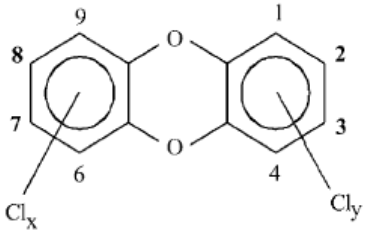
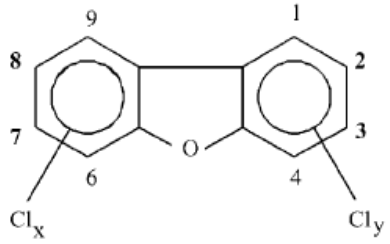
POLYCHLORINATED DIBENZO-*P*-DIOXINS (PCDDS), POLYCHLORINATED DIBENZOFURANS (PCDFS), AND DIOXIN-LIKE POLYCHLORINATED BIPHENYLS (DL-PCBS)

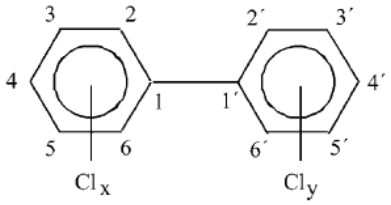
This EQS dossier was prepared by the Sub-Group on Review of the Priority Substances List (under Working Group E of the Common Implementation Strategy for the Water Framework Directive).

The dossier was reviewed by the Scientific Committee on Health and Environmental Risks (SCHER), which commented that the QS for sediment and the QS for secondary poisoning required better justification, and that more recent reviews regarding dioxin effects should be consulted. This has been done, and the dossier has been revised. The $QS_{biota,\ hh}$ is identified as the critical EQS because of consensus regarding the value used in existing food legislation and because there is greater uncertainty regarding the values calculated for $QS_{sec\ pois}$.

1 CHEMICAL IDENTITY

Common name	Dioxins and dioxin-like compounds (sometimes dioxins and dioxin-like compounds are referred to as "dioxins"). They include: ▪ seven polychlorinated dibenzo-*p*-dioxins (PCDDs) out of 75 theoretical possible congeners; ▪ 10 polychlorinated dibenzofurans (PCDFs) out of 135 theoretical possible congeners; ▪ 12 dioxin-like polychlorinated biphenyls (DL-PCBs) out of 209 theoretical possible congeners.
Chemical name (IUPAC)	
Synonym(s)	
Chemical class (when available/relevant)	
CAS number	<u>PCDDs</u> 2,3,7,8-T_4CDD 1746-01-6 1,2,3,7,8-P_5CDD 40321-76-4 1,2,3,4,7,8-H_6CDD 39227-28-6 1,2,3,6,7,8-H_6CDD 57653-85-7 1,2,3,7,8,9-H_6CDD 19408-74-3 1,2,3,4,6,7,8-H_7CDD 35822-46-9 1,2,3,4,6,7,8,9-O_8CDD 3268-87-9 <u>PCDFs</u> 2,3,7,8-T_4CDF 51207-31-9 1,2,3,7,8-P_5CDF 57117-41-6 2,3,4,7,8-P_5CDF 57117-31-4 1,2,3,4,7,8-H_6CDF 70648-26-9

	1,2,3,6,7,8-H_6CDF	57117-44-9
	1,2,3,7,8,9-H_6CDF	72918-21-9
	2,3,4,6,7,8-H_6CDF	60851-34-5
	1,2,3,4,6,7,8-H_7CDF	67562-39-4
	1,2,3,4,7,8,9-H_7CDF	55673-89-7
	1,2,3,4,6,7,8,9-O_8CDF	39001-02-0
	DL-PCBs	
	3,3',4,4'-T_4CB [77]	32598-13-3
	3,3',4',5-T_4CB [81]	70362-50-4
	2,3,3',4,4'-P_5CB [105]	32598-14-4
	2,3,4,4',5-P_5CB [114]	74472-37-0
	2,3',4,4',5-P_5CB [118]	31508-00-6
	2,3',4,4',5'-P_5CB [123]	65510-44-3
	3,3',4,4',5-P_5CB [126]	57465-28-8
	2,3,3',4,4',5-H_6CB [156]	38380-08-4
	2,3,3',4,4',5'-H_6CB [157]	69782-90-7
	2,3',4,4',5,5'-H_6CB [167]	52663-72-6
	3,3',4,4',5,5'-H_6CB [169]	32774-16-6
	2,3,3',4,4',5,5'-H_7CB [189]	39635-31-9
EU number	2,3,7,8-T_4CDD	1746-01-6
Molecular formula	PCDD $C_{12}Cl_xH_yO_2$ (x and y = 1–8) PCDF $C_{12}Cl_xH_yO$ (x = 4–8 and y = 0–4) PCB $C_{12}Cl_xH_y$ (x = 4–7 and y = 3–6)	
Molecular structure	 Seventy-five theoretical PCDD congeners (seven with binding affinity with the Ah receptor)  One-hundred-thirty-five theoretical PCDF congeners (10 with binding affinity with the Ah receptor)	

	Only the PCDD and PCDF congeners presenting chlorine at positions 2, 3, 7, and 8 have been reported to be toxic (WHO, 1989; IARC, 1997)  Two-hundred-nine theoretical PCB congeners (12 with binding affinity with the Ah receptor: these DL-PCBs exhibit *ortho*-positions which are unsubstituted or mono-chlorosubstituted and a chlorination degree greater than three)
Molecular weight	T_4CDDs 321.98 P_5CDDs 356.42 H_6CDDs 390.87 H_7CDDs 425.31 O_8CDD 460.76 T_4CDFs 305.98 P_5CDFs 340.42 H_6CDFs 374.87 H_7CDFs 409.31 O_8CDF 444.76 T_4CB 291.99 P_5CB 326.44 H_6CB 360.88 H_7CB 395.33

2 EXISTING EVALUATIONS AND REGULATORY INFORMATION

Annex III EQS Dir. (2008/105/EC)	Included
Existing Substances Reg. (793/93/EC)	No
Pesticides(91/414/EEC)	Not included in Annex I
Biocides (98/8/EC)	Not included in Annex I
PBT substances	Not investigated
Substances of Very High Concern (1907/2006/EC)	No
POPs (Stockholm convention)	Yes
Other relevant chemical regulation (veterinary products, medicament, etc.)	▪ Directive 96/82/EC (Seveso Directive) aims to prevent major accidents and hazards involving dangerous substances such as dioxins. ▪ Directive 96/59/EC contains stipulations regarding the elimination of PCBs. ▪ Directive 96/61/EC concerning integrated pollution prevention and control (IPPC) includes an information exchange on PCDD/PCDF prevention and abatement techniques, on associated emission values in the BREFs and on the development of emission values for PCDDs/PCDFs. ▪ Directive 2000/76/EC on the incineration of wastes sets an air emission limit value of 0.1 $ng_{TE}.m^{-3}$. ▪ Directive 2001/102/EC sets maximum content relative to feedstuff for animal nutrition. Dioxins are covered by this Directive. ▪ Regulation 2004/850/EC implements in the EU the provisions of the international agreements on POPs (i.e. the Stockholm Convention and the UN-ECE Protocol). Dioxins are covered by this Regulation. ▪ Recommendation 2006/88/EC concerning the reduction of the presence of dioxins, furans and PCBs in feeding stuffs and foodstuffs. ▪ Recommendation 2006/794/EC on the monitoring of background levels of dioxins, dioxin-like PCBs and non-dioxin-like PCBs in foodstuffs ▪ Regulation 1881/2006/EC setting maximum levels for certain contaminants in foodstuffs. Replaces Council Regulation 2375/2001 of 29 November 2001 amending Commission Regulation 466/2001/EC. ▪ Regulation 1883/2006/EC laying down methods of sampling and analysis for the official control of levels of dioxins and dioxin-like PCBs in certain foodstuffs
Endocrine disrupter	Several PCDD, PCDF, and PCB congeners are identified as substances with evidence of endocrine disruption (US-EPA, 1997; E.C., 2004).

It is to be noted as well the work done in the context of the Community strategy for dioxins, furans and polychlorinated biphenyls (COM(2001) 593) with the publication of two communications from the Commission, the Council, the European Parliament and the European Economic and Social Committee:

- First progress report in 2004 : COM(2004) 240
- Second progress report in 2007 : COM(2007) 396
- Third progress report in 2010 : COM(2010) 562

3 PROPOSED QUALITY STANDARDS (QS)

3.1 EXPRESSION OF CONCENTRATIONS FOR DIOXINS AND DL-COMPOUNDS

The present document aims at determining an EQS value for a group of substances so-called "dioxin and DL-compounds" representing polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and dioxin-like polychlorinated biphenyls (DL-PCBs). These compounds are considered together because they all elicit toxic effects through the Ah receptor and thereby contribute to dioxin-like toxic potency. Thus, the EQS for PCDD/Fs and DL-PCBs are intimately related and should be considered concurrently.

The Toxic Equivalence concept consists in converting the concentrations into toxic equivalents (TEQ), basing this conversion on the assumption that all 2,3,7,8-substituted PCDDs and PCDFs, as well as the dioxin-like PCBs, have the same mode of action, elicited by binding to the same receptor, the Ah receptor, and show comparable qualitative effects, but with different potencies. These differences in toxicity are expressed in the toxic equivalency factors (TEFs), estimated from the weaker toxicity of the respective congener in relation to the most toxic congener 2,3,7,8-TCDD, which is assigned the arbitrary TEF of 1.

TEF values have been defined in a 1998 publication for PCBs, PCDDs and PCDFs for humans and wildlife (Van den Berg *et al.*, 1998) and most recently revised during a WHO expert meeting in Geneva. The results of this reevaluation were published in another scientific article from the same authors (Van den Berg *et al.*, 2006) and are reported in the table here below. These are the most recent TEF value recognised by the scientific community but they are not taken into account yet in the European legislation. Indeed Regulation 1881/2006/EC setting maximum levels for certain contaminants in foodstuffs, for example dioxins and DL-compounds, uses the 1998's TEF values (E.C., 1998).[*]

List of TEF values for dioxin and DL-compounds defined by the WHO in one of its expert meeting and published in the scientific literature (Van den Berg *et al.*, 2006).

Compound	WHO 1998 TEF	WHO 2005 TEF	Compound	WHO 1998 TEF	WHO 2005 TEF
Chlorinated dibenzo-p-dioxins			**Non-*ortho*–substituted PCBs**		
2,3,7,8-TCDD	1	1	3,3',4,4'-tetraCB (PCB 77)	0.0001	0.0001
1,2,3,7,8-PeCDD	1	1	3,4,4',5-tetraCB (PCB 81)	0.0001	**0.0003**
1,2,3,4,7,8-HxCDD	0.1	0.1	3,3',4,4',5-pentaCB (PCB 126)	0.1	0.1
1,2,3,6,7,8-HxCDD	0.1	0.1	3,3',4,4',5,5'-hexaCB (PCB 169)	0.01	**0.03**
1,2,3,7,8,9-HxCDD	0.1	0.1			
1,2,3,4,6,7,8-HpCDD	0.01	0.01			
OCDD	0.0001	**0.0003**			
Chlorinated dibenzofurans			**Mono-*ortho*–substituted PCBs**		
2,3,7,8-TCDF	0.1	0.1	2,3,3',4,4'-pentaCB (PCB 105)	0.0001	**0.00003**
1,2,3,7,8-PeCDF	0.05	**0.03**	2,3,4,4',5-pentaCB (PCB 114)	0.0005	**0.00003**
2,3,4,7,8-PeCDF	0.5	**0.3**	2,3',4,4',5-pentaCB (PCB 118)	0.0001	**0.00003**
1,2,3,4,7,8-HxCDF	0.1	0.1	2',3,4,4',5-pentaCB (PCB 123)	0.0001	**0.00003**
1,2,3,6,7,8-HxCDF	0.1	0.1	2,3,3',4,4',5-hexaCB (PCB 156)	0.0005	**0.00003**
1,2,3,7,8,9-HxCDF	0.1	0.1	2,3,3',4,4',5'-hexaCB (PCB 157)	0.0005	**0.00003**
2,3,4,6,7,8-HxCDF	0.1	0.1	2,3',4,4',5,5'-hexaCB (PCB 167)	0.00001	**0.00003**
1,2,3,4,6,7,8-HpCDF	0.01	0.01	2,3,3',4,4',5,5'-heptaCB (PCB 189)	0.0001	**0.00003**
1,2,3,4,7,8,9-HpCDF	0.01	0.01			
OCDF	0.0001	**0.0003**			

Dose-additivity (i.e. assumption that the dose-response relationships are parallel for the different congeners) being a basic property of the TEF concept, the estimate of the toxic potency of a sample is the sum of the individual congener concentrations multiplied by their respective Toxic Equivalency Factors (TEFs).

[*] The assessor was informed however that the most recent 2005 TEF values would probably be taken into account in a new proposal for maximum levels in foodstuffs in a near future.

It has to be noted that the TEFs proposed by WHO 1998 and 2006 are all intake TEFs. They are consequently not directly applicable to other circumstances than those assessing the outcome of the total human exposure to dioxin-like chemicals from consumption of food products, breast milk, etc. Direct application of the TEFs for assessment of dioxins and DL-PCBs present in soil, sediment, or fly ash would lead to inaccurate assessment of the potential toxic potency of the matrix. This is primarily a result from the fact that the highly hydrophobic dioxins and DL-PCBs bind strongly to particles thereby significantly reducing their bioavailability for living organisms.

EQS estimated in this document are derived from biotic matrix (secondary poisoning for high predator, fish consumption and drinking water for human health), hence the intake TEFs are accordingly used. If a human risk assessment had to be performed for an abiotic matrix, it would rather be preferable to use congener-specific equations throughout the whole model rather than base it on total TEQ in an abiotic matrix.

In the present document, units were not reported as TEQ when it was not deemed relevant to, e.g. in many cases where effects and no effects data correspond to a single exposure to 2,3,7,8-TCDD.

3.2 ENVIRONMENTAL QUALITY STANDARD (EQS)

Because of their hydrophobic nature, the majority of PCDD/Fs and DL-PCBs released into aquatic systems ultimately become associated with the organic fraction of suspended and/or bed sediments and the lipid-rich tissues of aquatic organisms. Organisms are mainly exposed through accumulation in biota. The lowest QS has been calculated for predator exposed via the food chain. However, the calculated value, based on a study performed with 2,3,7,8 TCDD administered subcutaneously should be considered with caution for QS determination (see limitations and uncertainties in section 7.2). This calculated value is in the same range than the value recommended in Regulation (EC) No. 1881/2006 which is preferred. The EQS is driven by the standard for the protection of human health via consumption of fishery products. No EQS could be derived in sediment with the available information. Considering the properties of PCDD/Fs and DL-PCBs, EQS should be expressed preferentially in terms of concentration in biota.

	Value	**Comments**
Proposed AA-EQS in biota for sum of dioxins and DL-compounds [fresh and marine] [$\mu g_{WHO98\text{-}TEQ}.kg^{-1}_{ww}$]	$8\ 10^{-3}$	Critical QS is $QS_{biota,\ hh}$ **See section 7**
Corresponding AA-EQS [freshwater] Corresponding AA-EQS [marine water] [$\mu g_{WHO98\text{-}TEQ}.l^{-1}$]	$1.9\ 10^{-8}$ (freshwater) $1.9\ 10^{-9}$ (marine water)	High uncertainty due to aggregation of BCF for different congeners. Worst case taken into account. **See section 5.1 and 7.3**
Proposed MAC-EQS [fresh and marine waters] [$\mu g.l^{-1}$]	**Not possible to derive MAC values** **See section 7.1**	

3.3 SPECIFIC QUALITY STANDARD (QS)

Protection objective[†]	**Unit**	**Value**	**Comments**
Pelagic community (freshwater)	$[\mu g_{TEQ}.l^{-1}]$	Derivation not relevant	See section 7.1
Pelagic community (marine waters)	$[\mu g_{TEQ}.l^{-1}]$		
Benthic community (freshwater)	$[\mu g_{TEQ}.kg^{-1}_{dw}]$	Derivation not possible due to insufficient level of information on data	
	$[\mu g_{TEQ}.l^{-1}]$		
Benthic community (marine)	$[ng_{TEQ}.kg^{-1}_{dw}]$		
	$[\mu g_{TEQ}.l^{-1}]$		
Predators (secondary poisoning)	$[\mu g_{TEQ}.kg^{-1}_{biota\ ww}]$	$1.2\ 10^{-3}$	See section 7.2
	$[\mu g_{TEQ}.l^{-1}]$	$2.8\ 10^{-9}$ (freshwater) $2.8\ 10^{-10}$ (marine waters)	
Human health via consumption of fishery products	$[\mu g_{WHO98\text{-}TEQ}/kg_{ww}]$	Crustaceans or fish excluding eel: $8\ 10^{-3}$	See section 7.3
	$[\mu g_{WHO98\text{-}TEQ}.l^{-1}]$	Worst cases $1.9\ 10^{-8}$ (freshwater) $1.9\ 10^{-9}$ (marine waters)	

[†] Please note that as recommended in the draft Technical Guidance for deriving EQS (E.C., 2011), "EQSs […] are not reported for 'transitional and marine waters', but either for freshwater or marine waters". If justified by substance properties or data available, QS for the different protection objectives are given independently for transitional waters or coastal and territorial waters.

4 MAJOR USES AND ENVIRONMENTAL EMISSIONS

4.1 USES AND QUANTITIES

PCDDs and PCDFs are unintentionally formed and released from thermal processes involving organic matter and chlorine as a result of incomplete combustion or chemical reactions. In Annex C of the UNEP Convention on Persistent Organic Pollutants (POPs) the following industrial source categories have been identified to have the potential for comparatively high formation and release of these chemicals to the environment:

- waste incinerators, including co-incinerators of municipal, hazardous or medical waste or of sewage sludge;
- cement kilns firing hazardous waste;
- production of pulp using elemental chlorine or chemicals generating elemental chlorine for bleaching;
- thermal processes in the metallurgical industry (secondary copper production, sinter plants in the iron and steel industry, secondary aluminium production, secondary zinc production).

PCBs have been industrially produced in large amounts and for many years until their phasing-out in the western world in the 1970s–1980s. PCBs may also unintentionally form from thermal processes.

4.2 ESTIMATED ENVIRONMENTAL EMISSIONS

Air PCDD and PCDF emissions reported in the following table are derived from “The European Dioxin Emission Inventory, Stage II” as reported by EC DG ENV (Quass *et al.*, 2000). Values are based on results of the second stage of “The European Dioxin Project” implemented by the European Commission. Updated PCDD and PCDF emissions until 1995 for the most important emission sources in 17 western European countries and an evaluation of the emission time trend from 1995 to 2005 are presented in the following table. According to the assessment reported, for those industrial processes which were considered as the most relevant emission sources (i.e municipal solid waste incineration), a dramatic decrease of emissions between 1995 and 2005 can be observed (-81 %). This considerable improvement of the general situation concerning emissions to air was due to a large extent to abatement measures and plant closures carried out in the most industrialised member states. Contrary to that, a decrease of emissions (-13 %) from non-industrial sources such as domestic solid fuel combustion, making up more than 60 % of all non-industrial PCDD and PCDF emission sources, appears to be much less pronounced if compared to the industrial sector. This was predicted to result in a domination of non-industrial sources over the overall annual emissions of PCDDs and PCDFs in Europe as reported by EC DG ENV (Quass *et al.*, 2004).

Air PCDD and PCDF emission estimates (gTE/year) for the year 1995, 2000, and 2005 as reported by EC DG ENV (Quass *et al.*, 2000).

SNAP Code	Emission source	Revised for 1995	Actual data 2000	Projection 2005	Change 1995/2000	Change 1995/2005
01	Power plants	59–122	55–72	50–67	–30 %	–35 %
0202	Residue of combustion: boilers, stoves, fireplaces (wood)	544–989	532–971	523–969	–2 %	–3 %
0202	Residue of combustion: boilers, stoves, fireplaces (coal/lignite)	92–408	86–370	82–337	–9 %	–16 %
0301	Combustion in industry/boilers, gas turbines, stationary engines	32–83	34–81	39–78	0 %	–2 %
030301	Sinter plants	671–864	447–554	383–467	–35 %	–45 %
030308	Secondary zinc production	242–245	22–25	20–20	–90 %	–92 %
030309	Secondary copper production	31–33	15–17	15–17	–50%	–50%
030310	Secondary aluminium production	41–82	27–72	21–60	–20 %	–34 %
30311	Cement	14–50	13–49	14–50	–2 %	0 %
030326	Other: metal reclamation from cables	42–52	40–50	40–50	–3 %	–3 %
040207	Electric furnace steel plant	115–162	120–153	141–172	–1 %	+13 %
040309	Other: non-ferrous metal foundries	36–78	40–74	38–72	0 %	–4 %
040309	Other: sintering of special materials and drossing facilities	115–200	1–86	1–86	–72 %	–72 %
060406	Preservation of wood	145–388	131–349	118–310	–10 %	–20 %
0701	Road transport	57–138	37–82	41–60	–39 %	–48 %
090201	Incineration of domestic or municipal waste (legal combustion)	973–1213	412–506	178–232	–58 %	–81 %
09201	Incineration of domestic or municipal waste (illegal domestic combustion)	129–221	126–200	116–187	–7 %	–13 %
09202	Incineration of industrial waste (hazardous waste)	149–183	131–166	16–45	–10 %	–81 %
09207	Incineration of hospital waste	133–530	96–392	51–161	–27 %	–68 %
090901	Cremation: incineration of corpses	11–46	9–19	13–22	–51 %	–40 %
1201	Fires	54–382	60–371	60–371	–1%	–1%
Total of sources considered (gI-TE/year)		3685–6470	2435–4660	1959–3834	–30 %	–43 %
Industrial sources		2793–4165	1589–2516	1135–1786	–41 %	–58 %
Non-industrial sources		892–2305	846–2144	824–2048	–6 %	–10 %

5 ENVIRONMENTAL BEHAVIOUR

5.1 ENVIRONMENTAL DISTRIBUTION

Values reported in the table below are either estimated or calculated/extrapolated from experimental values.

			Master reference
Water solubility ($mg.l^{-1}$)	PCDDs		US-EPA, 2003
	2,3,7,8-T_4CDD	$1.93\ 10^{-5}$ (25°C)	
	1,2,3,7,8-P_5CDD	—	
	1,2,3,4,7,8-H_6CDD	$4.42\ 10^{-6}$ (25°C)	
	1,2,3,6,7,8-H_6CDD	—	
	1,2,3,7,8,9-H_6CDD	—	
	1,2,3,4,6,7,8-H_7CDD	$2.40\ 10^{-6}$ (20°C)	
	1,2,3,4,6,7,8,9-O_8CDD	$7.40\ 10^{-8}$ (25°C)	
	PCDFs		
	2,3,7,8-T_4CDF	$4.19\ 10^{-4}$ (22.7°C)	
	1,2,3,7,8-P_5CDF	—	
	2,3,4,7,8-P_5CDF	$2.36\ 10^{-4}$ (22.7°C)	
	1,2,3,4,7,8-H_6CDF	$8.25\ 10^{-6}$ (22.7°C)	
	1,2,3,6,7,8-H_6CDF	$1.77\ 10^{-4}$ (22.7°C)	
	1,2,3,7,8,9-H_6CDF	—	
	2,3,4,6,7,8-H_6CDF	—	
	1,2,3,4,6,7,8-H_7CDF	$1.35\ 10^{-6}$ (22.7°C)	
	1,2,3,4,7,8,9-H_7CDF	—	
	1,2,3,4,6,7,8,9-O_8CDF	$1.16\ 10^{-6}$ (25°C)	
	DL-PCBs		
	3,3',4,4'-T_4CB [77]	$1.0\ 10^{-3}$ (25°C)	
	3,3',4',5-T_4CB [81]	$2.92\ 10^{-3}$ (25°C)	
	2,3,3',4,4'-P_5CB [105]	$1.90\ 10^{-3}$ (25°C)	
	2,3,4,4',5-P_5CB [114]	$2.58\ 10^{-3}$ (20°C)	
	2,3',4,4',5-P_5CB [118]	$1.59\ 10^{-3}$ (20°C)	
	2,3',4,4',5'-P_5CB [123]	$1.64\ 10^{-3}$ (25°C)	
	3,3',4,4',5-P_5CB [126]	$1.03\ 10^{-3}$ (25°C)	
	2,3,3',4,4',5-H_6CB [156]	$4.10\ 10^{-4}$ (20°C)	
	2,3,3',4,4',5'-H_6CB [157]	$3.61\ 10^{-4}$ (25°C)	
	2,3',4,4',5,5'-H_6CB [167]	$3.61\ 10^{-4}$ (25°C)	
	3,3',4,4',5,5'-H_6CB [169]	$3.61\ 10^{-5}$ (25°C)	
	2,3,3',4,4',5,5'-H_7CB [189]	$6.26\ 10^{-5}$ (25°C)	

		Master reference
Volatilisation	Given the high potential of accumulation in organic tissues and the high potential to adsorb on particulate matter (see below), volatilisation and evaporation are not major processes of dissipation of DL-compounds from the water phase	
Vapour pressure (Pa)	<u>PCDDs</u> 2,3,7,8-T_4CDD: $2.0\ 10^{-7}$ 1,2,3,7,8-P_5CDD: $5.9\ 10^{-8}$ 1,2,3,4,7,8-H_6CDD: $5.1\ 10^{-9}$ 1,2,3,6,7,8-H_6CDD: $4.8\ 10^{-9}$ 1,2,3,7,8,9-H_6CDD: $6.5\ 10^{-9}$ 1,2,3,4,6,7,8-H_7CDD: $7.5\ 10^{-10}$ 1,2,3,4,6,7,8,9-O_8CDD: $1.1\ 10^{-10}$ <u>PCDFs</u> 2,3,7,8-T_4CDF: $2.0\ 10^{-6}$ 1,2,3,7,8-P_5CDF: $2.3\ 10^{-7}$ 2,3,4,7,8-P_5CDF: $3.5\ 10^{-7}$ 1,2,3,4,7,8-H_6CDF: $3.2\ 10^{-8}$ 1,2,3,6,7,8-H_6CDF: $2.9\ 10^{-8}$ 1,2,3,7,8,9-H_6CDF: — 2,3,4,6,7,8-H_6CDF: $2.7\ 10^{-8}$ 1,2,3,4,6,7,8-H_7CDF: $4.7\ 10^{-9}$ 1,2,3,4,7,8,9-H_7CDF: $1.4\ 10^{-8}$ 1,2,3,4,6,7,8,9-O_8CDF: $5.0\ 10^{-10}$ <u>DL-PCBs</u> 3,3',4,4'-T_4CB [77]: $5.96\ 10^{-5}$ 3,3',4',5-T_4CB [81]: $1.05\ 10^{-4}$ 2,3,3',4,4'-P_5CB [105]: $1.10\ 10^{-4}$ 2,3,4,4',5-P_5CB [114]: $5.57\ 10^{-5}$ 2,3',4,4',5-P_5CB [118]: $4.19\ 10^{-5}$ 2,3',4,4',5'-P_5CB [123]: $1.17\ 10^{-4}$ 3,3',4,4',5-P_5CB [126]: $3.95\ 10^{-5}$ 2,3,3',4,4',5-H_6CB [156]: $1.96\ 10^{-5}$ 2,3,3',4,4',5'-H_6CB [157]: $7.29\ 10^{-6}$ 2,3',4,4',5,5'-H_6CB [167]: $2.60\ 10^{-5}$ 3,3',4,4',5,5'-H_6CB [169]: $2.41\ 10^{-5}$ 2,3,3',4,4',5,5'-H_7CB [189]: $1.75\ 10^{-6}$	US-EPA, 2003

			Master reference
Henry's Law constant ($Pa.m^3.mol^{-1}$)	PCDDs		US-EPA, 2003
	2,3,7,8-T_4CDD	3.33	
	1,2,3,7,8-P_5CDD	—	
	1,2,3,4,7,8-H_6CDD	1.08	
	1,2,3,6,7,8-H_6CDD	—	
	1,2,3,7,8,9-H_6CDD	—	
	1,2,3,4,6,7,8-H_7CDD	1.28	
	1,2,3,4,6,7,8,9-O_8CDD	0.684	
	PCDFs		
	2,3,7,8-T_4CDF	1.46	
	1,2,3,7,8-P_5CDF	—	
	2,3,4,7,8-P_5CDF	0.505	
	1,2,3,4,7,8-H_6CDF	1.45	
	1,2,3,6,7,8-H_6CDF	0.741	
	1,2,3,7,8,9-H_6CDF	—	
	2,3,4,6,7,8-H_6CDF	—	
	1,2,3,4,6,7,8-H_7CDF	1.43	
	1,2,3,4,7,8,9-H_7CDF	—	
	1,2,3,4,6,7,8,9-O_8CDF	0.190	
	DL-PCBs		
	3,3',4,4'-T_4CB [77]	1.72	
	3,3',4',5-T_4CB [81]	13.0	
	2,3,3',4,4'-P_5CB [105]	10.1	
	2,3,4,4',5-P_5CB [114]	6.99	
	2,3',4,4',5-P_5CB [118]	8.61	
	2,3',4,4',5'-P_5CB [123]	17.6	
	3,3',4,4',5-P_5CB [126]	5.47	
	2,3,3',4,4',5-H_6CB [156]	88.2	
	2,3,3',4,4',5'-H_6CB [157]	58.8	
	2,3',4,4',5,5'-H_6CB [167]	11.2	
	3,3',4,4',5,5'-H_6CB [169]	6.61	
	2,3,3',4,4',5,5'-H_7CB [189]	6.74	

		Master reference
Adsorption	The value of $K_{sed-water}$ depends on several factors, including the type of sediment (e.g. grain size) and its organic carbon content (illustrated by K_{OC} values) and the chlorine content of the chemical. Therefore, a large variability can be expected. **For dioxins and DL-compounds, the range can be as wide as 30 – 7.9 10^6.**	
log Organic carbon – water partition coefficient (log K_{OC}) ($l.kg^{-1}$)	<u>PCDDs</u> 2,3,7,8-T_4CDD 3.06–8.50 1,2,3,4,7,8-H_6CDD 5.02–7.10 <u>PCDFs</u> 2,3,7,8-T_4CDF 5.20–7.50 1,2,3,4,7,8-H_6CDF 7.40 1,2,3,4,7,8,9-H_7CDF 5.00–6.70 1,2,3,4,6,7,8,9-O_8CDF 6.00–7.40 <u>DL-PCB</u> 3,3',4,4'-T_4CB [77] 4.41–5.75 A study from Tanaka et al. provides useful information for predicting the fates and toxicities of polychlorinated dibenzo-*p*-dioxins in the environment, actually it proves that K_{OC} values of hydrophobic organic pollutants such as DL-compounds are significantly related to the polarity and/or hydrophobicity of the humic substances (Tanaka *et al.*, 2005). This can be a reason for the wide range of log K_{OC} reported here.	US-EPA, 2003
Sediment – water partition coefficient ($K_{sed-water}$) ($m^3.m^{-3}$)	Calculated values based on the above K_{OC} values from US-EPA, 2003, using the equation recommended in the draft Technical Guidance for deriving EQS (E.C., 2010) $K_{sed-water}$ = 0,8+(0,2*((0,05*Koc)/1000)*2500) <u>PCDDs</u> 2,3,7,8-T_4CDD 30 – 7.9 10^6 1,2,3,4,7,8-H_6CDD 2 619 – 3.1 10^5 <u>PCDFs</u> 2,3,7,8-T_4CDF 3 963 – 7.9 10^5 1,2,3,4,7,8-H_6CDF 6.3 10^5 1,2,3,4,7,8,9-H_7CDF 2 501 – 1.3 10^5 1,2,3,4,6,7,8,9-O_8CDF 25 001 – 6.3 10^5 <u>DL-PCB</u> 3,3',4,4'-T_4CB [77] 643 – 14 059	US-EPA, 2003 E.C., 2010

		Master reference
Bioaccumulation / Biomagnification	According to the values reported thereafter, the BCF values for fish range from around 1 700 – 186 000 (log BCF range ~ 3.2 – 5.3). The range of value is very large depending on the congener considered. Therefore, **it is chosen to use the geometrical mean of the BCF values for derivation of quality standards: 41 540.** Whatever the choice made on the BCF value, it corresponds to **default BMF_1 and BMF_2 values of 10** according to the Technical Guidance for deriving EQS (E.C., 2011).	
Octanol-water partition coefficient (log Kow)	PCDDs 2,3,7,8-T_4CDD 6.80 1,2,3,7,8-P_5CDD 6.64 1,2,3,4,7,8-H_6CDD 7.80 1,2,3,6,7,8-H_6CDD — 1,2,3,7,8,9-H_6CDD — 1,2,3,4,6,7,8-H_7CDD 8.00 1,2,3,4,6,7,8,9-O_8CDD 8.20 PCDFs 2,3,7,8-T_4CDF 6.1 1,2,3,7,8-P_5CDF 6.79 2,3,4,7,8-P_5CDF 6.5 1,2,3,4,7,8-H_6CDF 7.0 1,2,3,6,7,8-H_6CDF — 1,2,3,7,8,9-H_6CDF — 2,3,4,6,7,8-H_6CDF — 1,2,3,4,6,7,8-H_7CDF 7.4 1,2,3,4,7,8,9-H_7CDF — 1,2,3,4,6,7,8,9-O_8CDF 8.0 DL-PCBs 3,3',4,4'-T_4CB [77] 6.5 3,3',4',5-T_4CB [81] 6.36 2,3,3',4,4'-P_5CB [105] 6.0 2,3,4,4',5-P_5CB [114] 6.65 2,3',4,4',5-P_5CB [118] 7.12 2,3',4,4',5'-P_5CB [123] 6.74 3,3',4,4',5-P_5CB [126] 6.89 2,3,3',4,4',5-H_6CB [156] 7.16 2,3,3',4,4',5'-H_6CB [157] 7.19 2,3',4,4',5,5'-H_6CB [167] 7.09 3,3',4,4',5,5'-H_6CB [169] 7.46 2,3,3',4,4',5,5'-H_7CB [189] 7.71	US-EPA, 2003
log BCF	Log BCF values for PCDD and PCDF congeners measured in fish (Guppy)	US-EPA, 2003

			Master reference
	PCDDs		
	2,3,7,8-T_4CDD	5.24	
	1,2,3,7,8-P_5CDD	5.27	
	1,2,3,4,7,8-H_6CDD	5.01	
	1,2,3,6,7,8-H_6CDD	4.94	
	1,2,3,7,8,9-H_6CDD	4.93	
	1,2,3,4,6,7,8-H_7CDD	4.68	
	1,2,3,4,6,7,8,9-O_8CDD	4.13	
	PCDFs		
	2,3,4,7,8-P_5CDF	5.14	
	1,2,3,6,7,8-H_6CDF	4.95	
	1,2,3,4,6,7,8-H_7CDF	4.46	
	1,2,3,4,6,7,8,9-O_8CDF	3.90	
	Log BCF value measured in various fish species		
	DL-PCB		
	3,3',4,4'-T_4CB [77]	3.24–4.15	

5.2 ABIOTIC AND BIOTIC DEGRADATIONS

Literature data indicate that PCDDs and PCDFs, particularly the tetra- and higher chlorinated congeners, are extremely stable compounds under most environmental conditions. The only environmentally significant transformation processes for these congeners are believed to be atmospheric photo-oxidation and photolysis of non-sorbed species in the gaseous phase or at the soil or water-air interface (US-EPA, 2003).

		Master reference
Hydrolysis	There is no available evidence indicating that hydrolysis would be an operative environmental process for degradation of dioxin-like compounds	US-EPA, 2003
Photolysis	*Half-life (days)* 2,3,7,8-T_4CDD 0.255–0.78 (water/organic solvent mixtures) 2,3,7,8-T_4CDF 0.25–1.2 (natural waters) 2,3,4,7,8-P_5CDF 0.19 (natural waters)	
Biodegradation	Several studies have indicated that certain ligninolytic fungi can degrade these higher-chlorinated congeners and that anaerobic degradation in sediment may occur at a slow rate. To a large extent, these degradation processes involve dechlorination to less-chlorinated (and possibly more toxic) congeners	

The degradation of PCDDs, PCDFs, and dioxin-like compounds in the environment depends largely on their chemical properties and the environmental media and conditions in consideration. For instance, photodegradation may determine the persistence of the aforesaid compounds in soil by processes predominantly occurring in the topsoil layer. Factors such as the degree of chlorination and the position of the chlorine atoms in the molecule may strongly affect the rate of decomposition (the persistence increases as the degree of chlorination increases). In addition, processes involving transport by wind or water (e.g. runoff) can influence the persistence of the chemicals in soil where they would be otherwise immobile. Reported half-lives in soil vary considerably. For instance, a half-life for 2,3,7,8-T_4CDD in soil of one up to 10 years has been reported, although a range of 10–12 years is viewed as most probable (UNEP, 2003).

6 AQUATIC ENVIRONMENTAL CONCENTRATIONS

6.1 ESTIMATED CONCENTRATIONS

Compartment	Predicted environmental concentration (PEC)	Master reference
Freshwater	No data available	-
Marine waters (coastal and/or transitional)		
Sediment		
Biota (freshwater)		
Biota (marine)		
Biota (marine predators)		

6.2 MEASURED CONCENTRATIONS

There are a number of TEF systems, which are not directly comparable. Some of the data reported in the tables thereafter are expressed according to the International System (1989), the Nordic System (1988) used in the Scandinavian countries or the WHO system, more recent (1998 and 2005) and already cited in the present document. The more recent system from WHO is not very different from the International System, except for the assessment of pentachloro- and octachloro-congeners, and the inclusion of TEFs for dioxin-like coplanar PCBs (OSPAR Commission, 2007).

Sum PCDDs + PCDFs + DL-PCBs

Compartment		**Measured environmental concentration (MEC)**	**Master reference and [TEF system]**
Freshwater ($\mu g_{WHO98\text{-}TEQ}.l^{-1}$)	EU	1.6 10^{-6}	James et al., 2009 [WHO_{98} TEF]
Marine waters ($\mu g_{WHO98\text{-}TEQ}.l^{-1}$)			
Sediment ($\mu g_{WHO98\text{-}TEQ}.kg^{-1}_{dw}$)		7.6 10^{-4} (fraction 2mm) 4.5 10^{-4} (fraction 20µm) 4.8 10^{-3} (fraction 63µm)	
Biota ($\mu g_{WHO98\text{-}TEQ}.kg^{-1}_{ww}$)		2.2 10^{-2} (fish)	
		1.8 10^{-3} (invertebrates)	

Sum PCDDs + PCDFs

Compartment		**Measured environmental concentration (MEC)**	**Master reference and [TEF system]**
Freshwater ($\mu g_{TEQ}.l^{-1}$)	*Eman River (Sweden)*	7.7 10^{-8} (particulate + dissolved fractions)	Rappe *et al.*, 1989 [International TEF]
	Elbe River (Germany)	4.0 – 17 10^{-9} (dissolved fractions)	Gotz *et al.*, 1994 [International TEF]
	Venice lagoon area (Italy)	5.3 – 8.2 10^{-5} (particulate + dissolved fractions)	Dalla Valle *et al.*, 2003
Marine waters (coastal and/or transitional) ($\mu g_{TEQ}.l^{-1}$)	*Grenlandsfjords (Norway)*	0.13 – 2.9 10^{-6} (particulate + dissolved fractions)	Ishaq *et al.*, 2009 [WHO_{98} TEF]
	Baltic Sea	2.3 – 2.4 10^{-9} (dissolved fractions)	Cornelissen *et al.*, 2008 [WHO TEF]
		0.4 – 3.6 10^{-9} (dissolved fractions)	Broman *et al.*, 1991 [Nordic TEF]
	Thau lagoon (France)	4.3 – 8.7 10^{-9} (particulate matter)	Castro-Jiménez *et al.*, 2008 [WHO_{98} TEF]
		5.3 10^{-8} (particulate + dissolved fractions)	Castro-Jiménez *et al.*, 2008 [WHO_{98} TEQ]
	Venice lagoon (Italy)	3 – 31 10^{-6} (particulate + dissolved fractions)	Dalla Valle *et al.*, 2003
		≈2 – 7.9 10^{-7} (particulate matter)	Cescon *et al.*, 2003 [WHO_{98} TEF]

Sum PCDDs + PCDFs

Compartment			Measured environmental concentration (MEC)	Master reference and [TEF system]
WWTP effluent ($\mu g_{TEQ}.l^{-1}$)			$2.6 – 38\ 10^{-7}$ (dissolved fractions)	Rappe *et al.*, 1989 [International TEF]
			$1.37\ 10^{-6}$ (particulate + dissolved fractions, ingoing water)	
			$9.8\ 10^{-7}$ (particulate + dissolved fractions, outgoing water)	
Particulate matter ($\mu g_{TEQ}.kg^{-1}_{dw}$)	*Elbe River (Germany)*		$7.3 – 4.1\ 10^{-2}$	Gotz *et al.*, 1994 [International TEF]
	Marine waters		$0.8 – 3.3\ 10^{-9}$	Broman *et al.*, 1991 [Nordic TEF]
	WWTP effluent		$1 – 2\ 10^{-6}$	Mahle *et al.*, 1989 [International TEF]
Sediment ($\mu g_{TEQ}.kg^{-1}_{dw}$)	*Grenlandsfjords (Norway)*		0.31 – 10	Ishaq *et al.*, 2009 [WHO_{98} TEF]
	Thau lagoon (France)		$2.9 – 13.8\ 10^{-3}$	Castro-Jiménez *et al.*, 2008 [WHO_{98} TEF]
	Venice lagoon (Italy)		$0.1 – 17.4\ 10^{-3}$	Miniero *et al.*, 2007 [International TEF]
			$2.2 – 6.2\ 10^{-3}$	Dalla Valle *et al.*, 2003 [International TEF]
	Almeria and Tarragona (Spain)		$0.1 – 48\ 10^{-3}$	Eljarrat *et al.*, 2005 [WHO_{98} TEF]
	Catalonia (Spain)		$0.42 – 8\ 10^{-3}$	Eljarrat *et al.*, 2001 [International TEF]
Biota ($\mu g_{TEQ}.kg^{-1}_{ww}$)	*Norway*	cod	$8.5\ 10^{-4}$	Knutzen *et al.*, 2003 [WHO_{98} TEF]
		flounder	$28\ 10^{-3}$	
	Norway (unpolluted fjord)	cod	$7\ 10^{-5}$	OSPAR Commission, 2007 [Nordic TEF]
		crab	$5.5\ 10^{-3}$	
	Norway (polluted fjord)	mussel	$5.4\ 10^{-3}$	
		crab	$4.4\ 10^{-2}$	
	Baltic Sea (as WHO_{98}-TEQ)	cod	$1.6\ 10^{-3}$	Piskorska-Pliszczynska *et al.*, 2004 [WHO_{98} TEF]
		salmon	$3.2\ 10^{-3}$	
		burbot	$1.3\ 10^{-4}$	Isosaari *et al.*, 2006 [WHO_{98} TEF]
		salmon	$1.7\ 10^{-2}$	
		herring	$3.4\ 10^{-3}$ (sum PCDD/Fs+DL-PCBs)	Szlinder-Richert *et al.*, 2009 [WHO_{98} TEF]
		salmon	$15.2\ 10^{-3}$ (sum PCDDs+PCDFs+DL-PCBs)	

Sum PCDDs + PCDFs

Compartment			Measured environmental concentration (MEC)	Master reference and [TEF system]
	Mediterranean area (as WHO_{98}-TEQ)	clam	$7\ 10^{-5}$	Bayarri *et al.*, 2001 [International TEF]
		mackarel	$1.1\ 10^{-3}$	
		trout	$1.8\ 10^{-4}$	Taioli *et al.*, 2005 [WHO_{98} TEF]
		eel	$1.1\ 10^{-3}$	
		wild fish	$1.2\ 10^{-4}$ (sum of PCBs 77 + 81 + 126 + 169)	Papadopoulos *et al.*, 2004 [WHO_{98} TEF]
		aquaculture fish	$4.7\ 10^{-4}$ (sum of PCBs 77 + 81 + 126 + 169)	
		barbel	$0.3 – 7.1\ 10^{-3}$ (sum PCDDs+PCDFs+DL-PCBs)	Eljarrat *et al.*, 2008 [WHO_{98} TEF]
		clam	$3\ 10^{-5}$	Miniero *et al.*, 2005 [WHO_{98} TEF]
		mussel	$6.8\ 10^{-4}$	
Biota (marine predators) ($\mu g_{WHO98\text{-}TEQ}.kg^{-1}{}_{ww}$)		swordfish	$6\ 10^{-5}$	Bocio *et al.*, 2007 [WHO_{98} TEF]
		tuna	$1.9\ 10^{-4}$	

DL-PCBs

Compartment		Measured environmental concentration (MEC)	Master reference
Freshwater ($\mu g.l^{-1}$)		No data available	
Marine waters (coastal and/or transitional) ($\mu g.l^{-1}$)	*Baltic Sea*	PCB 118 = $1 – 1.2\ 10^{-7}$ (dissolved)	Cornelissen *et al.*, 2008 [not reported]
	Thau lagoon (France)	PCB 118 = $1.4 – 2.2\ 10^{-5}$ (dissolved)	Castro-Jiménez *et al.*, 2008 [not reported]
Sediment ($\mu g.kg^{-1}{}_{dw}$)	*Thau lagoon (France)*	PCB 118 = 0.3 – 5.1	
	Almeria and Tarragona (Spain)	Sum DL-PCBs = $0.2 – 63\ 10^{-3}$ (as WHO-TEQ)	Eljarrat *et al.*, 2005 [WHO TEF]

7 EFFECTS AND QUALITY STANDARDS

7.1 ACUTE AND CHRONIC AQUATIC ECOTOXICITY

7.1.1 Organisms living in the water column

ACUTE EFFECTS			**Master reference**
Algae & aquatic plants (mg.l^{-1})	**Freshwater**	No data available	
	Marine	No data available	
Invertebrates (mg.l^{-1})	**Freshwater**	Crustacean, unknown species / unknown duration / most probably mixtures of PCB 118 and non-DL-PCBs $EC_{50\ -\ crustaceans}$ = 2 10^{-3}	FHI, as cited in FHI, 1999
	Marine	No data available	
	Sediment	No data available	
Fish (mg.l^{-1})	**Freshwater**	***Oncorhynchus mykiss* / 56 days / 2,3,7,8-T_4CDD** **EC_{50} = 4.6 10^{-8}**	**Ritter *et al.*, 1995**
	Marine	No data available	
	Sediment	No data available	
Other taxonomic groups		No data available	

CHRONIC EFFECTS			**Master reference**
Algae & aquatic plants (mg.l^{-1})	**Freshwater**	*Oedogonium cardiacum* / 33d / 2,3,7,8-T_4CDD NOEC = 3.1 10^{-9}	Yockim *et al.*, 1978
	Marine	No data available	
Invertebrates (mg.l^{-1})	**Freshwater**	*Daphnia magna* / 32d / 2,3,7,8-T_4CDD NOEC = 3.1 10^{-9}	Yockim *et al.*, 1978
	Marine	No data available	
Fish (mg.l^{-1})	**Freshwater**	***Oncorhynchus mykiss* / 28d / 2,3,7,8-T_4CDD** **NOEC = 1.1 10^{-9}**	**Mehrle *et al.*, 1998**
		Oncorhynchus mykiss / 56d / 2,3,7,8-T_4CDD $NOEC_{growth\ and\ mortality}$ = 3.8 10^{-8}	Ritter *et al.*, 1995
	Marine	No data available	
	Sediment	No data available	
Other taxonomic groups		No data available	

Acute exposure

Based on information on the knowledge of possible sources of dioxins and dioxin-like compounds (e.g. mainly unintentionally formed and released origins) long term or continuous releases into the aquatic environment are more likely than episodic releases of high concentrations, except in accidents cases. Chronic exposure of aquatic organisms is therefore expected rather than acute exposure and the need for a quality standards corresponding to short-term exposure may be questioned and is not felt appropriate at this stage based on the available dataset.

Chronic exposure

Valid toxicity data available for organisms living in the water column have been reported in the tables above for acute and chronic exposures for information. However, because of their hydrophobic nature, the majority of dioxins and dioxin-like compounds released into aquatic systems ultimately become associated with particulate matter and/or bioaccumulate in aquatic organisms. Given these considerations, uncertainties and difficulties of setting standards for the pelagic community based on waterborne exposure are substantial and it was recommended by the Scientific Committee on Health and Environmental Risks (SCHER, 2011) that biomarkers and other biological monitoring tools should be recommended rather than single chemical analysis in the case of dioxins assessment for water and sediment matrices (cf. section 7.1.3).

7.1.2 Sediment-dwelling organisms

Although sediment is a relevant matrix for Dioxins and DL-PCBs, there are no acute or chronic tests on exposure of sediment-dwelling to dioxins or DL-compounds (single or in mixtures) through true spiked sediment/water systems which allow determination of true $L(C)_{50}$, NOEC or EC_{10} values. There are however some sediment quality guidelines/criteria derived by NOAA (Buchman, 2008) or the Canadian Council of Ministers of the Environment (2001) and which are considered as screening/interim values. These are reported in the table below.

Freshwater sediment value – Upper Effect Treshold (UET) = 8.8 $ng_{TE}.kg^{-1}_{dw}$ (lowest reliable value among AET tests, on 1% total organic carbon basis and based on *Hyalella Azteca* exposed to 2,3,7,8-T_4CDD)	Buchman, 2008
Marine sediment value – Apparent Effect Treshold (AET) = 3.6 $ng_{TE}.kg^{-1}_{dw}$, (lowest reliable value among AET tests, based on *Neanthes* exposed to 2,3,7,8-T_4CDD)	
Interim Sediment Quality Guideline (ISQG) = 0.85 $ng_{TE}.kg^{-1}_{dw}$, based on benthic Invertebrates exposure exposed	Buchman, 2008 Canadian Council of Ministers of the Environment, 2001

However, these values are qualified as “screening tools” or "interim guidelines" by their authors and can therefore hardly be taken into account to derive legally-binding quality standards such as EQS without a “*clear analysis of the data and methodologies applied by the other bodies, and without a proper assessment of their applicability regarding WFD objectives*” (SCHER, 2011).

7.1.3 Relevance of biomarkers and other biological monitoring tools as alternative to single chemical analysis for deriving water and sediment quality standards

Data reported here above for direct ecotoxicity of water as well as toxicity to sediment-dwelling organisms are reported for illustration of the available dataset for these protection objective but can reasonably not be used to derive any $QS_{water, eco}$ nor $QS_{sediment}$ given the uncertainties, difficulties and low relevance of setting EQS for the pelagic community based on waterborne exposure.

However, the possibility for using effect-based monitoring tools for monitoring purposes should be noted here as alternatives to classical EQS compliance check. In this context, the use of *in vitro* cell-based bioassays to assess dioxin-like activities in organic extracts of sediments or water appears as a useful alternative methodology. This tool allows detecting substances characterized by a common mechanism of action: interaction with the Ah receptor. The direct measurement of the biological responses associated to the Ah receptor in water and sediment samples may provide a proper and rapid quantification of the overall potency of dioxin and dioxin-like compounds in the sample, that can be easily expressed as 2,3,7,8-TCDD TEQ. Hence, the results provide a global evaluation of environmental sample contamination by dioxin-like substances (Brack *et al.*, 2005; Brack *et al.*, 2007; Louiz *et al.*, 2008; Kinani *et al.*, 2010) conversely to chemical analysis. The joint effects can be considered as additive, and therefore quantified on the basis of their relative potencies using the TEF approach.

Several methods based on a similar mechanism are commercially available (e.g. DR-CALUX, PLHC 1-EROD) and are used to assess dioxin-like contamination in environmental samples. Some examples are given in the table thereafter for the sediments.

Bioassays	**Sites location**	**Relative potencies expressed as ng TCDD-TEQ.g$^{-1}_{dw}$**	**Master reference**
PLHC 1-EROD	*North of France area (France)*	0.67 – 48.38	Kinani *et al.*, 2010
	Bay of Kvarner area (Croatia)	6.0 – 132.1	Traven *et al.*, 1998
DR-CALUX	*UK estuaries (United Kingdom)*	1.1 – 154	Hurst *et al.*, 2004
RTL-W1	*Forellemback creek (Germany)*	0.01 – 70.06	Brack *et al.*, 2005

These methods can also be used to assess dioxin-like contamination in biota (Thomas *et al.*, 2006).

In biota, biomarker responses and particularly responses linked to cytochrome P450 1A (CYP1A; i.e. gene expression, protein or catalytic activity inductions) could be used to derive a QS for a specific mechanism of action. Indeed, CYP1A induction is due to activation of Ah receptor by dioxin-like substances and is described as a relevant biomarker of dioxin-like exposure (for review, see Whyte *et al.*, 2000). However, many chemicals such as heavy metals or estrogens are known to inhibit CYP1A activity and many biotic and biological factors are also documented as confounding factors. Hence, in a context of multi-contamination, application of a single biomarker such as CYP1A cannot be a relevant methodology to define accurately a QS which is by definition substance-specific. Gene expression/array systems also provide a sensitive measure of early changes but enzyme induction and results from gene expression arrays would only be considered as possible early indicators for potential adverse effects if they are demonstrated to be obligate precursors to an adverse effect and appropriate consideration is given to mechanisms of repair and homeostasis (EFSA, 2004).

As a conclusion, the direct measurement of the biological responses associated to the Ah receptor in water and sediment samples may provide a proper and rapid quantification of the overall potency of dioxin and dioxin-like compounds in the sample. Therefore, screening of ecotoxicological effects may rather be considered using biological responses together with classical EQS compliance check.

7.2 SECONDARY POISONING

The effects of dioxins and dioxin-like compounds have been extensively reviewed recently by some authors, (e.g. Bursian *et al.*, 2011) who conclude that these are numerous in humans, mammals and some avian species. These effects notably include female reproductive effects (e.g. reduction in women fecundity, increased preimplantation embryomortality in mammals, presence of endometrial glands and stroma outside the women uterus, reduced ovarian weight, declined fertility and persistent vaginal estrous in mammals, placental hypoxia and delayed maturation of the mammary gland which could increase the incidence of breast cancer due to increase susceptibility of the gland to carcinogens in rats) and male reproductive effects (e.g. decreased testis and accessory organ weight, abnormalities testicular morphology, decreased spermatogenesis and reduce fertility) when given to adult animals in doses sufficient to reduce feed intake and/or body weight. On the overall, these effects occurring at the lowest doses in animal studies result from *in utero* exposures.

Further to its Scientific Colloquium in 2004, EFSA concluded that "the neurological development may also be affected and neurobehavioural endpoints may be a future area for investigation. Also, they added that "non-developmental effects in laboratory animals include immune effects, endometriosis and cancer" (EFSA, 2004).

It is to be noted that the majority of the available toxicological studies on mammals involve acute bolus dosing, and therefore require extrapolation for long term dietary exposure.

Secondary poisoning of top predators		**Master reference**
Mammalian oral toxicity	Females Wistar rat / Oral / Reproductive study Exposure to 2,3,7,8 TCDD Initial subcutaneous loading doses (2 weeks prior to mating): 25, 60, or 300 $ng.kg^{-1}_{bw}$ Weekly maintenance doses: 5, 12, or 60 $ng.kg^{-1}_{bw}$ Observation of male offsprings on PND70 and PND170 Effects: Decreased sperm production and altered sexual behaviour in male offspring Uncertainties: no clear dose-effect response (E.C., 2001; Bell *et al.*, 2010) LOAEL = 2.5 10^{-5} $mg.kg^{-1}_{bw}.d^{-1}$ corresponding to a maternal body burden of 4 10^{-5} $mg.kg^{-1}_{bw}.d^{-1}$ at steady state following subchronic daily administration (E.C., 2001). NOAEL = 1.3 10^{-5} $mg.kg^{-1}_{bw}.d^{-1}$ ($CF_{LOAEL->NOAEL}$=3) NOEC = 1.1 10^{-4} $mg.kg^{-1}_{feed\ ww}$ (CF=8.33, E.C., 2010, to be considered with caution since administration route is subcutaneous)	Faqi *et al.*, 1998
	Females Holtzman rat / Oral / Reproductive study Exposure to 2,3,7,8 TCDD Single doses – GD15: 0, 12.5, 50, 200, 800 $ng.kg^{-1}_{bw}$ Observation of male offsprings on PND49 or PND120 Effects: - no changes seen on testicular or epididymal weights nor in daily sperm production or sperm reserve at any of the doses used - no apparent dose-dependent changes in levels of either serum testosterone or luteinizing hormone	Ohsako *et al.*, 2001

Secondary poisoning of top predators		**Master reference**
	- significant reduction of the urogenital complex weight, including the ventral prostate, at 200 and 800 ng.kg$^{-1}_{bw}$ doses on PND 120 - significant decrease of anogenital distance on PND 120 at 50 ng.kg$^{-1}_{bw}$ doses or higher Uncertainties: effects observed on prostate at PND49 not observed at PND120 (E.C., 2001) NOAEL = 1.25 10^{-5} mg.kg$^{-1}_{bw}$.d^{-1} corresponding to a maternal body burden of 3.1 10^{-5} mg.kg$^{-1}_{bw}$.d^{-1}, corresponding to a maternal body burden of 2 10^{-5} mg.kg$^{-1}_{bw}$.d^{-1} at steady state following subchronic daily administration (E.C., 2001). NOEC = 1.7 10^{-4} mg.kg$^{-1}_{feed\ ww}$ (CF=8.33, E.C., 2010)	
	Other studies available but which, according to E.C. (2001): - exert less stringent effects on rats (Gray et al., 1997;Mably et al., 1992) or body burdens deemed too low to affect fertility of the male offsprings rats (Murray et al., 1979) - did not address the sensitive endpoints included the above cited studies, - did address other effects than effects on rats reproductive system (e.g. immune system, Nohara *et al.*, 2000) but at higher dose. - did address effects on monkey but studies included too much uncertainties (e.g. Rier *et al.*, 1993) For all these reasons, the above cited studies were not considered as pivotal in the derivation of a threshold level for secondary poisoning.	
Avian oral toxicity	*Gallus domesticus* / Oral / 21 d NOAEL = 1 10^{-4} mg$_{TE}$.kg$^{-1}_{bw}$.d^{-1} NOEC = 8 10^{-4} mg.kg$^{-1}_{biota\ ww}$ (CF=8)	Schwetz *et al.*, 1973

In its opinion on the risk assessment of dioxins and dioxin-like PCBs in food, the scientific committee on food (E.C., 2001) recognized that the Wistar rats as used in the study by Faqi et al. (1998) might be the most sensitive rat strain and concluded that the value issued from this study should be considered as a tolerable intake for 2,3,7,8-TCDD. For comparison purpose, a conversion factor of 8.33 for converting NOAELs (dose) from mammalian toxicity studies into NOECs (concentration) according to TGD-EQS (E.C., 2011). This factor however may not be appropriate for this study where animals have been administered subcutaneously. For the back calculation of $QS_{biota,\ sec.pois.}$ into water, the BCF values of 41 540 is used as well as equal default BMF_1 and BMF_2 values of 10 (cf. section 5.1).

Tentative $QS_{biota,\ sec.pois.}$	**Relevant study for derivation of QS**	**Assessment factor**	**Tentative QS**
Biota	NOEC = 1.1 10^{-4} mg.kg$^{-1}_{feed\ ww}$ (using a provisional conversion factor of 8.33)	90[(1)]	For comparison purpose only: 1.2 10^{-3} µg.kg$^{-1}_{biota\ ww}$ corresponding to 2.8 10^{-9} µg.l^{-1} (freshwater) 2.8 10^{-10} µg.l^{-1} (marine waters)

(1) proposal made for the purpose of this dossier, according to REACH guidance on information requirements and chemical safety assessment (ECHA, 2008) and the draft technical guidance on deriving Environmental Quality Standards (E.C., 2010) estimating the study considered to be a "reproductive study".

Quality of the data set and uncertainties associated

According to a number of authors (e.g. Bell *et al.*, 2010; Foster *et al.*, 2010), the decreased sperm count observed in some publications as the most sensitive effect failed to be reproduced and that the although animal studies available provide clear evidence of an adverse effect of in utero dioxins exposure on epididymal sperm count, no clear link could be made with possible adverse effects on spermatogenesis. Foster *et al.* (2010) conclude that the mechanisms underlying decreased epididymal sperm count are not unknown but postulate that epididymal structure and/or function as well as developmental abnormalities of the male reproductive tract might be the key target of the adverse effects of dioxins.

Bell and its collaborator (2010), add that maternal doses of <1 µg TCDD/kg that produced adverse effects reported in offspring are "frequently within the range of historical variation seen in other laboratories" and that "the potency of TCDD to induce these effects appears to be much greater after chronic dosing, compared with acute dosing". They note as this regards that "maternal pharmacokinetics of TCDD vary considerably between acute and chronic dosing, and that these two differing dosing regimens have been shown to impact upon the potency of TCDD at inducing adverse effects" (Bell *et al.*, 2010).

Given these considerations and associated uncertainties, the proposed $QS_{biota,\ sec.pois.}$ has to be considered with caution and the $QS_{biota,\ human\ health}$ should be preferred.

7.3 HUMAN HEALTH

Considerations reported above in section 7.2 as regards effects on mammals and uncertainties related to these are to be considered for this section dealing with protection of human health from consumption of fishery products. Besides this, further to its Scientific Colloquium in 2004, EFSA considered that the currently available human data do not provide sufficient basis for effects assessment, and it is therefore necessary to use animal data to derive human tolerable daily intake values. Also, it was concluded that cancer was "recognised as a relevant endpoint in the epidemiological studies" but it was "still questioned whether it is a threshold or non-threshold effect" (EFSA, 2004).

Human health via consumption of fishery products		**Master reference**
Mammalian oral toxicity	Females Wistar rat / Oral / Reproductive study Exposure to 2,3,7,8 TCDD Initial subcutaneous loading doses (2 weeks prior to mating): 25, 60, or 300 $ng.kg^{-1}_{bw}$ Weekly maintenance doses: 5, 12, or 60 $ng.kg^{-1}_{bw}$ Observation of male offsprings on PND70 and PND170 Effects: Decreased sperm production and altered sexual behaviour in male offspring Uncertainties: no clear dose-effect response (E.C., 2001; Bell *et al.*, 2010) LOAEL = 2.5 10^{-5} $mg.kg^{-1}_{bw}.d^{-1}$ corresponding to a maternal body burden of 4 10^{-5} $mg.kg^{-1}_{bw}.d^{-1}$ at steady state following subchronic daily administration (E.C., 2001).	Faqi *et al.*, 1998
	Females Sprague-Dawley rat / Oral / Reproductive study Exposure to 2,3,7,8 TCDD Single doses – GD15: 0, 12.5, 50, 200, 800 $ng.kg^{-1}_{bw}$ Observation of male offsprings on PND49 or PND120 Effects: - no changes seen on testicular or epididymal weights nor in daily sperm production or sperm reserve at any of the doses used - no apparent dose-dependent changes in levels of either serum testosterone or luteinizing hormone - significant reduction of the urogenital complex weight, including the ventral prostate, at 200 and 800 $ng.kg^{-1}_{bw}$ doses on PND 120 - significant decrease of anogenital distance on PND 120 at 50 $ng.kg^{-1}_{bw}$ doses or higher Uncertainties: effects observed on prostate at PND49 not observed at PND120 (E.C., 2001) NOAEL = 1.25 10^{-5} $mg.kg^{-1}_{bw}.d^{-1}$ corresponding to a maternal body burden of 3.1 10^{-5} $mg.kg^{-1}_{bw}.d^{-1}$, corresponding to a maternal body burden of 2 10^{-5} $mg.kg^{-1}_{bw}.d^{-1}$ at steady state following subchronic daily administration (E.C., 2001).	Ohsako *et al.*, 2001

Human health via consumption of fishery products		**Master reference**
	Other studies available but which, according to E.C. (2001): - exert less stringent effects on rats (Gray et al., 1997;Mably et al., 1992) or body burdens deemed too low to affect fertility of the male offsprings rats (Murray et al., 1979) - did not address the sensitive endpoints included the above cited studies, - did address other effects than effects on rats reproductive system (e.g. immune system, Nohara et al., 2000) but at higher dose. - did address effects on monkey but studies included too much uncertainties (e.g. Rier et al., 1993) For all these reasons, the above cited studies were not considered as pivotal in the derivation of a threshold level for secondary poisoning.	
	2,3,7,8 T_4CDD Minimum Risk Levels (MRL): acute, oral, mice = 2 10^{-7} $mg.kg^{-1}.d^{-1}$ intermediate, oral, guinea pig = 2 10^{-8} $mg.kg^{-1}.d^{-1}$ chronic, oral, monkey = 1 10^{-9} $mg.kg^{-1}.d^{-1}$	ATSDR, 1998
CMR	There is sufficient evidence for carcinogenicity of 2,3,7,8,-T_4CDD, 2,3,4,7,8-P_5CDF and 3,3',4,4',5-P_5CB [CB-126] in experimental animals. The substance is classified as a human carcinogen (group 1). The other congeners are deemed not classifiable as to their carcinogenicity in humans (group 3).	IARC, 2009

In their evaluation of dioxins and dioxin-like compounds, WHO-JECFA (JECFA, 2002) and SCF (E.C., 2001) have taken into account the above cited studies from Faqi et al. (1998) and Ohsako et al. (2001) and their NOAEL or LOAEL (see table above) to derive, *via* application of a body burden approach, Estimated Human Daily Intake (EHDI) values. Application of assessment factors to these EHDI values were then used to derive tolerable intake for humans (see table below).

For PCDDs, PCDFs, and DL-PCBs, Regulation (EC) No. 1881/2006 setting maximum levels for certain contaminants in foodstuffs, reports thresholds levels in Section 5 of its Annex as follows.

- Muscle meat of fish and fishery products and products thereof with the exception of eel, applicable also to crustaceans, excluding the brown meat of crab and excluding head and thorax meat of lobster and similar large crustaceans (*Nephropidae* and *Palinuridae*):
    - o 4 10^{-6} $mg_{WHO98\text{-}TE}.kg^{-1}_{ww}$ (Σ PCDDs+PCDFs)
    - o 8 10^{-6} $mg_{WHO98\text{-}TE}.kg^{-1}_{ww}$ (Σ PCDDs+PCDFs+DL-PCBs)

Thresholds above concern fish but also shellfishes as mussels or clams. Fish and shellfishes are known indicators for the evaluation of dioxins bioaccumulation and are widely used in monitoring programmes of coastal and transitional waters (SCHER, 2011).

A level is recommended is also recommended in muscle meat of eel and products thereof,

- Muscle meat of eel (*Anguilla anguilla*) and products thereof:
    - o 4 10^{-6} $mg_{WHO98\text{-}TE}.kg^{-1}_{ww}$ (Σ PCDDs+PCDFs)
    - o 1.2 10^{-5} $mg_{WHO98\text{-}TE}.kg^{-1}_{ww}$ (Σ PCDDs+PCDFs+DL-PCBs)

However, eels are protected species so cannot be used for routine monitoring or sampling, therefore these data should not be retained in estimation of an EQS.

There are no other available valuable tolerable human intake values or maximum levels in foodstuffs values up to date than the one mentioned here above. This is confirmed by the short information document for decision makers published recently by the World Health Organization (WHO, 2010) which refers to the Provisional Tolerable Monthly Intake proposed by the WHO-JECFA in 2002 extracted from the above cited evaluation (JECFA, 2002). Therefore, these available data have been used to tentatively establish thereafter a $QS_{biota,\ hh}$.

For the back calculation of $QS_{biota,\ hh}$ into water, the BCF values of 41 540 is used as well as equal default BMF_1 and BMF_2 values of 10 (cf. section 5.1).

Tentative $QS_{biota\ hh}$	**Relevant data for derivation of QS**	**AF**	**Threshold Level** ($mg.kg^{-1}_{bw}.d^{-1}$)	**Tentative $QS_{biota,\ hh}$**
Human health	NOAEL = 1.25 10^{-5} $mg.kg^{-1}_{bw}.d^{-1}$ corresponding to (E.C., 2001): - maternal body burden of 2 10^{-5} $mg.kg^{-1}_{bw}.d^{-1}$ at steady state following subchronic daily administration - Estimated Human Daily Intake (EHDI) = 1 10^{-8} $mg.kg^{-1}_{bw}$	3.2	TDI = 3 10^{-9} **(*)**	**1.8 10^{-4} $\mu g.kg^{-1}_{biota\ ww}$ (**)** corresponding to 4.4 10^{-10} $\mu g.l^{-1}$ (freshwater) 4.4 10^{-11} $\mu g.l^{-1}$ (marine waters)
	LOAEL = 2.5 10^{-5} $mg.kg^{-1}_{bw}.d^{-1}$ corresponding to (E.C., 2001): - a maternal body burden of 4 10^{-5} $mg.kg^{-1}_{bw}.d^{-1}$ at steady state following subchronic daily administration - Estimated Human Daily Intake (EHDI) = 2 10^{-8} $mg.kg^{-1}_{bw}$	9.6	TDI = 2 10^{-9} **(*)**	**1.2 10^{-4} $\mu g.kg^{-1}_{biota\ ww}$ (*)** corresponding to 2.9 10^{-10} $\mu g.l^{-1}$ (freshwater) 2.9 10^{-11} $\mu g.l^{-1}$ (marine waters)
	Maximum levels given for foodstuffs content of the sum of PCDDs and PCDFs: **4 10^{-6} $mg_{WHO98\text{-}TEQ}.kg^{-1}_{ww}$ for fish including eel and for crustaceans** excluding brown meat of crab and excluding head and thorax meat of lobster and similar large crustaceans (*Nephropidae* and *Palinuridae*) corresponding to 9.6 10^{-9} $\mu g_{WHO98\text{-}TEQ}.l^{-11}$ (freshwater) 9.6 10^{-10} $\mu g_{WHO98\text{-}TEQ}.l^{-1}$ (marine waters)			
	Maximum levels given for foodstuffs content of the sum of DL-compounds (PCDDs, PCDFs and DL-PCBs): - **8 10^{-6} $mg_{WHO98\text{-}TE}.kg^{-1}_{ww}$ for fish excluding eel and for crustaceans** excluding brown meat of crab and excluding head and thorax meat of lobster and similar large crustaceans (*Nephropidae* and *Palinuridae*) corresponding to 1.9 10^{-8} $\mu g.l^{-1}$ (freshwater) 1.9 10^{-9} $\mu g_{WHO98\text{-}TEQ}.l^{-1}$ (marine waters)			

(*) *Assessment factors of 3.2 and 9.6 chosen by WHO-JECFA (JECFA, 2002) and SCF (E.C., 2001) to take account of a number of uncertainties, e.g. dose-response relationship (use of LOAEL instead of NOAEL) when deemed necessary, as well as intraspecies variations and potential differences in toxicodynamics between experimental animals and humans.*

(**) *an additional assessment factor of 10 to take account of possible carcinogenic effects was not applied in this calculation given that WHO-JECFA (JECFA, 2002) and SCF (E.C., 2001) judged that the critical effects considered to estimate TDI appeared to follow a dose response relationship at lower body burdens than those causing other effects, including cancer.*

Maximum level given for foodstuffs content of the sum of DL-compounds (PCDDs, PCDFs and DL-PCBs) in fish excluding eel and for crustaceans and shellfishes was chosen as the $QS_{biota, hh}$ because they represent the most relevant standard when compared with other proposal after back-calculation in water.

Quality of the data set and uncertainties associated

WHO-JECFA (JECFA, 2002) and SCF (E.C., 2001) concluded in 2001 and 2002 that "the weight of scientific evidence is sufficient to assume that there is likely a dose-response threshold for the critical effects of dioxin-like compounds, including cancer. The reproductive/developmental effects (sperm counts, accessory sex gland weights) were considered to be the critical effects; these appear to follow a dose response relationship at lower body burdens than those causing other effects including cancer. However there is uncertainty with respect to extrapolation from acute bolus to repeated dosing since internal doses in humans usually result from repeated exposure via diet and other sources. Furthermore, acute dosing may lead to particularly high internal doses at critical periods of development".(EFSA, 2004).

All the evaluations available up to date on dioxins and dioxin-like compounds (e.g. E.C., 2001; JECFA, 2002; EFSA, 2004) acknowledge a certain degree of uncertainty in deriving tolerable daily intakes for human. Hence, a number of gaps leading to these uncertainties have been listed such as (not exhaustive and not prioritised) the needs for:

- Longer duration studies with dietary exposures (e.g. a multigeneration study), including neurodevelopmental and endocrine effects (thyroid, gonadotropins, etc.), and information on relevant tissue doses for comparison with bolus dose studies (NOAEL for the critical effects rather than LOAELs).
- Mechanistic data in support of the critical effects of dioxins, including shape of the dose-response function at doses of interest, ranges in susceptibility and inter-species comparisons, as well as a need for accurate understanding how and when TCDD operates to cause adverse effects in F1 animals after low dose maternal exposure.
- Identification of additional chemicals contributing to the TEQ body burden.
- Information on which genes or gene combinations are responsible for toxic reactions, which would help to link early dose responses (enzyme induction, etc.) to outcome and facilitate low-dose extrapolation and cross-congener comparisons.
- Evaluation of response additivity of non-dioxin-like compounds not currently considered in TEFs.
- Evaluation of roles of naturally occurring AhR-ligands in overall responses to dioxins.
- Mode of action studies assisting dose-response modelling.
- Clarification of primary versus secondary effects related to initiation/promotion of cancer or induction of non-cancer effects, for example.
- Information on the possible relationship between hormonal mechanisms and critical effects of dioxins.

Finally, it should be noted that maximum levels in foodstuffs as indicated in EC Regulation 1881/2006 have been calculated using 1998 WHO TEF values (Van den Berg *et al.*, 1998), i.e. do not take into account recent knowledge taken on board during revision of these values by the WHO in 2005 (Van den Berg *et al.*, 2006). However, as TL_{hh} values from which these maximum levels are issued are not available, it is not possible to revise the 1881/2006 values according to the 2005 WHO TEF values.

Given all these considerations above cited and associated uncertainties, the proposed $QS_{biota, hh}$ has to be considered with caution.

Human health via consumption of drinking water		**Master reference**
Existing drinking water standard(s)	No available regulatory data	Directive 98/83/EC
	Calculation of a standard in water is not deemed relevant given DL-compounds lipophilic properties (see section 5)	

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