. PCBs

Most of the PCBs, including all seven indicator PCBs (ICES7), were detectable in every one of the eel samples from 2007, despite them having been banned from use in open systems in the UK since the early 70s and in closed systems since 1981. Total PCB levels (46 congeners) ranged from 7 to 232 μg kg^{-1}, fresh weight with the ICES7 indicator PCBs providing about half of that (Table 1). These values are towards the lower end of recent European measurements and fairly typical for recent UK data (see Table 2). Although the high PCB values reported in some UK sites in the 1990s (Table 2) were not repeated in this and other recent studies, there is insufficient data to show a clear trend over time for the UK. More extensive data exists for Belgium, where there is evidence that PCB contamination has decreased recently at a rate which would take about 14 years to reduce by an order of magnitude (Maes et al., 2008).

A number of PCBs have structural features that are similar to 2,3,7,8-tetra-chloro-dibenzo-dioxin (TCDD). These "dioxin-like" PCBs are the non-ortho and mono-ortho substituted PCBs and have been assigned toxicity equivalency factors (TEF) by the World Health Organization (Van den Berg et al., 1998,2006). There are indications that contamination with dioxin-like PCBs has adverse effects on eels: For example Sures and Knopf (2004) found that the most potent dioxin-like PCB126 (not analysed here) completely suppressed the immune response of eels experimentally infected with the nematode *A. crassus*, making them much more susceptible to this disease.

The European Union (European Union, 2013) recently agreed on a biota EQS to protect wildlife and humans from dioxin-like toxicity of 6.5 ng kg^{-1} for the sum of dioxins, furans and dioxin-like PCBs expressed as WHO 2005 TEQ, which is the same as the EU food standard for fish other than eel (European Commission, 2011). Of the dioxin-like substances only the mono-ortho PCBs were measured here and on their own contribute a maximum of 1 ng kg^{-1} (average 0.37) WHO 2005 TEQ. Canada has a more stringent tissue residue guideline of 0.79 ng kg^{-1} (WHO 1998 TEQ for mammals and humans) for the protection of wildlife consumers from PCBs in their prey (Canadian Council of Ministers of the Environment, 2001). This is based on studies with mink and includes a safety factor of 10 in case other mammalian predators are more sensitive. All but two of the eels analysed here (both from the non-tidal reach) exceeded this Canadian threshold even just for the mono-ortho substituted PCBs alone. The difference between passing the EU standards (at least for the measured part of the dioxin-like toxicity) and failing the Canadian ones is due both to the difference in EQS (6.5 ng kg^{-1} vs 0.79 ng kg^{-1}) and to the Canadian use of the older WHO 1998 assessment factors (Van den Berg et al., 1998), which assigned higher toxicity relative to 2,3,7,8 TCDD to the mono-ortho substituted PCBs, than the updated 2005 factors (Van den Berg et al., 2006). None of the lower Thames eels exceeded the food standards (European Commission, 2011) for eel for non-dioxin-like PCBs (300 μg kg^{-1}, sum of 6 ICES congeners) or dioxin-like toxicity (10 ng kg^{-1}, WHO 2005 TEQ), but as above, not all of the chemicals contributing to the TEQ were measured.

3.3. Organochlorine pesticides

All of the organochlorine pesticides and most of their by-products or degradation products were detected in the eel tissue despite having been banned or severely restricted decades ago (Table 1 and supporting information). The largest contribution to the pesticide burden is from the main DDT degradation product *pp′*DDE, which contributes on average 49% (SD 9%) to the total pesticides measured, with *pp′*DDD contributing a further 21% (SD 5%) (Table 1). The concentrations of *pp′*DDE ranged from 1.3 to 22 μg kg^{-1} fresh weight (average 10.0) in the upstream eels and from 4.4 to 25 μg kg^{-1} (average 10.9) in the tidal ones, with total DDT 1.7–38 (average 15.7) and 8.6–35 μg kg^{-1} (average 18.2) respectively. There is currently no EQS for DDT in the EU, but the Canadian tissue residue guidelines can give an idea as to whether contamination with that pesticide may be problematic to predators. The limit is 14 μg kg^{-1} for total DDT, which is based on the most sensitive endpoint (eggshell thinning in birds) with a safety factor of 10, to account for species differences, and the precautionary assumption that all members of the DDT family are as toxic as the most commonly studied *pp′*DDT (Canadian Council of Ministers of the Environment, 1999). At both sites more than half of the eels exceeded this value, suggesting that there may be some concern from the pesticide burden in particular to avian predators. It is however unclear, whether this level of pesticide contamination has an effect on the eels themselves.

The next-highest pesticide contribution was from HCB, which was on average 1.9 μg kg^{-1} fresh weight in the upstream eels and 2.5 μg kg^{-1} in the tidal ones (maximum 6.4 μg kg^{-1} for both groups). An EQS of 10 μg kg^{-1} fresh weight exists for HCB (European Union, 2013), which is not exceeded in any of the studied individuals. Lindane concentrations were on average 0.58 (0.05–1.9) and 1.1 (0.27–2.8) μg kg^{-1} in the two groups and α-Chlordane averaged 0.42 (0.03–1.2) μg kg^{-1} in the upstream eels and 0.46 (0.08–2.0) μg kg^{-1} in the tidal ones with γ-chlordane adding an average of 0.13 (0.003–0.43) and 0.54 (0.11–1.3) μg kg^{-1}. The β-endosulfan concentrations were never more than 0.5 μg kg^{-1}, with averages of 0.06 (<0.02–0.23) and 0.22 (0.09–0.50) μg kg^{-1} for the upstream and tidal groups. Of the pesticides measured, only the DDT family exceed the EU default limit for pesticide residues in food of 10 μg kg^{-1}, but for total DDT the much higher limit of 1000 μg kg^{-1} applies. The food limits for the other pesticides in this study are between 20 and 200 μg kg^{-1} (European Commission, 2005b).

The contamination of eels with DDE in this study was lower than much of the previously published UK and recent European eel data summarized in Table 2. Lindane was comparable to some studies from France and Italy but lower than in previous UK and recent studies from Germany and the Benelux countries. The lower values of those chemicals compared to older UK studies may reflect the expected declining trend following a ban. However, since the sites, sizes and methods vary between studies, such conclusions are only tentative. HCB was not measured in older UK studies and was in a similar range as most recent European studies that measured this chemical. Temporal downwards trends for some of these chemicals have been observed more clearly in other countries, for example in Belgium, where large numbers of eels were analysed over 11 years: Lindane concentrations fell by almost two orders of magnitude during that time, whereas the reduction

Table 2
Previous UK and recent European literature data for selected contaminants in yellow or silver eel (μg kg^{-1} fw) compared to the present study (in bold), median and range of site averages. Sorted by country and sampling date. Some data estimated from graphs or calculated from values given by lipid content or dry weight.

Year(s) of capture	Locations	Number of sites	Samples per site	DDE	γ-HCH (lindane)	HCB	ICES7 PCB	References
United Kingdom								
1983	Sheep dip impacted sites, SW England[a,b]	4	6–8	245 (77–298)	58 (30–79)	–	–	Hamilton (1985)
	Unimpacted sites, SW England[a]	3	7–8	54 (51–83)	48 (21–171)	–	–	
1984	Sheep dip impacted sites, SW England[a,b]	5	n.a.	<14 (<5–230)	–	–	–	
	Unimpacted sites, SW England[a]	3	n.a.	<15 (<5–<36)	–	–	–	
1985	Sheep dip impacted sites, SW England[a,b]	3	n.a.	<190 (<47–209)	–	–	–	
	Unimpacted sites, SW England[a]	1	n.a.	40	–	–	–	
1986	Urban sites in Scotland	8	1 Pooled	186 (43–557)	45 (25–63)	–	–	cited in Macgregor et al. (2010)
	Rural sites in Scotland	10	1 Pooled	322 (33–994)	33 (2.8–1413)	–	–	
	Mixed u/r sites in Scotland	2	1 Pooled	91 (61, 120)	56 (11 100)	–	–	
1991	Scottish Reed beds	11	1 Pooled	60 (<10–270)	–	–	Ca. 20 (ca. 3–ca. 250)[c]	Mason (1993)
1994/95	Contaminated sites Sussex, S England	18	5	79 (18–635)	16 (<0.1–60)	–	26 (6.8–383)[d]	Foster and Block (2006)
1995/96	Rivers Thames & Windrush SE England	2	2	–	3.3 (1.6, 4.9)	–	<13[e]	Yamaguchi et al. (2003)
1996	River Severn, W England/Wales	2	5 Pooled	–	–	–	100 (92 109)	Harrad and Smith (1999)
2004–08	Urban sites in Scotland	12	5	49 (<1–225)	<3.9 (<1–4.68)	ca. 1.5 (⩽1–ca. 2.5)	69 (7.1–1878)	Macgregor et al. (2010)
	Rural sites in Scotland	14	5	84 (<1.5–358)	<3.9 (<1–2.82)	ca. 1.5 (⩽1.1–ca. 2.5)	15 (5.9–54)	
	Mixed u/r sites in Scotland	3	5	33 (12–51)	<1 (<1–4.79)	<1 (<1–1.8)	22 (15–172)	
2005	Thames estuary, SE England	1	1 Pooled	–	–	–	136	Santillo et al. (2005)
2005/06	Contaminated sites Sussex, S England	21	5	43 (11–178)	<1.5 (<1–<25)	–	29 (7.5–89)	Foster and Block (2006)
2007	**Thames, near London SE England**	**2**	**11, 24**	**10 (10, 11)**	**0.84 (0.58, 1.1)**	**2.2 (1.9, 2.5)**	**44 (33, 56)**	**Current study**
Ireland								
2005/07	Lakes and rivers	5–7	1 Pooled	3.2 (1.6–7.1)	0.21 (<0.2–0.45)	<0.9 (<0.5–<2)	3.9 (1.9–18.1)	McHugh et al. (2010)
France								
2004/05	Gironde	4	13–58[a]	–	–	–	316 (278–345)	Tapie et al. (2011)
2005–07	Adour estuary	3	3–7	0.48 (0.43–0.57)	0.34 (0.33–1.49)	Total range <1–9.1[f]	98 (48–370)	Tabouret et al. (2011)
2008	3 Lagoons	3	12–22	32 (3.3–273)	–	–	3.7 (2.4–4.6)	Amilhat et al. (2014)
2008–10	All of France grouped into 6 major basins	6	16–160	–	–	2.3 (0.7–26)	587 (186–1276)	ONEMA (2012)
2009–11	Loire	3	11–16[a]	–	–	–	137 (80–193)	Blanchet-Letrouvé et al. (2014)
Italy								
2002	Tuscany	7	15	2.8 (1.3–6.1)	0.82 (0.21–45)	0.09 (0.06–0.16)	8.8 (5.7–14)[g]	Corsi et al. (2005)
2005/06	Garigiliano estuary	1 × 3[h]	10	28 (17–38)	–	2.0 (0.75–5.9)	239 (138–622)	Ferrante et al. (2010)
2007/08	River, lake, lagoon	3	15–23	98 (15–162)	0.20 (0.06–0.20)	1.2 (0.27–5.6)	32 (7.9–269)[g]	Quadroni et al. (2013)
2008/09	Campania region	7	1–2	–	–	–	22 (11–195)[e]	Pacini et al. (2012)
2009	Polluted R. Tiber + clean Lake Bolzena	2	30, 6	37 (29, 45)	–	5.7 (4.4, 7.0)	126 (38, 214)	Pujolar et al. (2012)
Belgium								
2000–07	Flanders	48	1 Pooled	–	–	–	226 (11–7753)	Belpaire et al. (2011b)
2001–05	Flanders	260[i]	1–21[j]	37 (3.0–232)	3.0 (<0.03–2076)	4.3 (0.11–62)	263 (7–5252)	Belpaire (2008)
The Netherlands								
2004	Lakes, rivers and canals	8	1 Pooled[k]	75 (25–96)	6.7 (3.5–11)	16 (4.5–30)	869 (308–1281)	de Boer et al. (2010)

Table 2 (*continued*)

Year(s) of capture	Locations	Number of sites	Samples per site	DDE	γ-HCH (lindane)	HCB	ICES7 PCB	References
Luxembourg								
2007	North Luxemburg	3	3–9	–	–	–	78 (53–346)	Boscher et al. (2010)
Germany								
1998/00	River Rhine	15–25	3–25	75 (11–180)[l]	9 (3–46)	110 (5–260)	480 (210–1330)[e]	Heinisch et al. (2004) and Heinisch et al. (2005a,b, 2006a,b, 2007)[m]
1996–03	Berlin area	10–11	3–20	750 (350–3300)	20 (4–40)	–	460 (90–1450)	
1999	River Elbe	7–8	3–20	190 (65–400)	–	–	290 (125–540)	
Europe-wide								
2005	10 European countries	20	1 Pooled	–	–	–	122 (<7–1512)	Santillo et al. (2005)

[a] Only eels >30 cm.
[b] Includes a site that was thought to be un-impacted, but showed high levels of dieldrin and DDE.
[c] Estimated using the conversion arochlor1260 = 3.6 * ICES7 PCB (Weatherley et al., 1997).
[d] Calculated from the individual PCB concentrations given in that report.
[e] Only 6 congeners.
[f] Site averages were not calculated due to non-detects.
[g] Includes additional congeners.
[h] One area three times.
[i] Only samples from 2001 onwards chosen: 260 sampling occasions from 219 sites.
[j] Typically 5.
[k] 6 Annual pooled samples from 2001 to 2006 chosen for PCBs, but only one of those (2004) supplied for the other chemicals.
[l] Sum of *op′* and *pp′* DDE.
[m] Only eels >10% lipid.

was slower for HCB, α-HCH and total DDT (estimated to take between 20 and 25 years to reduce by one order of magnitude, Maes et al., 2008).

In Belgium, an eel quality index (EQI) has been developed (Goemans et al., 2003; Belpaire and Goemans, 2007) in recognition that for successful reproduction, the quality of potential spawners is as important as their quantity. This is based on an original dataset of eels from 303 Belgian sites and is now also used in other countries (e.g., Amilhat et al., 2014). For each site the mean concentrations were calculated for a number of chemicals; for each compound these means were then ranked and the 5%ile defined as background or reference value (RV). Eels are classed depending on how much they deviate from that value with log(conc/RV)<0.4, classed as "I: not deviating" 0.4–0.8 "II: slightly deviating", 0.8–1.2 "III: deviating" and > 1.2 "IV: strongly deviating". An average classification can then also be derived across different chemicals. For example, the total DDT RV is: 16 μg kg^{-1}, therefore less than $16 * 10^{0.4} = 40$ μg kg^{-1} is class I, and therefore high quality. According to the EQI, the eels in the current study were all class I for total DDT, *pp′*DDE, and lindane, while for PCBs 91% of the upstream and 75% of the estuary eels were class I with the rest class II and for HCB the largest number (16) are in class II with 11 and 8 in classes I and III respectively. Although this is a purely statistical approach and does not state whether the observed concentrations are toxic, it helps to compare data from different studies and shows that the observed concentrations of most of the measured chemicals in the lower Thames eels are comparable to those from some of the less contaminated sites in Belgium.

3.4. Significance of pollutants in eels

In general the principle of assessing the risks of chemicals and setting appropriate standards is based on the most sensitive species and most sensitive endpoints observed, which should then (usually with some safety factor to account for a lack of data about the species or endpoints not analysed) be sufficient to protect any other species too. With regards to eels, there are however some difficulties with this approach. Until relatively recently, it was assumed that eels are fairly tolerant to pollution since they were observed in a very wide range of habitats including those with high organic loads and low oxygen content. However, very little is known about the critical life-stages of sexual maturation and spawning when, due to prolonged fasting, pollutants stored in the lipid can be re-mobilized and may affect either the eels themselves or their offspring via maternal transfer (Robinet and Feunteun, 2002). As it has so far neither been possible to observe most of the migration or the spawning or the early larval development at sea nor conduct entirely successful reproduction of European eels in captivity (for Japanese eels a full life-cycle in captivity was achieved for the first time as recently as 2010 (Ijiri et al., 2011)), we cannot yet know what the critical chemical thresholds are.

For the reasons mentioned in the introduction, eels are probably the best species for monitoring water quality, but that alone would not justify the use of a critically endangered species, as other organisms or methods are also suitable (see discussion in Jürgens et al., 2013). However, given that we still do not know for sure **why** their numbers are declining and therefore we do not know what, if anything, can be done to reverse the trend, it is necessary to learn as much as possible about eels. This includes their pollution status, especially with chemicals that may interfere with aspects of reproduction. For that reason the removal of a number of eels for analysis is justifiable and will give insights with regards to the state of the eels as well as the state of the watercourses from which they are taken.

While it is likely that the chemical pollution adds to the problems eels are facing, this alone does not seem to explain the phenomenon of the sharply declining eel numbers, given that the decline of eel recruitment corresponds to a period of generally *improving* water quality across Europe and reducing pollutant burdens in eels. However, as yet, chemicals cannot be completely ruled out, because due to the long generation times, effects on aspects of reproduction may only become apparent many years after an exposure. Climate change, water pollution, overfishing (including predation by fish eating birds), obstacles such as locks, and diseases or parasites may all be contributing factors to the decline (OSPAR Comission, 2010).

4. Conclusions

- The contamination of the 2007 Thames eels with PCBs and organochlorine pesticides appears to be relatively low compared to other UK and European studies.
- Eels from the estuary were slightly more contaminated than those from the non-tidal reach, but they also had higher lipid contents and condition factors and lower infection rates with *A. crassus*, making them possibly better spawning candidates overall.
- While none of the measured chemicals exceeded European food or environmental standards (although in the case of dioxin-like toxicity, only a small proportion of the contributing chemicals has been measured), over half the eels exceeded a Canadian tissue residue guideline to protect wildlife consumers from effects of total DDT and all but two individuals exceeded the equivalent Canadian guideline for dioxin-like PCBs, even though not all the congeners contributing to the standard were measured.
- Although not as highly contaminated with persistent organic pollutants as some of the eels from previous UK and European studies, the presence of so many of these harmful chemicals in the 2007 lower Thames eels may be a matter of concern for these fish, adding to other known or suspected problems eels face, such as fishing, infection with parasites, barriers impeding both upstream and downstream migration and climate change. Reducing the chemical burden alongside other measures should help towards the recovery of European eel populations.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.chemosphere.2014.06.088.

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