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Maximum Permissible Concentrations for polychlorinated biphenyls

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ABSTRACT

In the current report maximal permissible concentrations (MPCs) and negligible concentrations (NCs) are derived for polychlorinated biphenyls (PCBs). In this derivation probabilistic food web modeling was used. Toxicity data for aquatic organisms, mammals and birds were recalculated into equivalent toxic concentrations in the organic carbon of sediments or soils. In this way, all types of studies could be readily compared on the same concentration axis and could be integrated into one MPC.
As PCBs always occur in a mixture and the planar PCBs are supposed to act via the same toxicological mechanism and thus to be concentration-additive, a mixture-MPC was derived.

PREFACE

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CONTENTS

SAMENVATTING

In dit rapport zijn maximaal toelaatbare risiconiveaus (MTRs) en verwaarloosbare risiconiveaus (VRs) afgeleid voor individuele congeneren van poly gechloreerde bifenylen (PCBs). De MTRs zijn uitgedrukt in equivalente concentraties in het organisch koolstof en zijn afgeleid voor de non-ortho gesubstitueerde congeneren #77, #126 en #169, voor de mono-ortho gesubstitueerde congeneren #105, #118, #156 en #157, en de di-ortho gesubstitueerde congeneer #153. Omdat de non- en mono-ortho gesubstitueerde congeneren worden geacht via een zelfde toxicologisch werkingsmechanisme hun effecten te veroorzaken en zodoende concentratie-additief, is daarnaast voor deze congeneren een zogenaamd mengsel-MTR afgeleid.
Toxiciteitgegevens voor vogels, zoogdieren en waterorganismen zijn, na een normalisatie op het vetgehalte, omgerekend in toxische concentraties in het organisch koolstof van sediment of bodem. Hierbij is rekening gehouden met opstapeling van de stoffen in de voedselketen. Gegevens die verkregen zijn uit veldstudies aangaande het lot van PCBs in het milieu -denk aan biomagnificatie factoren en biota-naar-sediment accumulatie factoren- zijn gebruikt in de berekeningen. Het gebruik van parameters waarvan de waarde moeilijk experimenteel is vast te stellen omdat het om sterk hydrofobe verbindingen gaat, zoals de bioconcentratie factor of sediment/water partitie coëfficiënt, is waar mogelijk vermeden. Met probabilistische technieken is de spreiding in de data die ten grondslag liggen aan de gebruikte parameters geïncorporeerd. Op deze wijze zijn waarschijnlijkheidsverdelingen van concentraties in sediment of bodem verkregen die ongewenste effecten veroorzaken. De bijdrage van de gebruikte parameters aan de spreiding in de resulterende waarschijnlijkheidsverdelingen is bepaald. Wanneer het statistisch geoorloofd bleek, zijn de waarschijnlijkheidsverdelingen voor de verschillende soorten gecombineerd per congeneer; de MTR is gebaseerd op het vijfde percentiel van deze gecombineerde verdelingen (zie Tabel I).
Om de mengsel-MTR af te leiden voor de non- en mono-ortho gesubstitueerde PCBs, is gebruik gemaakt van congeneer patronen zoals deze voorkomen in verscheidene sedimenten en invertebraten in Nederland. Opgemerkt dient te worden dat op een specifieke locatie het congeneer patroon kan afwijken van het patroon zoals dat gebruikt is voor de afleiding van de mengsel-MTR. Voor zo'n specifieke situatie kan een mengsel-MTR berekend worden op eenzelfde wijze als beschreven in hoofdstuk 6.3. De mengsel-MTR staat voor de toxiciteit van het hele mengsel van vlakke PCBs dat beschouwd is in dit rapport. Deze wordt uitgedrukt in een concentratie van PCB #118, omdat deze congeneer deel uitmaakt van de standaard monitoring set zoals gebruikt in Nederland. Wanneer de concentratie van PCB #118 in een veldmonster lager is dan 5 µg/kg o.c., wordt het ecosysteem geacht beschermd te zijn tegen de negatieve effecten van het hele mengsel van platte PCBs.
De MTRs zijn vergeleken met concentraties die een correlatief verband houden met ongewenste effecten in de veldsituatie in dieren aan de top van de voedselketen. Het blijkt dat de concentraties die in het veld ongewenste effecten veroorzaken, vergelijkbaar zijn met de concentraties die zouden resulteren als alle vlakke congeneren op MTR-niveau aanwezig zouden zijn.
De relatieve potenties van de verschillende congeneren voor toxicologische effecten zijn vergeleken met die voor biochemische en histopathologische effecten. Omdat

beide effect-typen veroorzaakt worden door hetzelfde werkingsmechanisme zouden de relatieve potenties vergelijkbaar moeten zijn. Met uitzondering van #105 blijkt dit inderdaad het geval te zijn. Hoewel er voor sommige congeneren relatief weinig toxiciteitsdata aanwezig zijn, zijn de data kennelijk wel een goede uitsnede van de toxiciteit. Biochemische en histopathologische effecten komen voor bij concentraties die 1,5 tot 3 log-eenheden lager liggen dan benodigd voor effecten op groei, reproductie of overleving.
De MTRs zoals afgeleid in dit rapport zijn vergeleken met MTRs die nu geldig zijn in Nederland. Dit kon voor congeneren #118 en #153, en de verschillen blijken kleiner te zijn dan een factor drie.

Tabel I. Maximaal Toelaatbaar Risiconiveau (MTR) en Verwaarloosbaar Risiconiveau (VR) voor individuele congeneren, en voor het mengsel van vlakke PCBs (alle congeneren met uitzondering van #153) uitgedrukt als concentratie van congeneer #118

PCB	MTR (µg/kg o.c.)	VR (µg/kg o.c.)
77	7.2	0.072
105	26	0.26
118	25	0.25
126	0.042	0.00042
153	151	1.51
156	55	0.55
157	32	0.32
169	0.83	0.0083
mengsel-MTR vlakke PCBs	5 (conc. van #118)	0.05 (conc. van #118)

SUMMARY

Maximum permissible concentrations (MPCs) and negligible concentrations (NCs) for individual congeners of polychlorinated biphenyls (PCBs) are derived. MPCs in organic carbon are derived for the non-ortho substituted congeners #77, #126 and #169, the mono-ortho substituted congeners #105, #118, #156 and #157 and the di-ortho substituted congener #153. A mixture-MPC is derived for the non- and mono-ortho substituted PCBs, which are thought to act via the same toxicological mechanism and thus to be concentration-additive.
After lipid-normalisation, toxicity data for birds, mammals and aquatic organisms are recalculated into equivalent toxic concentrations in organic carbon. Accumulation in the food chain is taken into account. Field-derived data on the environmental fate of PCBs, e.g. biomagnification factors and biota-to-sediment accumulation factors, are used in these calculations. The use of parameters that are difficult to measure for these hydrophobic compounds, such as the bioconcentration factor or the sediment/water partition coefficient, is avoided where possible. Probabilistic techniques are used to incorporate the spread in the data on environmental fate. In this way, probability distributions of sediment/soil concentrations associated with adverse effects are obtained. The contribution of the underlying parameters to the variance in the resulting probability distribution is determined. Distributions for the various species are combined per congener when statistically appropriate; MPCs are based on the 5th percentile of these combined distributions (see Table I).
Congener patterns as occurring in various sediments and invertebrates in the Netherlands are used for determining a mixture-MPC for non- and mono-ortho PCBs. It should be noted that at a specific location the congener pattern of planar PCBs may deviate from the congener pattern used in the derivation of the mixture MPC. A mixture-MPC for that specific situation can then be calculated in a similar way as described in section 6.3. The mixture-MPC addresses the toxicity of the mixture of planar congeners considered in this report. The mixture-MPC is expressed in a concentration of PCB #118, as this congener is part of the standard monitoring set used in the Netherlands. If the concentration of PCB #118 in a field sample is lower that 5 µg/kg o.c., the ecosystem is assumed to be protected for the whole mixture of planar PCBs.
The derived MPCs are compared to concentrations associated with adverse effects in the field in top predators such as fish-eating birds, otter and mink. Concentrations associated with adverse effects in field studies, are comparable to concentrations that would result if all planar congeners would be present on MPC-level.
The relative potencies of the different congeners based on toxicological effects (the data used in the derivation of the MPC) are compared with relative potencies based on biochemical and histopathological effect data. As both types of effects are exerted via the same mechanism of action these relative potencies should be similar. Except for congener #105, this turns out to be the case. Thus although for some congeners little toxicity data are available, the dataset is apparently a good predictor of toxicity. Biochemical and histopathological effects occur at concentrations1.5 to 3 log-units lower than needed for effects on growth, reproduction or survival.

The MPCs derived in the present report are compared with the MPCs currently in use in the Netherlands. This could be done for congeners #118 and #153, differences are lower than three-fold.

Table I. Maximum Permissible Concentrations (MPCs) and Negligible Concentrations (NCs) for individual congener and for the mixture of planar PCBs expressed as concentration of #118

PCB	MPC (µg/kg o.c.)	NC (µg/kg o.c.)
77	7.2	0.072
105	26	0.26
118	25	0.25
126	0.042	0.00042
153	151	1.51
156	55	0.55
157	32	0.32
169	0.83	0.0083
mixture-MPC planar PCBs	5 (conc. of #118)	0.05 (conc. of #118)

ABBREVATIONS

A	Assimilation efficiency
AhR	Arylhydrocarbon Receptor
ANOVA	Analysis of Variance
Arnt	Ah-receptor nuclear translocator
BCF	Lipid normalised concentration factor between organism and water (C_o/C_w)
$BCF_{w.w.}$	Wet-weight based concentration factor between organism and water ($C_{o,w.w.}/C_w$)
BMF	Lipid normalised concentration ratio between predator and prey ($C_{o,predator}/C_{o,prey}$)
BSAF	Lipid and organic carbon normalised concentration ratio between an organism and sediment or soil ($C_o/C_{o.c.}$)
C_f	Concentration in the food, expressed on wet weight basis
C_o	Lipid based concentration in the organism
$C_{o,w.w.}$	Wet-weight based concentration in the organism
$C_{o,predator}$	Lipid based concentration in the predator
$C_{o,prey}$	Lipid based concentration in the prey
$C_{o.c.}$	Organic carbon normalised concentration in sediment or soil
C_w	Concentration in the water
CSR	Centre for Substances and Risk Assessment
CYP 1A1	Cytochrome P450 1A1
DRE	Dioxin Responsive Element
EBR	Concentration ratio between the egg and the parent on lipid weight basis
$EC50_{o.c.}$	Organic carbon based concentration in the sediment exerting effect in 50% of the organisms
$ED50_{egg,w.w.}$	Wet weight based dose in the egg exerting effect in 50% of the organisms
$ED50_{o,w.w.}$	Wet weight based dose in the organism exerting effect in 50% of the organisms
ECOTEF	TEF specific for group of species
EQS	Environmental Quality Standard
K	Elimination rate
K_{ow}	N-octanol/water partition coefficient
K_{oc}	Organic carbon normalised sediment/water or soil/water partition coefficient ($C_{o.c.}/C_w$)
LD50	Dose lethal to 50% of the organisms, expressed as C_o
$LD50_{o.c.}$	Dose lethal to 50% of the organisms, expressed as $C_{o.c.}$
l.f.	Lipid fraction of the organism, subscripts refer to respectively 'fish egg', 'mammal' and 'bird egg'
MPC	Maximum Permissible Concentration
NC	Negligible Concentration
NEPP	National Environmental Policy Plan
NOEC	No-observed-effect concentration
$NOEC_{o.c.}$	No-observed-effect concentration in organic carbon

OECD	Organisation for Economic Co-operation and Development
PAF	Potentially affected fraction of all species
PAH	Polycyclic Aromatic Hydrocarbon
PCB	Polychlorinated Biphenyl
PCDD	Polychlorinated dibenzo-*p*-dioxin
PCDF	Polychlorinated dibenzofuran
QSAR	Quantitative Structure Activity Relationship
R	Daily ration of food
RIVM	National Institute of Public Health and the Environment
t	Duration of the diet
TCDD	Tetrachlorodibenzo-*p*-dioxin
TEF	Toxic Equivalency Factor
TEQ	Toxic Equivalence
VROM	Ministry of Housing, Spatial Planning and the Environment
WHO	World Health Organisation

CHAPTER 1. INTRODUCTION

1.1. The project 'Setting Integrated Environmental Quality Standards'

This report is produced in the context of the project 'Setting Integrated Environmental Quality Standards'. The project started in 1989 as a result of the actions proposed in the National Environmental Policy Plan (NEPP) (VROM, 1989a). The aim of the project is to derive concentration limits for substances in the environment for the different compartments, air, water, sediment and soil based on the risk philosophy of the Ministry of Housing, Spatial Planning and the Environment (Ministry of VROM). This philosophy is laid down in the policy document "Premises for Risk Management" (VROM, 1989b). The concentration limits are referred to as Environmental Quality Standards (EQSs) in Dutch Environmental Policy.

The EQSs set by the Ministry of VROM are based on risk limits, the Maximum Permissible Concentrations (MPC), presented in this report for polychlorinated biphenyls. The MPCs are derived using data on (eco)toxicology and environmental chemistry, and represent the potential risk of the substance in question. The derivation of the MPC is performed at the National Institute of Public Health and the Environment (RIVM), under the authority of the Ministry of Housing, Spatial Planning and the Environment (Ministry of VROM). A commission of experts from a variety of governmental research institutes, industry and non-governmental organisations is involved in the supervision of the derivation of risk limits. For this specific report, a second expert commission (scientific counselling group) with experts on the ecotoxicology and environmental chemistry of PCBs from universities and research institutes was involved in the preparation of this report. The process of deriving integrated EQSs is shown schematically in Figure 1.1.
The results which are obtained until now in the project 'Setting Integrated Environmental Quality Standards' are laid down in several reports. In the report 'Desire for Levels' (Van de Meent et al., 1990) a methodology was proposed for deriving MPCs for several compounds such as heavy metals, chlorophenols, pesticides and polycyclic aromatic hydrocarbons (PAHs). Based on this method, EQSs for water, sediment and soil were set by the Minister of VROM (VROM, 1991). In following reports, MPCs were proposed for nine trace metals (Van de Plassche and De Bruijn, 1992), several volatile compounds (Van de Plassche and Bockting, 1993) and substances with a potential for secondary poisoning (Van de Plassche, 1994). The MPCs for PAHs as derived in Van de Meent et al. (1990), have been updated by Kalf et al. (1995; 1997). The MPCs for metals and pesticides have been updated recently by Crommentuijn et al. (1997ab). MPCs for aniline derivatives have been derived by Reuther et al. (1998), and risk limits for boron, silver, titanium, tellurium, uranium and organosilicon compounds are derived in Van de Plassche et al. (1999). The MPCs derived until 1997 are summarised by IWINS (1997).

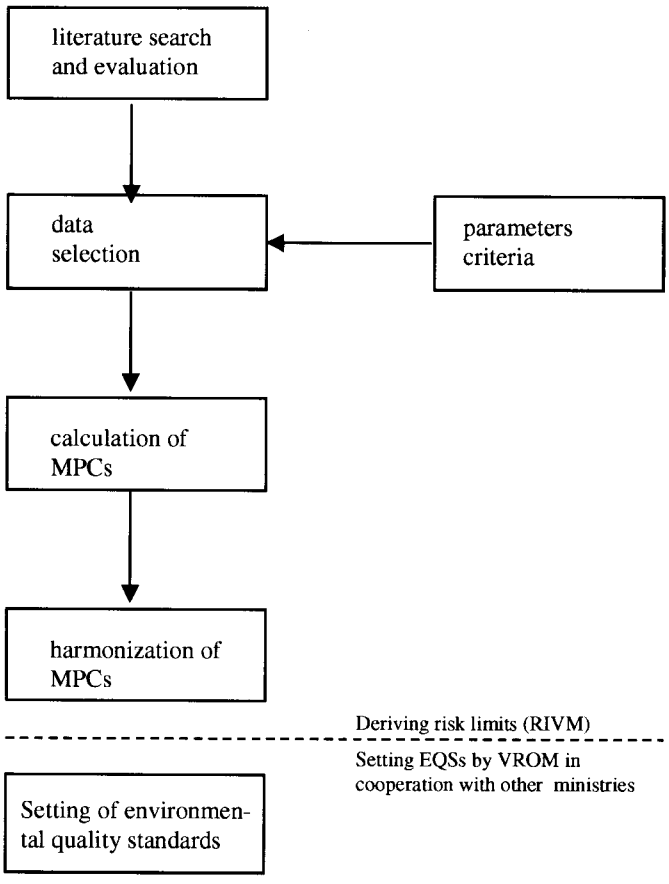

Figure 1.1 The process of deriving Integrated Environmental Quality Standards.

1.2. Selection of PCBs for the derivation of MPCs

In the present report MPCs are derived for polychlorinated biphenyls (PCBs). The environmental quality standards for PCBs that are currently in use in the Netherlands (IWINS, 1997; Van de Guchte 1998) are not underpinned by data on ecotoxicology and environmental chemistry.

As inheritance of the past relatively high concentrations of PCBs can still be encountered in sediments and soils. For a proper management of these contaminated sediments and soils, information on the ecotoxicological risk is essential.
For general information on PCBs it is referred to chapter 2.

Starting points in the derivation of MPCs for PCBs are:
- The MPC should protect the ecosystem.
- As sediments and soils are the major sinks for PCBs; organisms are exposed to PCBs via water and food from those sinks. PCBs are seldom measured in water or air, therefore MPCs are derived for sediments and soil.
- Information on occurrence of routinely analysed PCBs from monitoring programmes, must be able to be used in comparison with the MPCs.

A choice had to be made for which of the PCB-congeners an MPC is derived. For the non-ortho and mono-ortho substituted PCBs congeners (such as congeners #77, #105, #118, #126, #156, #157 and #169) much more toxicity data are available than for the multiple-ortho substituted congeners. They are considered the most toxic congeners; their supposed mechanism of action is via the aryl hydrocarbon receptor (AhR). For multiple-ortho substituted congeners less toxicological information is available, however they can certainly exert toxic action via other mechanisms than the AhR-mediated toxicity e.g. on the reproductive system, neurotoxicity, carcinogenity etc.
Monitoring studies on PCBs focused on congeners that occur in high concentrations and can be analysed with high accuracy. In the Netherlands, a set of 7 PCBs is routinely analysed (congeners #28, #52, #101, #118, #138, #153 and #180, numbers refer to Table 2.1.), later extended by congeners #18, #44, #49, #170 and #187 (Akkerman, 1997). For these congeners information on environmental concentrations (e.g. in sediment) and on environmental fate (e.g. the *n*-octanol/water partition coefficient, solubility, bioconcentration and -magnification) is readily available. Information on toxicity however is scarcely available for these congeners. Therefore MPCs for these individual congeners cannot be well founded by toxicological information. As one of the objectives of monitoring is to follow trends in time, it is highly desirable that measurement of the aforementioned congeners is continued. A second objective of monitoring is to compare the measured concentrations to EQSs, in order to evaluate policy, and to set priorities based on risk assessment. So, the MPCs derived in this report must be able to be used in comparison with monitored concentrations.

Based on the aforementioned considerations, MPCs are derived for the congeners #77, #105, #118, #126, #153, #156, #157 and #169. Congeners #77, #126 and #169 are not substituted on the ortho position, and congeners #105, #118, #156 and #157 are substituted on one of the ortho-positions. Both the non- and the monosubstituted congeners act via AhR-mediated toxicity. Congener nr. #153 is substituted on two ortho-positions, and is thought to stand for other toxic mechanisms exerted by PCBs. In addition, a mixture-MPC is derived for the non- and mono-ortho substituted PCBs, which are assumed to be concentration-additive. This mixture-MPC is expressed as concentration of PCB #118 as a guide. The mixture-MPC can be used to evaluate concentrations monitored in the environment.

Table 1.1. Congeners for which an MPC is derived

PCB	Substitution pattern		routinely analysed?
#77	3,3',4,4'	non-ortho	no
#105	2,3,3',4,4'	mono-ortho	no
#118	2,3',4,4',5	mono-ortho	yes
#126	3,3',4,4',5	non-ortho	no
#153	2,2',4,4',5,5'	di-ortho	yes
#156	2,3,3',4,4',5	mono-ortho	no
#157	2,3,3',4,4',5'	mono-ortho	no
#169	3,3',4,4',5,5'	non-ortho	no

As PCBs are seldom measured in water or air, no MPCs were derived for these matrices. Toxicity data were recalculated into an equivalent toxic concentration in organic carbon of sediment/soil. In this process, accumulation via the food chain was taken into account. Data on environmental fate of PCBs, such as biomagnification

factors and biota-to-sediment accumulation factors were used in the calculations. Probabilistic techniques were used, to incorporate the wide variety in data for the above-mentioned environmental fate processes in the derivation of MPCs.

CHAPTER 2. POLYCHLORINATED BIPHENYLS

2.1 Introduction

PCBs consist of two aromatic ring structures, linked by a single bond and substituted with 1 to 10 chlorine atoms.
The general structure of PCBs is given in Figure 2.1.

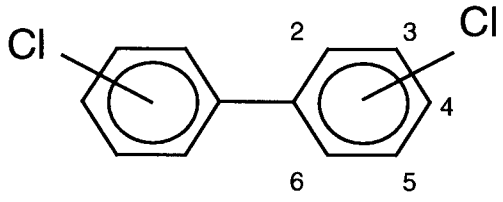

Figure 2.1. General structure of polychlorinated biphenyls.

Table 2.1. Structure of 209 PCB congeners and their IUPAC numbers (Ballschmitter and Zell, 1980)

no	Structure	no	structure	no	structure	no	Structure	No	structure
1	2	43	2,2',3,5	85	2,2',3,4,4'	127	3,3',4,5,5'	169	3,3',4,4',5,5'
2	3	44	2,2',3,5'	86	2,2',3,4,5	128	2,2',3,3',4,4'	170	2,2',3,3',4,4',5
3	4	45	2,2',3,6	87	2,2',3,4,5'	129	2,2',3,3',4,5	171	2,2',3,3',4,4',6
4	2,2'	46	2,2',3,6'	88	2,2',3,4,6	130	2,2',3,3',4,5'	172	2,2',3,3',4,5,5'
5	2,3	47	2,2',4,4'	89	2,2',3,4,6'	131	2,2',3,3',4,6	173	2,2',3,3',4,5,6
6	2,3'	48	2,2',4,5	90	2,2',3,4',5	132	2,2',3,3',4,6'	174	2,2',3,3',4,5,6'
7	2,4	49	2,2',4,5'	91	2,2',3,4',6	133	2,2',3,3',5,5'	175	2,2',3,3',4,5',6
8	2,4'	50	2,2',4,6	92	2,2',3,5,5'	134	2,2',3,3',5,6	176	2,2',3,3',4,6,6'
9	2,5	51	2,2',4,6'	93	2,2',3,5,6	135	2,2',3,3',5,6'	177	2,2',3,3',4',5,6
10	2,6	52	2,2',5,5'	94	2,2',3,5,6'	136	2,2',3,3',6,6'	178	2,2',3,3',5,5',6
11	3,3'	53	2,2',5,6'	95	2,2',3,5',6	137	2,2',3,4,4',5	179	2,2',3,3',5,6,6'
12	3,4	54	2,2',6,6'	96	2,2',3,6,6'	138	2,2',3,4,4',5'	180	2,2',3,4,4',5,5'
13	3,4'	55	2,3,3',4	97	2,2',3',4,5	139	2,2',3,4,4',6	181	2,2',3,4,4',5,6
14	3,5	56	2,3,3',4'	98	2,2',3',4,6	140	2,2',3,4,4',6'	182	2,2',3,4,4',5,6'
15	4,4'	57	2,3,3',5	99	2,2',4,4',5	141	2,2',3,4,5,5'	183	2,2',3,4,4',5',6
16	2,2',3	58	2,3,3',5'	100	2,2',4,4',6	142	2,2',3,4,5,6	184	2,2',3,4,4',6,6'
17	2,2',4	59	2,3,3',6	101	2,2',4,5,5'	143	2,2',3,4,5,6'	185	2,2',3,4,5,5',6
18	2,2',5	60	2,3,4,4'	102	2,2',4,5,6'	144	2,2',3,4,5',6	186	2,2',3,4,5,6,6'
19	2,2',6	61	2,3,4,5	103	2,2',4,5',6	145	2,2',3,4,6,6'	187	2,2',3,4',5,5',6
20	2,3,3'	62	2,3,4,6	104	2,2',4,6,6'	146	2,2',3,4',5,5'	188	2,2',3,4',5,6,6'
21	2,3,4	63	2,3,4',5	105	2,3,3',4,4'	147	2,2',3,4',5,6	189	2,3,3',4,4',5,5'
22	2,3,4'	64	2,3,4',6	106	2,3,3',4,5	148	2,2',3,4',5,6'	190	2,3,3',4,4',5,6
23	2,3,5	65	2,3,5,6	107	2,3,3',4',5	149	2,2',3,4',5',6	191	2,3,3',4,4',5',6
24	2,3,6	66	2,3',4,4'	108	2,3,3',4,5'	150	2,2',3,4',6,6'	192	2,3,3',4,5,5',6
25	2,3',4	67	2,3',4,5	109	2,3,3',4,6	151	2,2',3,5,5',6	193	2,3,3',4',5,5',6
26	2,3',5	68	2,3',4,5'	110	2,3,3',4',6	152	2,2',3,5,6,6'	194	2,2',3,3',4,4',5,5'
27	2,3',6	69	2,3',5,6	111	2,3,3',5,5'	153	2,2',4,4',5,5'	195	2,2',3,3',4,4',5,6
28	2,4,4'	70	2,3',4',5	112	2,3,3',5,6	154	2,2',4,4',5,6'	196	2,2',3,3',4,4',5,6'
29	2,4,5	71	2,3',4',6	113	2,2',3',5',6	155	2,2',4,4',6,6'	197	2,2',3,3',4,4',6,6'
30	2,4,6	72	2,3',5,5'	114	2,3,4,4',5	156	2,3,3',4,4',5	198	2,2',3,3',4,5,5',6
31	2,4',5	73	2,3',5',6	115	2,3,4,4',6	157	2,3,3',4,4',5'	199	2,2',3,3',4,5,5',6'
32	2,4',6	74	2,4,4',5	116	2,3,4,5,6	158	2,3,3',4,4',6	200	2,2',3,3',4,5,6,6'
33	2',3',4	75	2,4,4',6	117	2,3,4',5,6	159	2,3,3',4,5,5'	201	2,2',3,3',4,5',6,6'
34	2',3',5	76	2',3,4,5	118	2,3',4,4',5	160	2,3,3',4,5,6	202	2,2',3,3',5,5',6,6'
35	3,3',4	77	3,3',4,4'	119	2,3',4,4',6	161	2,3,3',4,5',6	203	2,2',3,4,4',5,5',6
36	3,3',5	78	3,3',4,5	120	2,3',4,5,5'	162	2,3,3',5,5'	204	2,2',3,4,4',5,6,6'
37	3,4,4'	79	3,3',4,5'	121	2,3',4,5',6	163	2,3,3',4',5,6	205	2,3,3',4,4',5,5',6
38	3,4,5	80	3,3',5,5'	122	2',3,3',4,5	164	2,3,3',4',5',6	206	2,2',3,3',4,4',5,5',6
39	3,4',5	81	3,4,4',5	123	2',3,4,4',5	165	2,3,3',5,5',6	207	2,2',3,3',4,4',5,6,6'
40	2,2',3,3'	82	2,2',3,3',4	124	2',3,4,5,5'	166	2,3,4,4',5,6	208	2,2',3,3',4,5,5',6,6'
41	2,2',3,4	83	2,2',3,3',5	125	2',3,4,5,6'	167	2,3',4,4',5,5'	209	2,2',3,3',4,4',5,5',6,6'
42	2,2',3,4'	84	2,2',3,3',6	126	3,3',4,4',5	168	2,3',4,4',5',6		

There are 209 congeners, which are numbered according to Table 2.1. (Ballschmitter & Zell, 1980). The positions 2, 2', 6 and 6' are the ortho-positions; 3, 3', 5 and 5' are the meta positions; and 4 and 4' are the para positions. If there is no substitution of the ortho-position, the PCBs can adopt a planar configuration. When two or more ortho positions are substituted, no coplanarity of the two phenyl rings is possible.

PCBs are banned in industrialised countries since the 1980s, but have been used abundantly in the past. They still enter the environment by leakage, recycling, transboundary influx via the major rivers and long-range atmospheric transport (Annema et al, 1995; Ockenden et al., 1998). PCBs are biomagnified in the food chain, and found in relatively high concentrations in top predators such as otters, seals and fish-eating birds (e.g. Leonards et al., 1998).

2.2 Toxic Responses

Many toxic responses are described for dioxin-like as well as non-planar PCBs: hepatotoxicity, body weight loss, thymus atrophy, impairment of immune responses, dermal lesions, reproductive toxicity, alterations in vitamin A and thyroid hormone metabolism, (developmental) neurotoxicity, teratogenicity and promotor activity in carcinogenesis (e.g. Shain, 1991; Safe, 1994; Leonards et al., 1995; Ross et al., 1995; Darnerud et al., 1996; Niimi, 1996).

2.2.1. Reproductive toxicity

PCBs and their metabolites are reported to disrupt the endocrine and reproductive system (Colborn et al. 1993). Feminisation of fish-eating birds and diminished breeding results have been related to PCB contamination (EPA, 1997). Exposure to environmentally realistically concentrations of Aroclor 1260 resulted in altered sex ratios in newly hatched rainbow trout (Matta et al., 1998).
The rigid para-hydroxylated ortho-substituted PCBs are suggested to belong to the strongest estrogen-receptor binding xenobiotics (Korach et al., 1988; McKinney & Waller, 1994; Vethaak & Opperhuizen, 1996; Gierthy et al. 1997). It was shown that PCB compounds that showed receptor binding activity also increased *in vivo* the uterine weight (Korach et al. 1988). The estrogenic activity is not related to binding to the aryl hydrocarbon receptor (see 2.2.3.); (hydroxylated) PCB congeners that do not bind strongly to AhR are reported to have estrogenic effects. For chemicals that strongly bind to the AhR, such as 2,3,7,8-TCDD, on the contrary an anti-estrogenic activity is reported (Safe et al., 1991).
PCBs and their metabolites may influence the reproductive system by changing hormone metabolism. PCBs induce the enzyme CYP1A1; several steroids bind strongly to this enzyme (Chan & Hollebone, 1995) and are probably biotransformated thereby.

2.2.2. Immunotoxicity

Mass mortalities among seals, for example after the phocine distemper virus (PDV) epizootic in Europe 1988, were related to environmental pollution causing an impaired immune function (e.g. Hall et al., 1992). In seals which were fed field contaminated fish for 2.5 years, a diminished immunological response to ovalbumin

was observed (Ross et al. 1995) and lymphocytes were functionally impaired (De Swart et al., 1994), whereas in seals that were fed relatively unpolluted fish these effects were much less observed. It was made plausible by the authors that PCBs contributed for the greater part to the observed effects (Ross et al., 1995).
For Danish otters, a relation was found between the incidence of diseases as well as the number of diseases per sick otter and the concentration of PCBs expresses as TEQ (Leonards et al., 1996). A causal relationship was not demonstrated and other compounds may have attributed to the disease incidences. However, PCBs contributed for over 90% to the total TEQ-concentration.

2.2.3. Mechanism of toxicity mediated by the Ah-receptor
The non-ortho PCBs and to a lesser extent the mono-ortho PCBs may adopt a coplanar configuration. They are thought to work via the cytosolic aryl hydrocarbon receptor (AhR), similar to the mechanism of dioxins:
When dioxin-like compound binds to the AhR; heat-shock proteins are abstracted which results in a conformational change of the AhR. The Ah-receptor nuclear translocator (Arnt) mediates the transport of the ligand-receptor complex to the nucleus. In the nucleus the complex binds to so-called dioxin-responsive elements (DREs) at the DNA, after which genes are expressed coding for CYP 1A1 or toxic effects (Goldstein & Safe, 1989; Perdew, 1988; Hoffman et al. 1991; Denison et al., 1989; Whitlock, 1990).
Many of the toxic responses described for PCBs have been related to this mechanism of action, although some are not. For reproductive toxicity, neurotoxicity, and alterations in thyroid hormone levels, multiple ortho-substituted PCBs or hydroxylated PCBs show effects. Therefore, these effects are not believed to be exerted via the AhR-mediated pathway.

2.2.4. Toxic equivalency factors
Toxic equivalency factors (TEFs) relate the toxicity of an individual dioxin-like compound such as a PCDD, PCDF or PCB congener, to the toxicity of 2,3,7,8-TCDD which is set to unity (e.g. Safe, 1994; Ahlborg et al., 1994; Van den Berg et al., in press). TEFs are derived for congeners that have a structural similarity with PCDDs and PCDFs, which bind to the aryl hydrocarbon receptor, which have dioxin-like biochemical and toxicological effects and which are persistent and accumulative in the food chain. As TEFs apply to AhR-mediated responses, the concept cannot be applied to toxicity by other mechanisms such as reproductive toxicity. The relative potency of a congener is dependent on the species as well as the effect studied. TEFs are based upon multiple *in vivo* and *in vitro* studies, with toxic as well as biochemical endpoints. Preferably *in vivo* studies with toxic endpoints are used, however, these are not always available.
With help of TEFs, the toxicity of a mixture can be expressed as TEQ (toxic equivalency of exposure to the single chemical 2,3,7,8-TCDD) by multiplying the concentration of each individual congener with its TEF and summing these products for all compounds. It is assumed that the combined effects of the different congeners are additive, although it is known that some PCBs are antagonistic or synergistic with dioxins (Van den Berg, 1994).

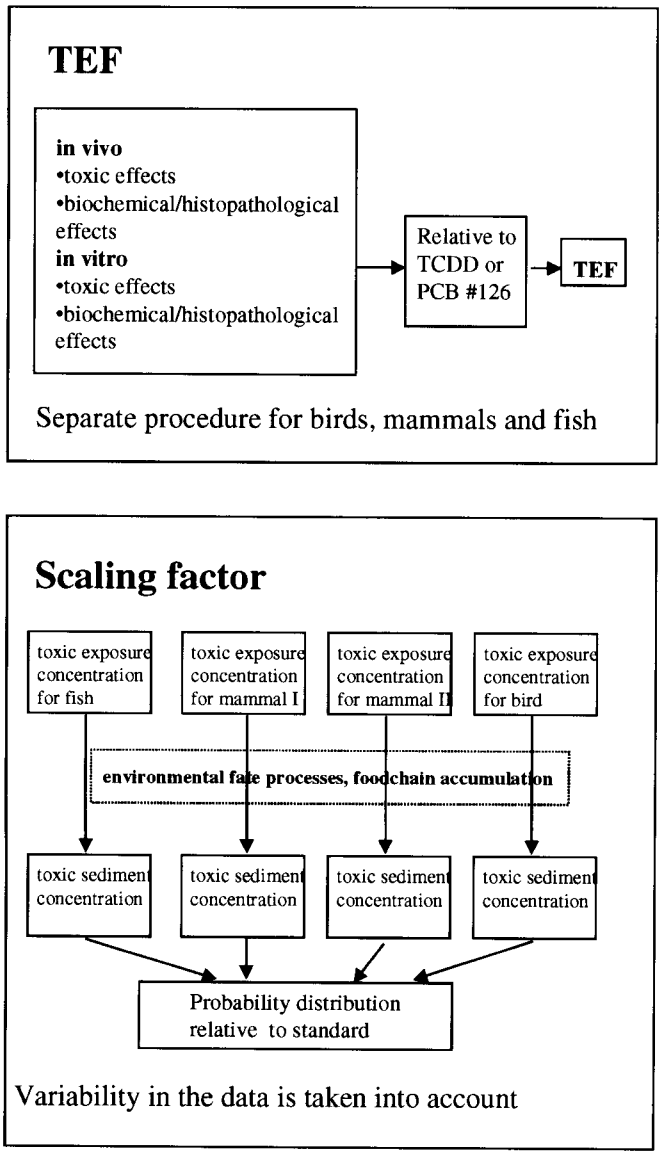

Figure 2.2. Overview on differences in procedure to derive TEFs or scaling factors.

In the present study TEFs are not used in the derivation of MPCs for single congeners, as sufficient ecotoxicological data as well as data on environmental fate are available for the individual congeners. Neither are the TEFs used in the derivation of a mixture-MPC (see chapter 6). Instead the equivalent effect concentrations in the organic carbon of sediment/soil to combined species, are scaled by scaling factors (see Fig. 2.2.). Per congener, a scaling factor is derived based upon the same information as used for deriving the MPCs. Environmental fate processes and food-chain accumulation are incorporated in the scaling factors. This information on environmental chemistry of the congeners is not incorporated in TEFs. Another difference between TEFs and scaling factors is that in scaling factors, only endpoints related to toxic effects measured in *in vivo* studies are included. In the derivation of TEFs biochemical and toxicological effects as measured in *in vivo* and *in vitro* studies are incorporated. Finally, TEFs are derived for specific species or groups of species (mammals, birds, fishes), whereas the scaling factors integrate combined data over different species.

2.3. Incorporation of the different toxic mechanisms exerted by PCBs in the derivation of MPCs

As stated in 1.2., MPCs are derived for the congeners #77, #105, #118, #126, #153, #156, #157 and #169. For the planar congeners that exert AhR-mediated toxicity, i.e. except for #153, a mixture MPC is derived which is based on concentration addition of the planar congeners.
Congener nr. #153 is substituted on two ortho-positions, and is thought to represent other toxic mechanisms exerted by PCBs. Several studies show that multiple-ortho substituted PCBs exert toxic effects which probably are not mediated by AhR (e.g. Davis & Safe, 1990; Stonard & Grieg, 1976). Examples are the aforementioned reproductive toxicity, promotor-activity in the presence of genotoxic compounds (Silberhorn et al. 1990; Safe, 1994), effects on thyroid hormones and on vitamin A and K metabolism (Van Birgelen et al., 1992; Bouwman et al., 1992), and neurotoxic effects (Seegal et al. 1990; Safe, 1994). Also for the mechanisms of toxicity that are not AhR-mediated, concentration-addition with other congeners is plausible. Because of a lack of toxicological data for other congeners, however, no mixture toxicity could be taken into account for these effects.

CHAPTER 3. METHODOLOGY

3.1. General scheme and deviation from standard procedures

Sediments and soils are a major sink for PCBs. From the organic carbon in sediments and soils, PCBs repartition into (pore)water, air and organisms. Most monitoring activities on PCBs concentrate on sediments and soils.
The Maximum Permissible Concentration (MPC) is derived based upon equivalent toxic concentrations in the sediment/soil, taking into account secondary poisoning. Therefore all individual toxicity data are recalculated into equivalent toxic concentrations in the sediment/soil (cf. Starodub et al., 1996). For the compartments water and air, no MPCs are derived.
The procedure used to derive MPCs for PCBs deviates on certain points from the procedures as earlier used and described in CSR (1996), Van de Plassche (1994) and Crommentuijn et al. (in prep.). Where there are no adjustments, in the following paragraphs the normal procedure is described in short. The adjustments are summarised below, and explained in the subsequent paragraphs:

- For toxicity studies on mammals, studies with dosing per diet, gavage as well as per injection are evaluated and included when relevant. For birds, egg injection studies are included in the derivation of MPCs. For fish, studies with dosing directly to eggs are included when relevant (see 3.3). For all types of studies where organisms are directly dosed, i.e. per injection or gavage, it is assumed that 100% of the administered dose is taken up and distributed evenly over the lipids (see 3.3.). For diet studies, the whole-body concentration is estimated using a one-compartment bioaccumulation model (see 3.3.).
- For each species and for each compound the most sensitive toxicity test is selected. Not only NOEC values are included but also other percentages of effect such as EC50s (see 3.3.).
- Toxicity data from studies on aquatic organisms, mammals and birds are recalculated into equivalent toxic concentrations in sediment or soil. Bioconcentration factors, biota-to-sediment concentration factors and biomagnification factors are used in these calculations (see 3.4.). In this way all types of studies can be readily compared on one and the same concentration axis, and can be integrated into one MPC. The procedure used deviates from previously used methods to incorporate risks for compounds with a potential for secondary poisoning (as described by Van der Plassche, 1994). In the discussion (8.2.) a comparison is made with this previously used method.
- The variability in the parameters in the above-mentioned calculations is taken into account. Distributions are fitted based on the information gained from the literature, and these distributions are used in the calculations instead of absolute values (probabilistic modelling, see 3.4.).
- Probability distributions for different species based on the individual toxicity data are combined, and new distributions are fitted including all species. The 5^{th} percentile of the combined distribution on bird and mammal toxicity data is set as MPC (see 3.5. and 3.6.).

3.2. Data collection

Data are collected on the toxicity of the chosen PCB-congeners for aquatic and terrestrial organisms, mammals and birds. In addition, data are collected on the environmental fate of the PCBs, i.e. data on the bioconcentration factor (BCF, the lipid normalised concentration ratio between an organism and the surrounding water, C_o/C_w), the biomagnification factor (BMF, the lipid normalised concentration ratio between predator and prey, $C_{o,predator}/C_{o,prey}$), the biota-to-sediment/soil accumulation factor (BSAF, the lipid and organic carbon normalised concentration ratio between an organism and sediment or soil, $C_o/C_{o.c.}$), the organic carbon normalised partition coefficient between sediment/water or soil/water (K_{oc}) and the partition coefficient between *n*-octanol and water (K_{ow}).
As toxicity data are often expressed on a wet weight basis, data are collected on the lipid content of organisms in order to lipid-normalise the toxicity data.

Sources used for the collection of data are:
- The documentation present at the Centre for Substances and Risk Assessment (CSR),
- The library of the Centre for Substances and Risk Assessment and that of the National Institute of Public Health and the Environment,
- On-line search in databases containing evaluated data: Aquire (AQUatic Information Retrieval, sponsored by the US EPA, Duluth, Minnesota), Aquapol 1.04 (1996, Ministry of Transport, Public Works and Water Management), AQUATOX version 3.20 (1997, BKH Consulting Engineers, Delft, The Netherlands) and DOSE (1997, The Directionary Of Substances and their Effects, The Royal Society of Chemistry).
- On-line search in the bibliographic databases Biosis, Toxline and Chemical Abstracts
- Retrospective literature search using public literature and reviews as a basis
- Data collected in the scientific counselling group

Data are collected until summer 1998.
A toxicity study is considered reliable if the design of the experiment is in agreement with international accepted guidelines such as the OECD guidelines (OECD, 1984a-e, 1992a-b). To judge studies which have not been performed according to these guidelines, criteria are developed at the Centre for Substances and Risk Assessment (CSR, 1996).

3.3. Data selection

Effects on growth, reproduction, or mortality are used in the derivation of MPCs. For each species and for each compound the most sensitive toxicity test is selected, and used in the extrapolation towards an MPC. Not only NOEC values are included but also other percentages of effect such as EC50s. This is evaluated in section 8.3., by studying the steepness of the dose-response curves, and the differences in sensitivity between several endpoints.

For toxicity studies on mammals and birds, diet studies are considered the most reliable. Other, and more often used, dosing methods include dosing per gavage or per injection. Until now, only diet studies have been used in the derivation of MPCs (CSR, 1996). In the current study also studies where dosing takes place per gavage or per injection are included. It is assumed that the injected dose partitions homogeneously over the lipids of the egg or the organism. The inclusion of these data will be evaluated in section 8.3., by comparing the results of the different types of studies.

3.4. Recalculating toxicity data into equivalent toxic concentrations in the soil/sediment

The toxicity data of the different species are recalculated into equivalent toxic concentrations in the organic carbon fraction of soil or sediment.
For all studies with dosing per injection or per gavage, it is assumed that 100% of the administered dose is taken up.
For diet-studies and multiple oral gavage studies for mammals, the concentration in the organism is estimated using a one-compartment bioaccumulation model (cf. Leonards et al., 1995).

Toxicity data that are expressed as water concentrations (NOEC, C_w), are translated into equivalent no-effect concentrations in the organic carbon of sediment/soil ($NOEC_{oc}$) by using the ratio BCF/BSAF:

$$NOEC_{oc} = NOEC_w \frac{BCF}{BSAF}$$ Equation 1

Aquatic toxicity data that are obtained by injection of fish eggs (LD50, C_o) are translated to an equivalent toxic concentration in the organic carbon by:

$$LC50_{oc} = \frac{LD50_o}{l.f._{fishegg} \cdot BSAF}$$ Equation 2

where $l.f._{fish\ egg}$ stands for the lipid fraction in the fish eggs.
For diet-studies and multiple oral gavage studies for mammals, the concentration in the organism (C_o, mg/kg) is estimated as follows (cf. Leonards et al., 1995):

$$C_o = \frac{A \cdot R \cdot C_f}{K}(1 - e^{-Kt})$$ Equation 3

in which A stands for the assimilation efficiency, R stands for the daily ration of food (kg/kg, from annex II to WHO, 1990), C_f stands for the concentration of PCBs in the food (mg/kg). For studies whit dosing via oral gavage, $R \cdot C_f$ is replaced by the daily gavage concentration (mg/kg). K stands for the elimination rate (day^{-1}), and is for mustelids extended by the secretion rate via the anal gland which is assumed constant at 0.0165 day^{-1} (Leonards et al., 1995). Finally, t is the duration of the diet (day).

Values for K and A are taken from Leonards et al., 1995. In the information in appendix IV, toxicity is expressed in mg/kg body weight.
The toxic concentration is lipid-normalised, then a one-level food-chain is assumed. The recalculation into equivalent toxic concentration in the sediment takes place using the following formula:

$$EC50_{oc} = \frac{ED50_{o,w.w.}}{l.f._m \cdot BMF \cdot BSAF} \qquad \text{Equation 4}$$

in which $l.f._m$ stands for the lipid fraction of the mammal.

Almost all data on toxicity of PCBs to birds are obtained in studies with dosing via egg injection. The dose in the egg is lipid-normalised first. Then, a correction for the transfer of PCBs from the parent bird to the egg is applied (EBR; the concentration ratio between the egg and the parent on a lipid weight basis). In this way a lipid-normalised concentration in the parent is obtained. A one-level food-chain is assumed, so the recalculation into equivalent concentration in sediment takes place by dividing the toxicity data by the BMF and the BSAF. The resulting equation is:

$$EC50_{o.c.} = \frac{ED50_{egg,w.w.}}{l.f._{bir\ deg\ g} \cdot EBR \cdot BMF \cdot BSAF} \qquad \text{Equation 5}$$

3.5. Including variability in the parameters; probabilistic modelling

The values for the parameters used in the equations given in 3.4. are based upon literature information. The variability in these data is taken into account by using probabilistic modelling (Traas et al, 1996; Jongbloed et al., 1996).
Distributions are fitted to the literature data on the diverse parameters using the software package Crystal Ball 4.0 (Decisioneering, Denver CO). Then the calculations are performed; the distributions for the different parameters involved in a calculation are sampled by Latin Hypercube sampling. Each calculation is performed 1000 times, using Crystal Ball 4.0. The described procedure yields a probability distribution of the equivalent toxic concentration in the organic carbon of soil or sediment for each original toxicity value.

3.6. Deriving MPCs

In order to derive an MPC for PCBs, data are extracted from each probability distribution (1000 data per distribution). Per congener, these data are combined and new distributions are fitted to these combined data. These combined distributions are normal distributions based on the logtransformed data. Combined distributions are fitted for all toxicity data together, or for only the data on birds and mammals. This distinction is made as in the case that aquatic species are far less sensitive than birds and mammals, a bimodal distribution might be the result. The goodness-of-fit of the distributions to all data or to all mammal and bird data is tested by the Kolgomorov-

Smirnov test. If the s-value is lower than 0.1, the distribution is accepted to base the MPC upon. If not, the most sensitive probability distribution is chosen as a basis to derive the MPC.
Of the chosen distribution the 5th percentiles are taken and backtransformed into µg/kg o.c., these values are set as MPC. This procedure is analogous to the procedure as described by Aldenberg and Slob (1993), with the difference that probability distribution are used as input instead of single values.

3.7. Summary of procedure

For a summary of the whole procedure as described in the above, see figure 3.1.

Step 1: recalculation of individual toxicity data into a probability distribution of the equivalent toxic concentration in the organic carbon

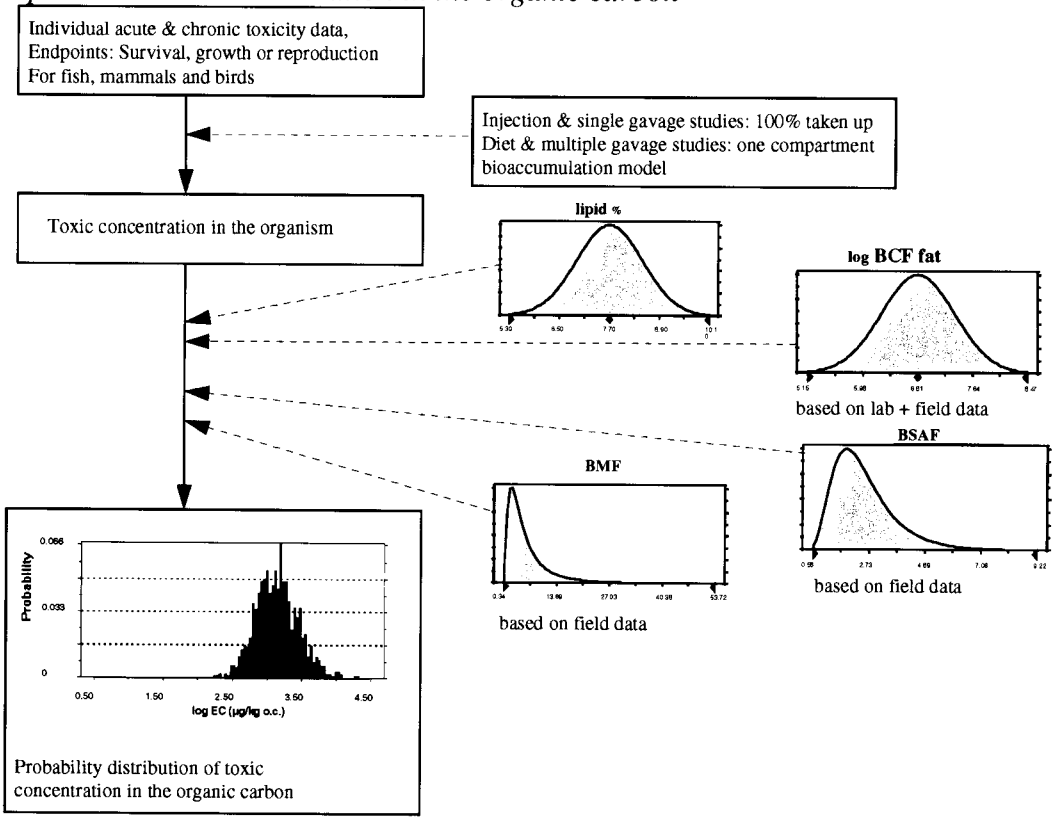

Figure 3.1.a.: Overview on the different steps taken in the derivation of MPCs for individual congeners of PCBs: step 1.

Step 2: Combining the individual probability distributions per congener

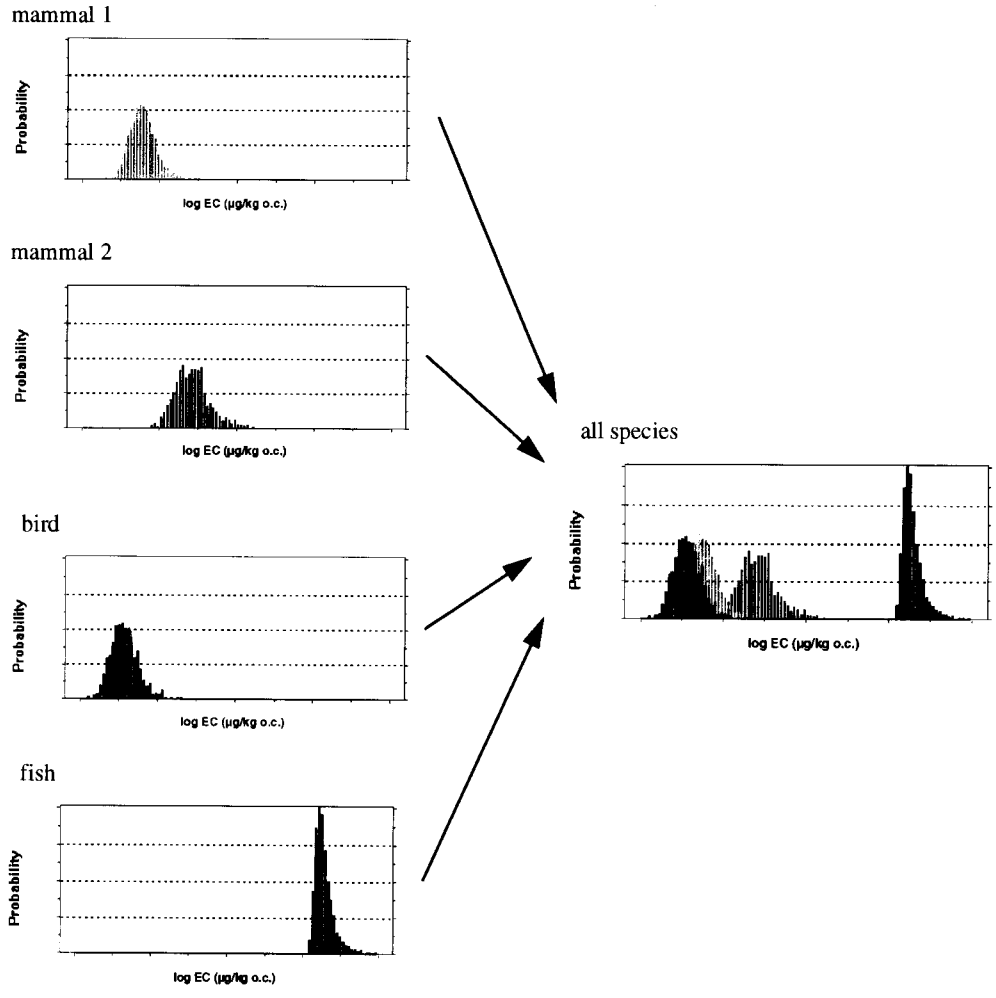

Figure 3.1.b.: Overview on the different steps taken in the derivation of MPCs for individual congeners of PCBs: step 2.

Step 3: Deriving the MPC for a congener

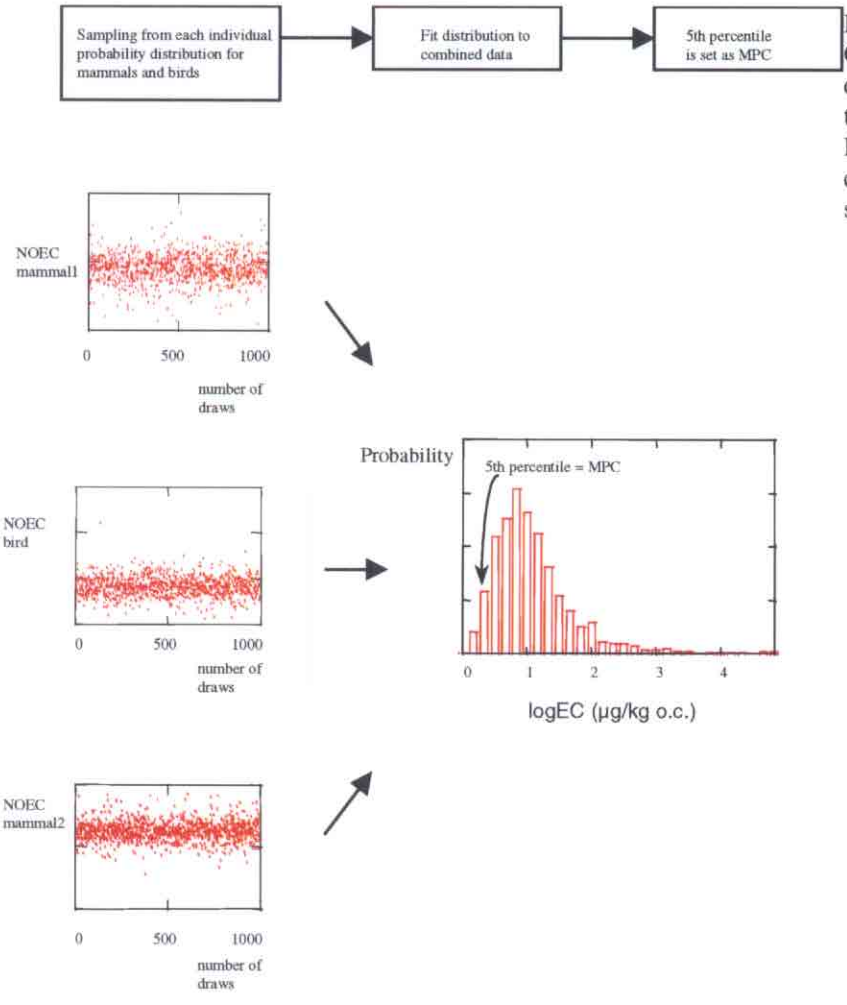

Figure 3.1.c.: Overview on the different steps taken in the derivation of MPCs for individual congeners of PCBs: step 3.

CHAPTER 4. DESCRIPTION OF THE DATA UNDERLYING THE MPC

4.1 Data on the toxicity of PCBs

Data on the toxicity of PCBs to aquatic organisms, mammals and birds can be found in appendices 1.a.-1.e. The toxicity data were collected, evaluated and selected as described in chapter 3. In table 4.1., an overview is given on those toxicity data that are finally used in the derivation of the MPC for the different congeners.

4.1.1. Acute toxicity of PCBs to aquatic organisms
Data are found for PCB #77, #105 and #153, see appendix 1.a..
In the time frame of acute toxicity tests, toxicity cannot be found at concentrations near the aqueous solubility for PCB #77 and #153.
These results are in accordance with the cut-off theory; for very hydrophobic compounds no acute toxicity can be measured (Veith et al., 1983). This effect is shown for compounds with a *n*-octanol/water partition coefficient roughly higher than 10^6.

4.1.2. Chronic toxicity of PCBs to aquatic organisms
Studies are performed for PCB #77, #118, #126 and #153, see appendix 1.b.
Toxicity in studies with PCB #118 and #153 did not occur at the highest tested concentration.

4.1.3. Toxicity of PCBs to aquatic organisms in injection experiments
Several authors exposed fish by direct injection of PCBs instead of exposure via the water phase. In appendix 1.c. the results of this type of study for the congeners of interest are summarised. Results are found for congeners #77, #105, #118, #126 and #153.

4.1.4. Toxicity of PCBs to mammals
The results of studies of the toxicity of PCBs to mammals can be found in appendix 1.d.. Rat, mouse, guinea pig, mink and monkey have been used as test animal.
The dosing methods of the data that are used in the MPC derivation vary; single or multiple intraperitoneal injections, single or multiple oral gavage, and diets are used.

4.1.5. Toxicity of PCBs to birds
In appendix 1.e. data are summarised on the toxicity of PCBs to birds. The exposure occurred in most cases by injection of the congener into the air sac or into the yolk of eggs. *Gallus gallus* is most often used as a test animal. Also other species are used, especially for PCB #77.
The PCB concentrations in the eggs are translated to concentrations in the parent birds by assuming an egg to parent ratio of 0.60±0.11 on a lipid basis. This value is derived from a field study on herring gulls (Braune & Norstrom, 1989), no significant differences between homologue groups are reported in this study. Few data on egg-to-parent bird ratio are available for PCBs (see also Bosveld & van den Berg, 1994). However by the absence of an influence of the substitution pattern it is implicated that the PCBs partition non-selectively between the various lipid pools in the avian

body so the mentioned value is also believed to be applicable to other bird species. A more recent study (Russell et al., 1999) did not lead to other conclusions. This value is assumed representative to all bird species used in the toxicity studies.

Table 4.1. Overview on the toxicity endpoints that are used in the derivation of the MPC for the different congeners

PCB congener	Aquatic organisms		Mammals		Birds	
	species	toxicity endpoint	species	toxicity endpoint	species	toxicity endpoint
77	*Salvelinus namaycush*, eggs	mortality, LD50	*Mus musculus*	reproduction, NOEC	*Falco sparverius*	mortality, LD50
	Oncorhynchus mykiss, eggs	mortality, LD50			*Meleagris gallopova*	mortality, LD60
	Daphnia magna	growth, NOEC			*Gallus gallus*	reproduction, NOEC
105	*Brachydanio rerio*	mortality, LC50	*Rattus rattus*	growth, ED50	*Gallus gallus*	growth, LOAEL
118			*Rattus rattus*	growth, NOEC	*Gallus gallus*	mortality, LD45
126	*Oryzias latipes*	mortality, NOEC	*Rattus rattus*	growth, NOEC	*Meleagris gallopova*	mortality, LD36
	Oncorhynchus mykiss, eggs	mortality, LD50			*Gallus gallus*	reproduction, NOEC
					Falco sparverius	reproduction, NOEC
153			*Rattus rattus*	growth, NOEC		
			Mustela vison	growth, NOEC		
156			*Mus musculus*	reproduction, LOEC	*Gallus gallus*	mortality, LD50
			Rattus rattus	growth, NOEC		
157			*Rattus rattus*	growth, ED50	*Gallus gallus*	mortality, LD50
169			*Cavia porcellus*	mortality, LD50	*Gallus gallus*	mortality, LD80
			Mus musculus	reproduction, NOEC		
			Rattus rattus	reproduction, NOEC		
			Mustela vison	growth, LOEC		

4.2. Lipid content of the different species

BCF, BSAF and BMF data are expressed on a lipid basis, while the toxicity data are expressed on a wet weight basis. Therefore, the toxicity data must be lipid-normalised before recalculating them into equivalent toxic concentrations in the organic carbon (see 3.4.). Information on the lipid content of the test organisms used is thus required. This information is not reported standard in toxicity studies.
Lipid content varies per species, but also within a species depending on the season, reproductive phase, food availability etc. For example, the lipid content of waders varies between less than 2 to over 40% through the season (Zwart et al., 1990). Lipid content of bird eggs vary less dramatically, however a distinction is observed between eggs of altricials -which stay a while in their nest after hatch- and eggs of precocials - species that fly almost immediately (Carey et al., 1980). In addition to the

(intra)species variation, the extraction method used for the determination of the lipid content influences the amount of lipids extracted (De Boer, 1988). For an impression of the ranges of lipid content (extractable lipids as percentage of the wet weight) in diverse mammal and bird species, and in bird and fish eggs, it is referred to appendix II.g.

In the calculations for the derivation of MPCs, a normal distribution of 7.7±0.8% is used for lipid content of bird eggs (Braune & Norstrom, 1989). This value is in the mean of values as reported in appendix II.g..
For lipid content of fish eggs a normal distribution of 10.1±0.2% is used (Fisk et al., 1997).
For lipid content of mammals two alternatives are used in the calculations. First, a normal distribution of 9.4±7.7% is used (Jongbloed et al. 1994), based upon a compilation of literature data on laboratory raised test organisms. This value is high compared to the range of lipid contents observed in mammals taken from the field (see appendix II.g.). However data from toxicity studies performed with laboratory animals, must be lipid-corrected. As laboratory animals are generally well-fed and do not have much movement, they can be assumed to have a higher lipid content than wild-living organisms. Second, a uniform distribution of the lipid content between 2 and 30% was used. The two alternatives are included to check the importance of the parameter; as the variability in the literature data is high, uncertainty in the assumption is a consequence.

4.3. Data on environmental fate of PCBs

4.3.1. Bioconcentration

Data on bioconcentration factors for fish and molluscs derived from laboratory studies are listed in appendix II.a.1. Data on BCFs for crustaceans are listed in appendix II.a.2. Data on bioconcentration derived from field studies are given in appendix II.d.
In Figure 4.1., for each congener the lipid-normalised BCF values from laboratory of field studies are related to log K_{ow} values (data from Hawker & Connell, 1988).
It is well known from the literature that a relationship exists between hydrophobicity (expressed as K_{ow}) and BCF for persistent organic compounds (e.g. Hermens, 1989). This relationship appears to be linear up to a log K_{ow} of about 5.5. At higher hydrophobicity the relation levels off (Hawker, 1990). Various explanations have been given for this phenomenon (McCarthy & Jimenez, 1985; Sijm et al., 1993; Loonen et al., 1994; Gover et al., 1996). From Figure 4.1. it is obvious that for the congeners considered here there is no relationship between BCF and log K_{ow}, which is most likely due to the fact that the data points are within the range where linearity is no longer observed.
BCFs from field studies seem somewhat higher than those derived from laboratory studies. Exposure to PCBs in the field is via both water and food/sediment, while in the laboratory studies exposure is mainly via the water. As for highly hydrophobic compounds (log $K_{ow} > 5$) it is known that exposure via the food contributes significantly to the accumulation in the organism (Belfroid, 1994; Weston, 1990; Thomann, 1989) this may explain the observed differences. PCB #153 is the only congener for which laboratory and field data could be compared statistically. For

PCB #153, no significant differences are observed in BCF-values derived from laboratory
or field studies (T-test). It is assumed that also for the other congeners there are no differences in laboratory- and field derived data. Thus, laboratory and field data are combined to fit distributions upon.
In Figure 4.2. wet weight based BCFs are depicted per congener for crustaceans, and for fish and molluscs. Crustaceans show lower values than fish and molluscs, explained by their lower lipid content.
Testing with a single factor ANOVA ($T<0.05$) showed that the available lipid-based BCF data did not differ significantly per congener, both considering only laboratory data and combined laboratory and field data. Therefore, for performing the calculations as described in chapter 3, one distribution was fitted on all log-transformed BCF data for fish and molluscs for all congeners.

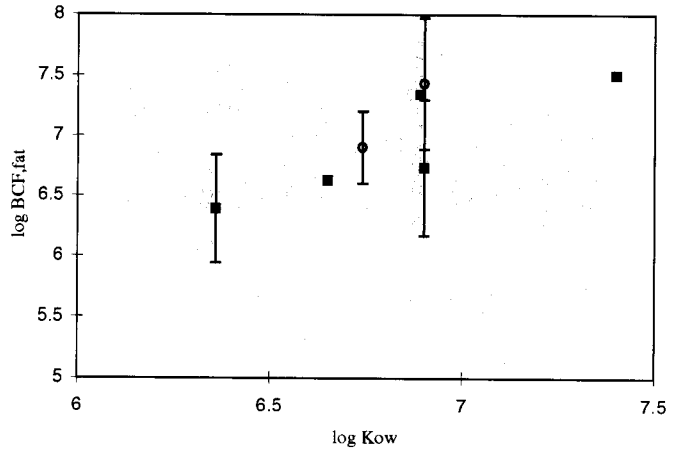

Figure 4.1. Bioconcentration factors for fish and molluscs for the PCBs under study as related to the log K_{ow}; in black square the mean and std. of data from laboratory studies, in open circle the mean and std of data from field studies.

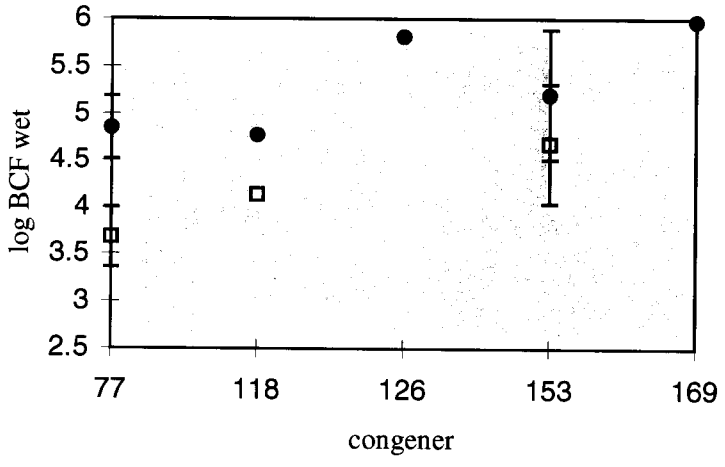

Figure 4.2. Bioconcentration factors for the relevant PCB congeners; in black circle the mean and std. of laboratory data for fish and molluscs, in open square the mean and std of laboratory data for crustaceans.

4.3.2. Biomagnification

Data on biomagnification can be found in appendix II.b. and II.e.. In appendix II.b. laboratory studies are summarised which are mainly performed with fish species, and in appendix II.e. data from field studies on mammals and birds are given. In Figure 4.3. the BMF values for the different congeners are depicted for both the laboratory and the field studies.
For the field-based BMFs, significant differences are observed between the congeners as tested by ANOVA. For birds, less data are found than for mammals. Most data are available for PCB #153, no significant differences are observed between the BMF for birds or mammals. This was tested by two-sample T-test assuming equal variances. So, distributions for BMFs are fitted to the data per congener, one distribution is used for both birds and mammals. The distributions are lognormal, and based on the field data.
For harbour seals, lipid-normalised biomagnification factors are equal to or lower than 3.9 for a whole range of PCB congeners (Boon et al., 1987 and 1992). Except for congener #118 and #153 no data are generated in the mentioned studies on the congeners discussed in this report. It seems reasonable however, to assume that BMF values for seals are in the range of BMF values as given in the appendix.

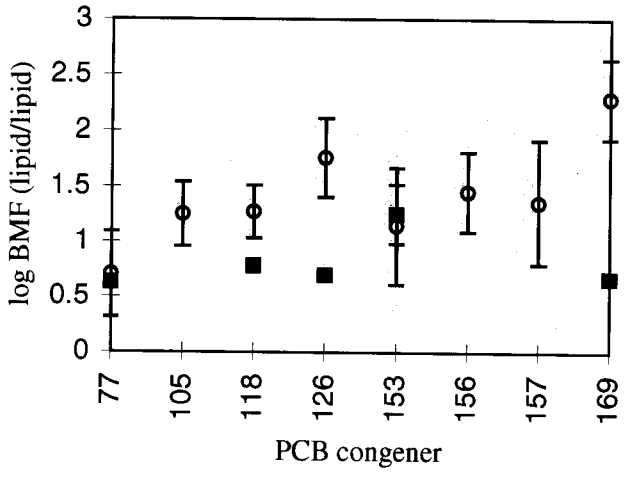

Figure 4.3. Biomagnification factors for the congeners of interest, in filled square the mean and std. of data from laboratory studies on fish species, in open circle the mean and std of data from field studies on mammal and bird species.

4.3.3. Biota-to-sediment accumulation

Data on biota-to-sediment accumulation factors (BSAF) derived from laboratory studies can be found in appendix II.c.. In most cases the bivalve *Macoma nasuta* is used as a test species. For PCB 153 also BSAF data for other species are found, but they do not differ from the *Macoma* data, tested by a two-sample T-test assuming equal variances.
BSAF-values derived from studies in the field are given in appendix II.f. Data on various species are obtained. No clear relationship is observed between type of species and the value of the BSAF.

For biota-to-soil accumulation little data are found, they do not seem to deviate from the BSAFs for sediment, however.
In Figure 4.4. the BSAF-values derived from laboratory and field studies are given for the separate congeners as well as averaged over all congeners. BSAF-values derived from field studies are higher than values from studies in the laboratory. This might be explained because the data from laboratory studies are not measured in a steady state and therefore the concentration in the organism is underestimated. Concentrations of PCBs in sediment and soil do not show clear temporal differences, therefore data from field studies will better approximate an equilibrium situation. A lower bioavailability due to ageing (Alexander, 1995; Belfroid et al., 1996) could be expected in the field, but is not reflected in the data.
An ANOVA on the field data yielded significant differences between the congeners. For performing the calculations as described in chapter 3, normal distributions are fitted to the field-derived BSAF data per congener. As the minimum limit of the distribution is bound at zero, a part of the left tail is truncated especially for congeners #153 and #157. For congeners #126 and #169, no standard deviation is available because of a lack of data. For these congeners the standard deviation is estimated based on the variation in the data for other congeners, as described by Luttik and Aldenberg (1997).
As in a normal distribution a significant part of the left tail is truncated for several congeners with an unrealistic high probability density at and near to zero as a result, as an alternative a lognormal distribution is fitted to the data.

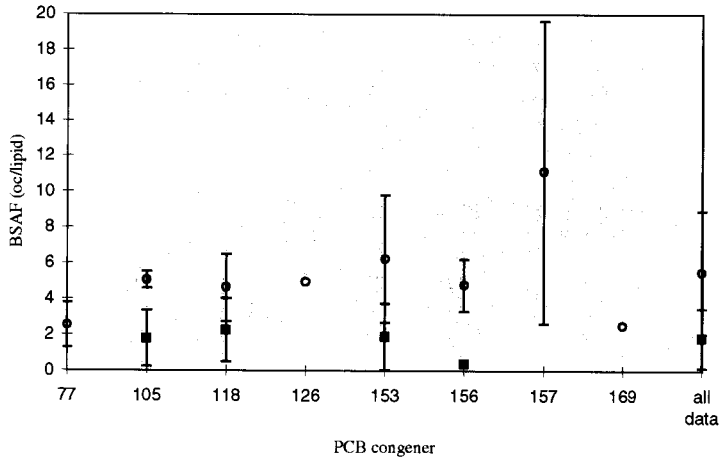

Figure 4.4. Biota to sediment accumulation factors for the congeners of interest, in black square the mean and std. of data from laboratory studies, in open circle the mean and std of data from field studies.

CHAPTER 5. MAXIMUM PERMISSIBLE CONCENTRATIONS FOR PCBS IN SEDIMENTS AND SOIL

The toxicity data and the distributions of environmental fate data and lipid content that are used in the calculation of MPCs are given in appendix IV. In appendix IV.a., those distributions are listed that are used for all congeners. These are the probability distributions of the lipid content of fish eggs, the lipid content of bird eggs, the lipid content of mammals, the bird egg-to-whole body ratio, and the bioconcentration factor. In appendix IV.b. to IV.i., congener specific information is given on the toxicity data as well as the probability distributions for BMF and BSAF that are used in the calculations of the MPC.
Figures 5.1. to 5.10. in this chapter depict the resulting probability distributions of equivalent concentrations in the organic carbon that are associated with adverse effects on survival, growth or reproduction. On the x-axis of each figure the logarithm of the EC in µg/kg organic carbon is given; on the y-axis the probability is given that this organic carbon EC has a certain value.

5.1. PCB 77

The MPC for 3,3'4,4'-tetrachlorobiphenyl is based upon 7 toxicity data, see appendix IV.b. for the details and for the congener specific distributions that are used.
The calculations are performed with two alternatives for the lipid content for reasons as described in 4.2., i.e. a normal or a uniform distribution. They do not give different results (Figure 5.1.). Also using a normal or a lognormal distribution for the BSAF (discussed in 4.3.3.) does not yield different results (Figure 5.2.). This increases the credibility of the calculated probability densities, as they are apparently not sensible to distributions that are not well-founded because of a lack on data or a wide variability in data.
To recalculate the toxicity data into equivalent concentrations in organic carbon in order to derive MPCs, normal distributions for the BSAF as well as for the lipid content are used.

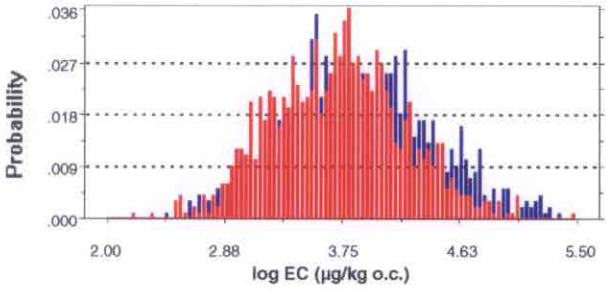

Figure 5.1. Probability distributions of sediment/soil concentrations (µg/kg o.c.) of PCB # 77 associated with no-observable effect on reproduction in the mouse. The blue distribution is based upon a normal distribution of the lipid content, while the red data are based upon a uniform distribution of the lipid content. In both calculations a lognormal distribution for BSAF was used. For specific characteristics of the distributions used it is referred to appendix IV.

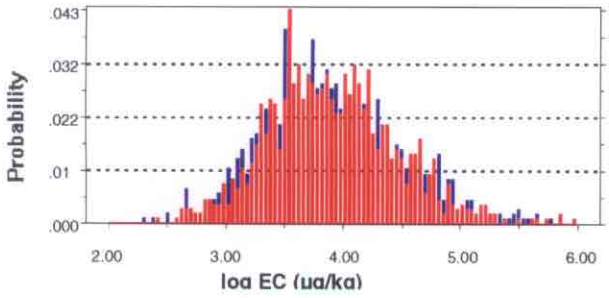

Figure 5.2. Probability distributions of sediment/soil concentrations (µg/kg o.c.) of PCB # 77 associated with no observable effect on reproduction in the mouse. The blue distribution is based upon a normal distribution of BSAF values, while the red one is based upon a lognormal distribution of BSAF values. In both calculations a normal distribution of the lipid content was used. For specific characteristics of the distributions used it is referred to appendix IV.

In Figure 5.3. the probability densities of toxicity of PCB 77 are depicted for different species. There is a wide variety in the sensitivity for different endpoints and species.

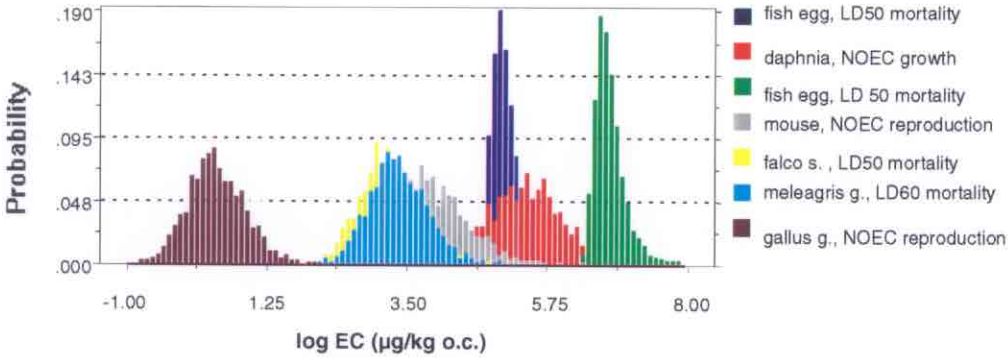

Figure 5.3. Probability distributions of organic carbon concentrations associated with critical levels of PCB #77 in different species.

5.2. PCB 105

The MPC for 2,3,3'4,4'-pentachlorobiphenyl is based upon 3 toxicity data, see appendix IV.c. for the details and for the congener specific probability distributions that are used (Figure 5.4.).

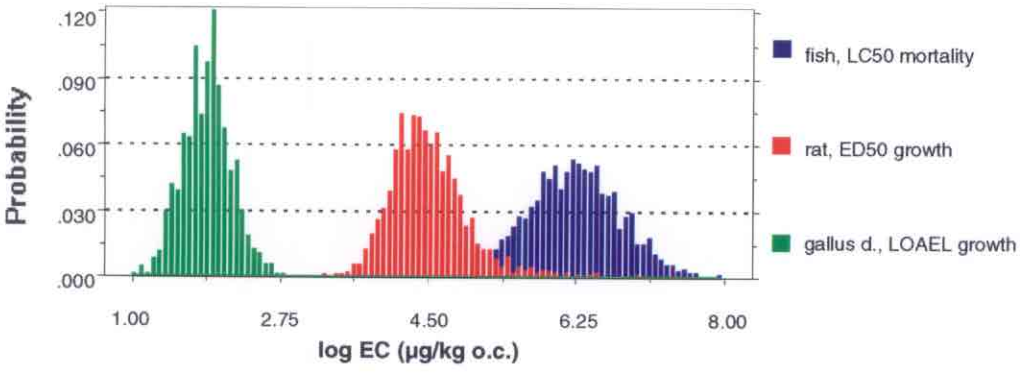

Figure 5.4. Probability distributions of organic carbon concentrations associated with critical levels of PCB #105 in different species.

5.3. PCB 118

The MPC for 2,3',4,4',5-pentachlorobiphenyl is based upon 2 toxicity data, see appendix IV.d. for the details and for the congener specific probability distributions that are used. The rat is more sensitive for PCB 118 than a bird (Figure 5.5.); the test endpoint for the bird study is embryo mortality while the endpoint for the rat is a NOEC for body weight.

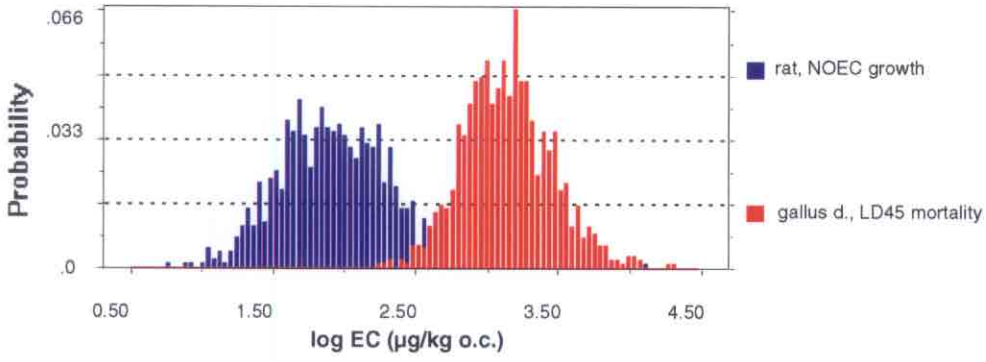

Figure 5.5. Probability distributions of organic carbon concentrations associated with critical levels of PCB #118 in rat and bird.

5.4. PCB 126

The MPC for 3,3',4,4',5-pentachlorobiphenyl is based upon 6 toxicity data, see appendix IV.e. for the details and for the congener specific distributions that are used. Again, there is a distinct difference in sensitivity between the aquatic species and the birds/mammals (Figure 5.6.).

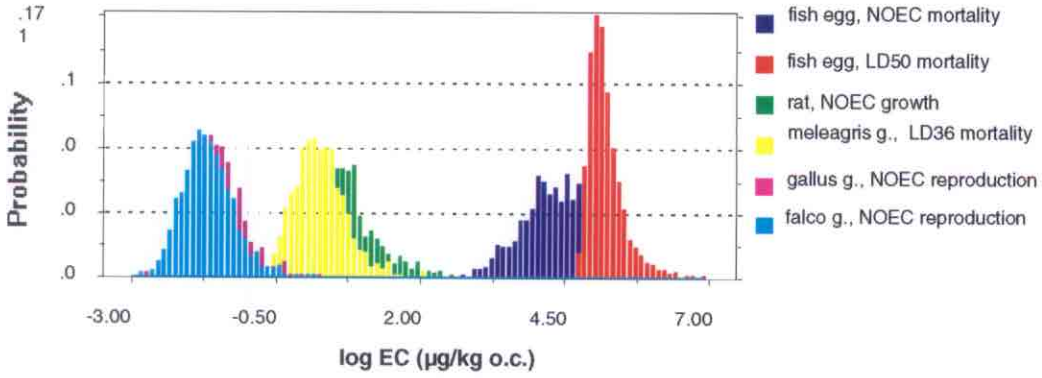

Figure 5.6. Probability distributions of organic carbon concentrations associated with critical levels of PCB #126 in different species.

5.5. PCB 153

The MPC for 2,2',4,4',5,5'-hexachlorobiphenyl is based upon 2 toxicity data, see appendix IV.f. for the details and for the congener specific distributions that are used in the calculations.
Because for the normal distribution of BSAF for PCB #153 a substantial part of the tail of the distribution is truncated (see appendix IV.f.), calculations with a lognormal distribution of the BSAF are also performed. As shown in Figure 5.7., the results do not differ much. The toxicity of PCB 153 to mink results in the lowest equivalent organic carbon concentrations.

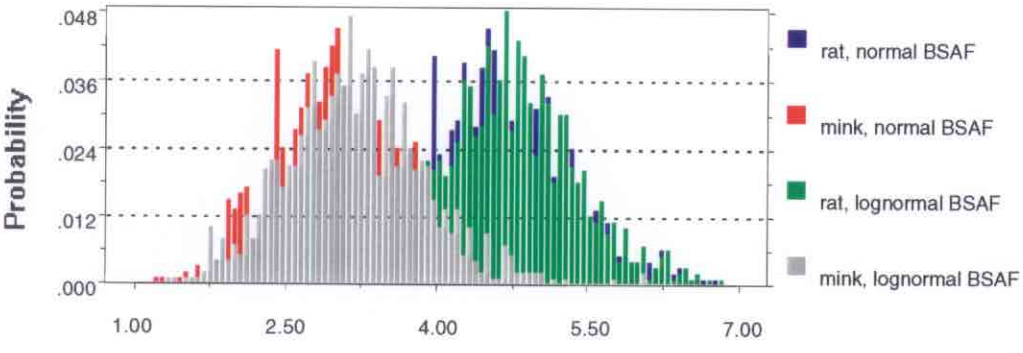

Figure 5.7. Probability distributions of organic carbon concentrations associated with critical levels of PCB #153 in different species. Normal as well as lognormal distributions for BSAF were used in the calculations. The probability distribution for mink and rat are both based a NOEC for growth (see also Table 4.1.).

5.6. PCB 156

The MPC for 2,3,3',4,4',5-hexachlorobiphenyl is based upon 3 toxicity data, see appendix IV.g. for the details and for the distributions that are used.

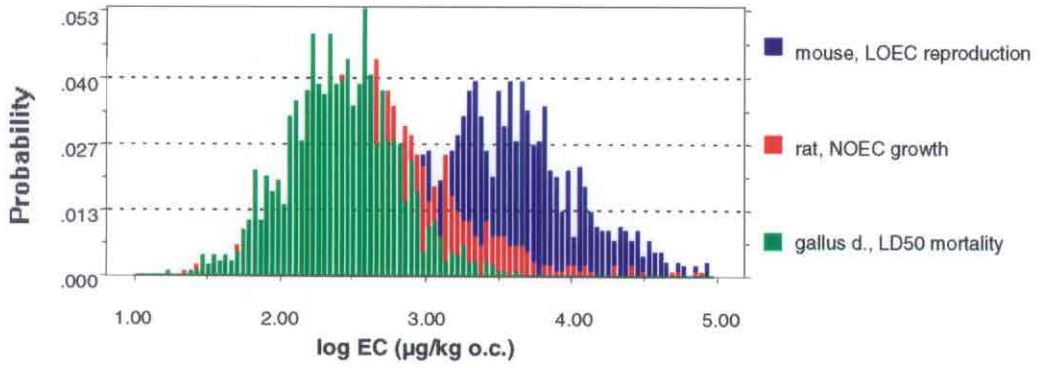

Figure 5.8. Probability distributions of organic carbon concentrations associated with critical levels of PCB #156 in different species.

5.7. PCB 157

The MPC for 2,3,3',4,4',5'-hexachlorobiphenyl is based upon 2 toxicity data, see appendix IV.h. for the details and for the congener specific distributions that are used. The distribution for the body weight gain of the rat appears to be less sensitive than that for embryo mortality of the bird (Figure 5.9.).

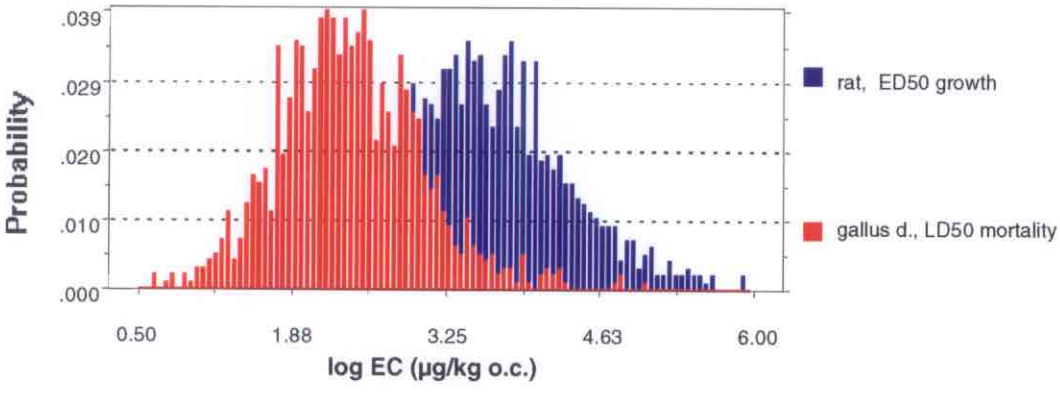

Figure 5.9. Probability distributions of organic carbon concentrations associated with critical levels of PCB #157 in rat and bird.

5.8. PCB 169

The MPC for 3,3',4,4',5,5'-hexachlorobiphenyl is based upon 5 toxicity data, see appendix IV.i. for the details and for the congener-specific distributions that are used. Because a significant part of the tail of the normal distribution of BSAF for PCB 169 was truncated (see appendix IV.i.), calculations with a lognormal distribution of BSAF values are also performed but again no different results are obtained (data not shown).

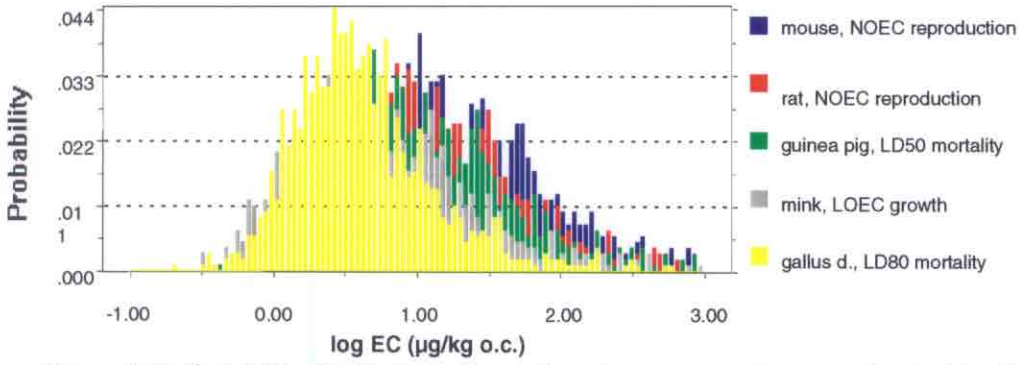

Figure 5.10. Probability distributions of organic carbon concentrations associated with critical levels of PCB #169 in different species.

5.9. Contribution of underlying parameters to the variance

The original toxicity data are recalculated to equivalent toxic concentrations in the organic carbon using equations 1 (aquatic organisms), 2 (fish egg injection studies), 4 (mammals) or 5 (bird egg injection studies) as given in section 3.4. An analysis is made of the contribution of the underlying parameters to the variance in the result of the calculation. The results are given in Table 5.1., only those parameters that contribute for over 10% to the variance are mentioned.
Several conclusions can be drawn, given the underlying distributions as used for the parameters in equations 1, 2, 4 and 5, that were fitted based on literature data. For the translation of aquatic toxicity data into equivalent effect concentrations in o.c. (equation 1), the parameter BCF is most important for the magnitude of the variance. For the fish egg injection studies (equation 2), BSAF is the main contributor to the variance. For the recalculation of effect concentration in the mammal into equivalent effect concentration in o.c. (equation 4), all the parameters used in the equation ($l.f._m$, BMF and BSAF) contribute to the variance. The importance of the various parameters varies per congener. For equation 5, which is used for the recalculation of effect concentrations in o.c. for birds, only BMF and BSAF are of importance for the resulting variance.

Table 5.1. Contribution of the underlying parameters to the variance in the resulting probability distribution of equivalent effect concentration in o.c.

PCB	Equation 1 Parameters: BCF, BSAF		Equation 2 Parameters: $l.f._{fish\ egg}$, BSAF		Equation 4 Parameters: $l.f._m$, BMF, BSAF		Equation 5 Parameters: $l.f._{bird\ egg}$, EBR, BMF, BSAF	
	% c.t.v.[a]	Parameter	% c.t.v.[a]	Parameter	% c.t.v.[a]	Parameter	% c.t.v.[a]	Parameter
77	80 12	BCF BSAF	94	BSAF	41 30 20	BMF $l.f._m$ BSAF	63 26	BMF BSAF
105	93	BCF			54 40	$l.f._m$ BMF	84	BMF
118					51 23 18	$l.f._m$ BMF BSAF	46 36	BMF BSAF
126	77 15	BCF BSAF	93	BSAF	34 30 28	$l.f._m$ BMF BSAF	47 42	BMF BSAF
153					62 22	BMF $l.f._m$		
156					42 41	BMF $l.f._m$	74 12	BMF BSAF
157					53 21 20	BMF BSAF $l.f._m$	68 24	BMF BSAF
169					42 31 19	BSAF $l.f._m$ BMF	58 31	BSAF BMF

[a]Contribution to the variance (in percentage)

5.10. Derivation of MPCs

As explained in section 3.6., the individual probability distributions are combined per congener and new distributions are fitted to these combined data to base the MPC upon.
Combined distributions are fitted to all toxicity data together, or only to the data on birds and mammals. The goodness-of-fit of the distributions is tested by the Kolgomorov-Smirnov test. If the s-value is lower than 0.1, the distribution is accepted to base the MPC upon. If not, the most sensitive probability distribution is chosen as a basis to derive the MPC.
Results of the Kolgomorov-Smirnov test are presented in Table 5.2. It can be seen that only for congeners #77, #105 and #126 fish toxicity data were available in the combined dataset. For most congeners (#118, #126, #153, #156, #157 and #169) the MPC is based upon the combined mammal and bird data, as the Kolgomorov-Smirnov test of goodness of fit yielded acceptable results. For congener #77, the goodness of fit to only the mammal and bird data was not acceptable, whereas the goodness of fit to all toxicity data including the data to fish was acceptable. For #105, the goodness of fit of the distributions over all combined toxicity data nor to the combined data on mammals and birds was acceptable. Therefore the MPC for congener #105 was based upon the most sensitive probability distribution.

Table 5.2. Goodness-of-fit of distributions to combined data (Kolgomorov-Smirnov test, distribution is accepted if s<0.1), and the distribution where the MPC is based upon

PCB	all toxicity data	mammal & bird data	MPC is based upon:
77	0.07	0.20	all toxicity data
105	0.16	0.21	most sensitive probability distribution
118	n.a.	0.09	mammal & bird data
126	0.20	0.07	mammal & bird data
153	n.a.	0.04	mammal & bird data
156	n.a.	0.07	mammal & bird data
157	n.a.	0.03	mammal & bird data
169	n.a.	0.05	mammal & bird data

n.a.: Not available as no toxicity data on aquatic organisms are available

Of the chosen distribution, the 5th percentiles are taken and backtransformed into µg/kg o.c., these values are set as MPC. The MPCs and the characteristics of the underlying distribution are presented in Table 5.3.

Table 5.3. The MPC (in µg/kg o.c.) per congener, and the characteristics of the underlying distribution (mean ± std of logarithmic data)

PCB	MPC (µg/kg o.c.)	characteristics of underlying distribution (mean ± std of logarithmic data)
77	7.2	4.04±1.93
105	26	1.87±0.28
118	25	2.57±0.72
126	0.042	0.07±0.88
153	151	3.86±1.03
156	55	2.87±0.69
157	32	3.00±0.92
169	0.83	0.98±0.65

CHAPTER 6. A MIXTURE-MPC FOR PCBS, AND THE WAY OF APPLICATION TO A FIELD SAMPLE

6.1. Introduction

In the foregoing chapters MPCs are derived for individual congeners. PCBs in the environment however always occur in a mixture. The planar PCBs are generally hypothesised to act via the same toxicological mechanism. So concentration-additivity can be assumed. If all these congeners occur in concentrations at or near their MPC, problems in the ecosystem can be expected.
In this chapter a so-called 'mixture-MPC' is derived, which is assumed to be protective for the whole mixture of planar PCBs. The mixture-MPC is expressed on the basis of PCB #118, as this congener is monitored routinely. In addition, an example of the use of this mixture-MPC is given.
For the derivation of the mixture-MPC both the congener pattern is used as the toxicological potency of the individual congener. The congener pattern gives information on the relative contribution of a congener to the total concentration of planar PCBs. Both the relative contribution of a congener to the total concentration of planar PCBs as the toxicological potency of each congener, determines the toxicity of the mixture of planar PCBs. The fraction of the toxicity of the mixture of planar PCBs explained by PCB #118 is calculated. The MPC for PCB #118 is multiplied by this fraction to yield the mixture-MPC.

6.2. Congener pattern in the Netherlands

To derive the mixture-MPC, information on the congener patterns of PCBs as they are found in the Netherlands is required. In Figure 6.1. an overview is given on PCB patterns in sediments from different areas in the Netherlands, normalised relative to the concentration of PCB 153 in that specific sediment. PCB 153 is chosen to normalise the concentrations upon, as this congener occurs in high concentrations, can be analysed with precision and is not metabolised. Note that the y-axis is on a logarithmic scale; there are big concentration differences between the congeners.
The most toxic congeners, #126 and #169, occur at the lowest concentrations (10^3-10^4 times less than PCB #153). There is seldom more than a 7-fold difference in #153-normalised concentrations between locations (except for congener #180, up to 23-fold).
As the planar PCBs occur in low concentrations, they are rarely measured. Therefore only four data-sets are available for the Netherlands to base Figure 6.1. upon. More data are available on concentrations of planar PCBs in organisms low in the food-chain. The congener patterns in the sediment should be reflected in organisms low in the food-chain with no or low metabolic capacities such as arthropods and molluscs. When the mean of data measured in various Dutch locations are compared between sediments, arthropods and molluscs, patterns are almost equal (see Figure 6.2.).
Based on these data it is concluded that PCB patterns are comparable for different

Dutch locations. This might be explained by the fact that the Netherlands can be considered as one sedimentation area (of the river Rhine), and that the contribution of atmospheric input to concentrations observed in the field is relatively high and can be considered evenly distributed over the Netherlands. Unfortunately, data on congener patterns in sediment influenced by the rivers Scheldt and Meuse are lacking for the congeners studied in this report. Also, information on congener patterns in the estuarine and marine waters is lacking. It is therefore assumed that the congener patterns as given in Figure 6.2. are also applicable to these types of sediments. Future measurements on these types of sediments might change the congener patterns used to derive the mixture-MPC.

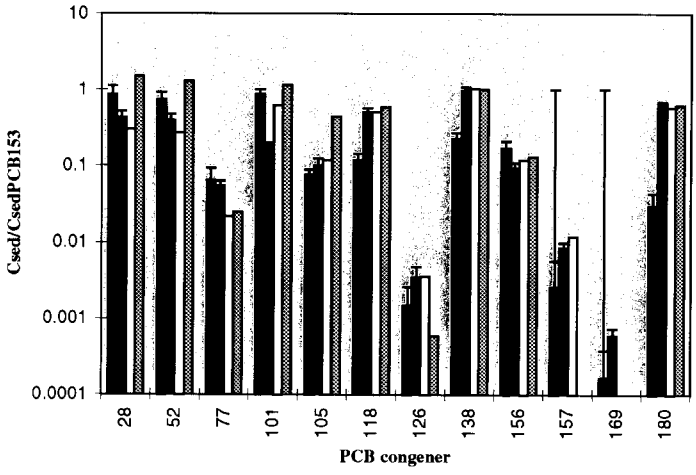

Figure 6.1.: PCB patterns in various Dutch sediments, normalised to the concentration of PCB 153. Data were kindly provided by dr. B. van Hattum (Hollandsch Diep and Bieschbosch, first two bars) and by dr. P. Leonards (Zandmeer, blank bar). The data for Ketelmeer (fourth bar) were obtained from Beurskens & Stortelder (1995) and Winkels (1993).

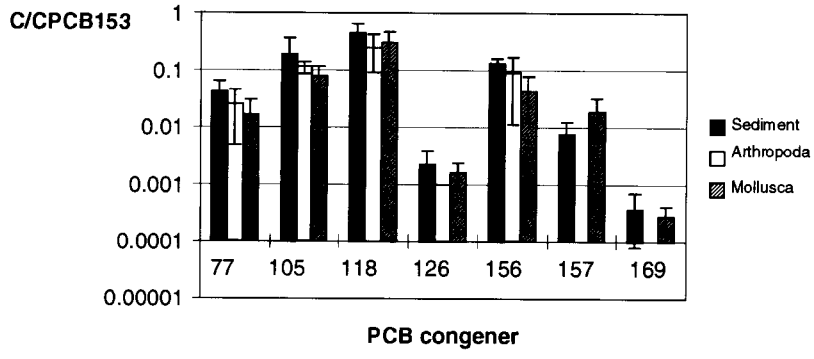

Figure 6.2.: PCB patterns in Dutch sediments, arthropods/plankters and molluscs, normalised to the concentration of PCB 153. Data for sediment are from figure 6.1., data for arthropods are from Leonards, P. et al., 1997a. ET&C 16:1807-1815 and Reinhold et al., submitted and are measured in lake 'Zandmeer' and the Biesbosch. Data for the molluscs are obtained from Hendriks et al. (1998) and Leonards et al. (1997), these data are measured in the rivers Rhine and Meuse, and in the lakes IJsselmeer and Zandmeer.

6.3. Derivation of a mixture-MPC

The mixture-MPC addresses the toxicity of the mixture of planar congeners considered in this report (#77, #105, #118, #126, #156, #167 and #169). The mixture-MPC is expressed in a concentration of PCB #118, as this congener is part of the standard monitoring set used in the Netherlands (see 1.2). The fraction of the toxicity of the mixture of planar PCBs explained by PCB #118 is calculated, and the MPC for PCB #118 is multiplied by this fraction to yield the mixture-MPC:

- The data as depicted in Figure 6.2. are averaged (second column in Table 6.1.), and expressed as percentage of the summed concentration of planar PCBs (third column).
- The mean of the distribution which was the basis for the MPC (Table 5.3.) was backtransformed (fourth column of Table 6.1.), and used as scaling factors to scale the toxicological importance of the fraction of that specific congener (see 2.2.4.). Means are used instead of MPCs, as they are not influenced by the dispersion in the underlying data whereas the MPCs (5^{th} percentiles) are.
- The fractions are divided by the scaling factor of that specific congener, and again these figures are expressed in such a way that the total is 1 (5th column in Table 6.1.).
- Then, the MPC for the single congener #118 (25 µg/kg o.c.) is multiplied with the fraction of the total toxicity that is explained by #118 (0.21); this yields the mixture-MPC. So, a concentration for #118 of 5 µg/kg o.c. as the mixture-MPC is considered protective for the total mixture of toxic PCB congeners (Table 6.1.).

Table 6.1. Different steps to derive a mixture-MPC that accounts for the total toxicity of the toxic PCBs (see text for further explanation).

PCB congener	fraction in pattern ($C_{sed}/C_{sed\#153}$*1000)	fraction in pattern (% of total concentration of planar PCBs)	Scaling factor	fraction/scaling factor expressed as %	mixture-MPC (µg/kg o.c.)
77	28±13	4.73	11*1000	0.06	
105	124±54	21.4	74	41	
118	327±94	56.2	370	21	5
126	1.6±0.6	0.28	1.2	34	
156	87±43	15.0	740	2.8	
157	13±8.0	2.29	1000	0.32	
169	0.33±0.07	0.057	9.5	0.85	

PCB-congeners other than listed in Table 6.1. are not taken into account. Multiple-ortho substituted congeners show CYP1A1 induction which points to AhR-binding, although with 1000-10000 higher EC_{50}s than PCB #126 (Van der Burght, 1997). Given their high environmental residues these multiple ortho-substituted congeners will probably contribute to total toxicity. Unfortunately information lacked to include these congeners in a mixture-MPC. Also other types of halogenated aromatics that will act via concentration addition to the planar PCBs, e.g. dioxins, are not incorporated in the mixture-MPC.

6.4. Application of a mixture-MPC

The mixture-MPC gives protection for the whole group of planar PCBs considered in this report (i.e. #77, #105, #118, #126, #156, #157 and #169). If the concentration of

PCB #118 in a field sample is lower than 5 µg/kg o.c., the ecosystem is assumed to be protected for the whole mixture of planar PCBs.

In the below, an example is given on the use of the mixture-MPC.
Suppose, the concentration of PCB #118 is determined in a sediment in the field on dry weight basis as $C_{s,d.w.}$. First, this concentration is normalised to organic carbon ($C_{s,o.c.}$):

$$C_{s,o.c.} = \frac{C_{s,d.w.}}{o.c.\%/100} \qquad \text{Equation 6}$$

in which o.c.% stands for the percentage of organic carbon. If only the percentage of organic matter is known (o.m.%), this can be recalculated into o.c.% by:

$$o.c.\% = 0.58 \cdot o.m.\% \qquad \text{Equation 7}$$

although the factor 0.58 (1/1.724) is under discussion (Smedes et al., 1997). If the organic matter content is higher than 30% or lower than 2%, or if the organic carbon content is higher than 18% or lower than 1.2%, the concentration is normalised to these percentages (IWINS, 1997).
Then, the $C_{s,o.c.}$ of PCB #118 can be compared to the mixture-MPC of 5 µg/kg o.c.. If the $C_{s,o.c.}$ does not exceed the mixture-MPC, we assume the ecosystem to be protected against adverse effects of the mixture of planar PCBs.

The current monitoring programmes as carried out by the Directorate-General for Public Works and Water Management do not have to be adjusted, as the mixture-MPC is expressed as PCB #118 which is currently already routinely measured. However, more measured data on the congener pattern of the planar PCBs are useful to strengthen the assumptions that are made to derive a mixture-MPC.
It should be noted that at a specific location the congener pattern of planar PCBs may deviate from the congener pattern used in the derivation of the mixture MPC. A mixture-MPC for that specific situation can then be calculated in a similar way as described in section 6.3.

The sediment concentration of PCB #153 can be normalised to organic carbon in a similar way, and this concentration can be compared with the MPC for the individual congener #153. PCB #153 is not included in the derivation of the mixture-MPC as it has a different toxicological working mechanism.

CHAPTER 7. COMPARISON OF THE MPC WITH EFFECTS FOUND IN THE FIELD AND WITH BIOCHEMICAL AND HISTOPATHOLOGICAL EFFECTS

7.1. Introduction

In this chapter the MPCs as derived in chapter 5 are compared with levels of PCBs that are associated with adverse effects in field studies.
Several studies have been performed in which effects as observed in wild-living organisms are related to the concentration of PCBs in these organisms. Mostly top-predators are studied; due to the bio-accumulative properties of PCBs the highest concentrations are found in these organisms. These studies are compared with the value of the MPCs derived in the foregoing. A drawback is that effects as observed in the field can only be related in a correlative way with concentrations of chemicals. Deriving a causal relationship between concentrations of (a group of) chemicals and an effect is impossible, as the total composition of the mixture of chemicals in the field is unknown and other substances in the mixture may have attributed to the effects observed. We focused on field studies from the Netherlands, as PCB congener patterns may be region-specific and with them the related effects. Region-specificity is due to the type of PCB-mixtures that have been in use in a certain region or river basin, the principal sources of PCB contamination, and to abiotic circumstances that influence rate constants of environmental fate processes (e.g. Ockenden et al., 1998).

In addition, the relative potencies of the different congeners based on toxicological effects (data used for the derivation of MPCs) are compared to the relative potencies based on biochemical and histopathological effect data. Ideally these relative potencies should be comparable for both types effects, as they are exerted via the same mechanism of action. For biochemical or histopathological effects raised by exposure to PCBs, more literature data are available than for effects on growth, reproduction and survival. In this way it is checked if the sometimes scanty dataset on toxic effects for a certain congener is a good spot check, where it is concerned the potency of that specific congener.

7.2. Effects in field-exposed fish-eating birds, otters and minks

7.2.1. Fish-eating birds

Fish-eating birds are top predators at the end of the aquatic food chain, and are known to accumulate high concentrations of PCBs and related chemicals (Bosveld & Van den Berg, 1994).
In a study described by Bosveld et al. (1995), eggs from the common tern were taken from seven colonies in the Netherlands. Concentrations of PCBs (including the planar PCBs), PCDDs and PCDFs were analysed in the yolk, and the eggs were artificially incubated. Murk et al. (1994) reported on the effects on biochemical parameters. At the level of the individual birds, a concentration-response relationship was observed with incubation period as an endpoint. If the concentration of PCBs was higher or equal to 3.5 ng TEQ/g lipid (Chicken TEFs were used, see Bosveld et al. 1995) a significant longer incubation period was observed. It was shown that a longer incubation period in the laboratory also means a longer incubation period in the field

(Murk et al., 1996). A longer incubation period is suggested to reduce the reproduction success because of an increased risk on egg predation etc.. Murk et al. (1996) also showed that at the most contaminated colony, with a total TEQ of 14.1±5.6 ng TEQ/g lipid(calculated with TEFs from Safe, 1990) there was a reduction in chicken weight. The study by Bosveld, showing a sensitive and relevant effect to population growth, was used to validate the MPCs derived in the present report (see 7.2.3.).

7.2.2. Otters and minks

Leonards et al. (1995) derived an EC50 for PCBs exerting effects on reproduction in mink, based on a series of literature data. The literature data were based upon laboratory feeding studies with a duration of 35-400 days. Results were expressed as TEQ with help of the TEFs as derived by Safe (1994), and based upon whole body concentrations. A one-compartment bioaccumulation model was used with congener specific bioaccumulation. An EC50 for litter size of 0.16 ng TEQ/g w.w., and for kit survival of 0.2 ng TEQ/g w.w. was proposed. The dose-response curves were steep, only one or two orders of magnitude where partial effects were observed. Based on lipid, EC50s are 5-10 ng TEQ/g lipid.

Leonards et al. (1996) showed that above a level of 4 ng Safe-TEQ/g lipid disease incidences are increased in Danish otters.

7.2.3. Conclusions based upon the field studies with respect to the derived MPCs

Bosveld et al. (1995) concluded that at 3.5 ng chicken-TEQ/g lipid a significant longer incubation period before hatching was observed. The MPCs for the individual congeners, as given in Table 5.3., are recalculated into concentrations in the lipid of the egg. The following formula is used:

$$C_{egg} = MPC_{o.c.} \cdot EBR \cdot BMF \cdot BSAF \qquad \text{Equation 8}$$

The resulting concentrations in the eggs are reformulated in TEQs and summed. Chicken-TEFs are used therefore. The mean values as derived in this report for EBR, BMF and BSAF were used in the calculation (given in appendix IV). If concentrations for PCB congeners #77, #105, #118, #126, #156, #157 and #169 in the sediment are all equal to the MPC, this result in a chicken-TEQ of 1.9 ng/g lipid in the egg. This is approximately a factor or two below the lowest observed effect concentration as observed in the study by Bosveld et al. (1995).

In analogy, the resulting Safe-TEQ in the lipid of mink or otter resulting from concentrations for all planar congeners on MPC level can be calculated via:

$$C_{mammal,lipid} = MPC_{o.c.} \cdot BMF \cdot BSAF \qquad \text{Equation 9}$$

If concentrations for PCB congeners #77, #105, #118, #126, #156, #157 and #169 in the sediment are all equal to the MPC, this results in a Safe-TEQ of 34 ng/g lipid. This is approximately 7 times above critical levels as deduced from mixture and field studies by Leonards (1995, 1996).

It is concluded that the concentrations expressed in TEQ that are associated with adverse effects in field studies, are comparable with the TEQ concentrations that would result if all planar congeners would be present on MPC-level.

7.3. Potency-rating of the different congeners; comparison of toxicological effects with biochemical and histopathological effects

The relative potencies of the different congeners based on toxicological effects (data used for the derivation of MPCs) are compared to the relative potencies based on biochemical and histopathological effect data. As both type of effects are believed to be exerted via the same mechanism of action these relative potencies should be comparable for both types effects.
For biochemical or histopathological effects raised by exposure to PCBs, more literature data are available than for effects on growth, reproduction and survival. Applying the same methods as described for the derivation of MPCs, also critical levels can be derived that are associated with biochemical and histopathological effects of PCB congeners. If effect levels relative to PCB #126 for the individual congeners are comparable for toxicological and for biochemical/histopathological effects, this supports the idea that the underlying datasets are good descriptors of the effects and thus supports the validity of the derived MPCs.
The studies that are used for this exercise are given in *italics* in appendix 1. The types of effects used are e.g. splenic immunosupression in the mouse, EROD activity in the rat or mouse, free thyroxin levels in the monkey or rat pups, liver cell abnormalities in the rat etc.. An example the results (for #77) is given in Figure 7.1, where equivalent effect concentrations in the organic carbon that exert a variety of biochemical and histopathological effects of congener #77 are depicted. For the other congeners, this information is given in appendix VI.

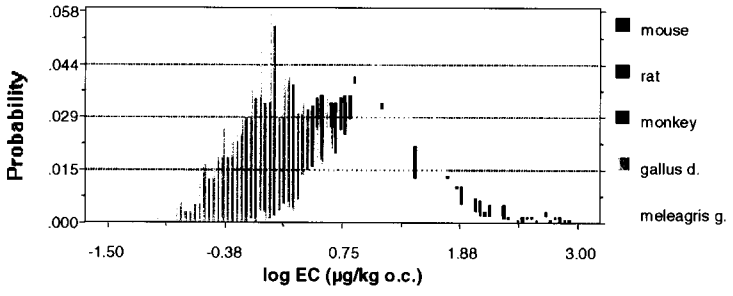

Figure 7.1. : Probability distributions of organic carbon concentrations associated with levels of PCB #77 in mammals and birds that exert biochemical or histopathological effects. These concentrations in the organic carbon are equal to an EC50 in mouse for splenic immunosupression, to a NOEC of EROD induction in the rat, to a NOEC in free thyroxin index in monkey, and to an ED50 of AHH activity in both birds.

Statistics of the combined distributions are given in Table 7.1. Means of the distribution for biochemical/histopathological effects are 1.5-3 log units lower than means of the distributions for toxicological effects where the MPC was based upon (Table 5.3). An exception is congener #157 where the difference is 3.7 log units, and congener #105 where there is no difference is zero. The latter might be explained by the fact that the distribution for the MPC for congener # 105 is based upon the most sensitive probability distribution.

Table 7.1.: Characteristics of the combined distribution over the probability distributions for biochemical and histopathological effects in mammals and birds per congener.

PCB congener nr.	Characteristics of the normal distribution of all log-transformed data (mean ± std)
77	0.76±0.64
105	1.85±2.02
118	-0.01±1.44
126	-2.89±0.82
153	2.34±0.88
156	0.42±1.19
157	-0.76±0.65
169	-1.25±1.17

For a comparison of the relative effect levels normalised to PCB #126 see Table 7.2. The effect levels relative to PCB #126 are comparable for the backtransformed means of the distributions for toxicological and biochemical/histopathological effects (less than 10 times difference between both columns in Table 7.2.); an exception is congener #105 where a difference can be observed of a factor 1000. Again, this might be explained by the fact that the MPC for this congener is based upon the most sensitive individual probability distribution.
In general, this leads to the conclusion although for some congeners little data on toxicity are available, the dataset is a good predictor of the toxicity to other species.

Table 7.2. The effect concentrations (mean of probability distribution) in organic carbon, relative to that of PCB #126 for distributions based upon toxicity data or upon biochemical/histopathological effect data for birds and mammals.

Congener	Normalised means of distribution for toxicological effect	Normalised means of distribution for biochemical/ histopathological effect
77	0.00001	0.00002
105	0.002	0.000002
118	0.0003	0.0001
126	0.1	0.1
156	0.00002	0.00005
157	0.0001	0.0007
169	0.01	0.002

CHAPTER 8. DISCUSSION AND CONCLUSIONS

8.1. Comparison with currently used environmental quality standards for PCBs in the Netherlands

The MPCs and NCs as derived in this report (see Table 8.1.) are meant to replace the environmental quality standards for PCBs that are currently in use.

Table 8.1. MPCs derived in this report

PCB	MPC (µg/kg o.c.)	NC (µg/kg o.c.)
77	7.2	0.072
105	26	0.26
118	25	0.25
126	0.042	0.00042
153	151	1.51
156	55	0.55
157	32	0.32
169	0.83	0.0083
mixture-MPC planar PCBs	5	0.05

The PCB quality standard that is currently in use in the Netherlands is 4 µg/kg d.m., in 'standard sediment' with 10% o.m. and 25% lutum for each of the congeners #28, #51, #101, #118, #138, #153 and #180 (IWINS, 1997). This equals 40 µg/kg o.m. or 70 µg/kg o.c. In the current system (IWINS, 1997) there is no mixture-MPC available. For #118 and #153, new and old MPCs can be compared; their ratio is respectively 0.35 and 1.5.
Dredge material can be dispersed if the concentration of one of the above mentioned individual PCB congeners is below 0.03 mg/kg d.w. (10% o.m., 25% lutum; equals 0.5 mg/kg o.c.), and if the total concentration of these 7 PCBs is below 0.2 mg/kg d.w. (10% o.m., 25% lutum; equals 3.5 mg/kg o.c.) (Van de Guchte, 1998).

8.2. Comparison with previously used method for compounds with a potential for secondary poisoning

For comparison, the MPCs for the individual congeners are also derived following the method as used earlier in the project 'Setting integrated environmental quality standards', which is described in the report 'Towards integrated environmental quality objectives for several compounds with a potential for secondary poisoning' by Van de Plassche (1994). Motivation for the deviation from this method is given earlier, and will be discussed in the next section.
In the method as described by van der Plassche (1994), a NOEC derived for birds and mammals by using extrapolation methods, is divided by the BCF (ratio of concentration in the organism and concentration in the water at steady state) for mussels or fish (aquatic route) to obtain the MPC:

$$MPC_{water} = NOEC_{bird/mammal}/BCF_{fish} \qquad \text{Equation 10}$$

For the terrestrial route, the NOEC is derived by the BSAF for earthworms (in the report by Van de Plassche, it is referred to BCF for worms, but the text and QSARs used clarify that the concentration ratio between worm and soil is meant):

$$MPC_{soil}=NOEC_{bird/mammal}/BSAF_{worm} \qquad \text{Equation 11}$$

The MPC for direct effects is compared to the MPC for effects due to secondary poisoning, and the most critical one is chosen.
At the time the report by van de Plassche was written, there was much discussion on the methods (see Health Council, 1993; TCB, 1994) . This discussion focused on the sequence in which bioaccumulation and extrapolation of the toxicity data should be incorporated in the calculation of the MPC (see Figure 8.1.). In addition there was discussion on the correction factors that should be applied to laboratory studies, which are used to predict field effects (differences in caloric content of food). For details on this discussion, it is referred to van de Plassche (1994). In the report, method I was chosen to derive MPCs.

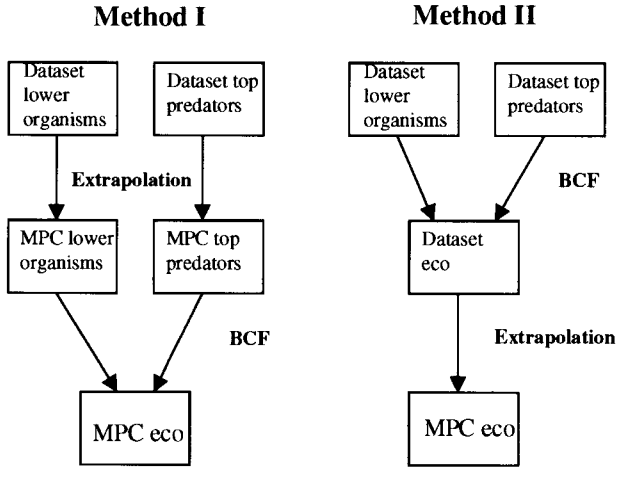

Figure 8.1. Two methods for incorporating effects of secondary poisoning in the derivation of MPCs as elaborated by van de Plassche (1994)

For comparison, we also apply this method (I) to our dataset. Only NOECs derived from diet studies, expressed as mg/kg food, are to be used. The BCF/BSAFs are applied to the source of food (fish/mussel/earthworm), see equations 10 and 11. An MPC for water was calculated by following equation 10. Following the modified EPA-method, an assessment factor of 10 was applied (Van de Plassche, 1994). This factor is applied when less than 3 chronic NOECs are available. It should be mentioned that the assessment factors used in the report by Van der Plassche differ from the assessment factors mentioned in the 'Technical Guidance Document' from the EU that will be used in the future in the project 'Setting Integrated Environmental Quality Standards'. As can be seen in Table 8.2., only one NOEC-value was available per congener. This value has to be compared with extrapolated LC50 values. However, as can be seen in appendix I.D. and I.E., we did not have acute values based on diet studies at our disposal. The MPC_{water} was recalculated into $MPC_{o.c.}$ via equilibrium partitioning (EP), using geometric means of the organic carbon

normalised partition coefficients from soil or sediment to water (data derived from AQUAPOL database, see appendix III.C.).
It may be noted that the values calculated in this manner are 3.5 to 40 times higher than the MPCs derived following the methods as described in the present report (see Table 8.1.), and that the results would be different if another system of assessment factors was used (EU versus modified EPA).

Table 8.2. Calculation of MPC_{water} and $MPC_{o.c.}$ following the methods as described in van de Plassche 1994.

PCB	endpoint	para-meter	species	value (mg/kg food)	ref	log $BCF_{fish,w.w.}$ (see 4.3.1.)	MPC_{water} (ng/l)	log K_{oc} (in l/kg o.c.)	$MPC_{o.c.}$ via EP (µg/kg o.c.)
PCB 118	NOEC	growth	rat	1	Chu et al., 1995	5.18	0.21	6.61	861
PCB126	NOEC	growth	rat	0.01	Chu et al., 1994	5.18	0.002	5.92	1.76
PCB153	NOEC	growth	mink	2.5	Aulerich et al., 1985	5.18	0.53	6.48	1596
PCB156	NOEC	growth	rat	1.2	Van Birgelen et al., 1994	5.18	0.25	6.45[a]	715
PCB169	LOEC/3	growth	mink	0.017	Aulerich et al., 1987	5.18	0.004	5.92	2.93

[a]As no data for this specific congener were available in appendix III.C., this is a geometric mean over all data

8.3. Discussion on the methods used

Part of the methods as used in this report, are new in the context of the project 'Setting Integrated Environmental Quality Standards'.
The procedure used to derive MPCs for PCBs deviates on certain points from the procedures as earlier used and described in CSR (1996), Van de Plassche (1994) and Crommentuijn et al. (in prep.). The adjustments are summarised and discussed in the below.

8.3.1. Inclusion of different dosing methods

For toxicity studies on mammals, studies with dosing per diet, gavage as well as per injection are evaluated and included when relevant. For birds, egg injection studies are included in the derivation of MPCs. For fish, studies with dosing directly to eggs are included when relevant.
For toxicity studies on mammals and birds, diet studies are considered the most reliable. Other and more often used dosing methods include dosing by gavage or per injection. The inclusion of these data is evaluated by comparing the results of the different types of studies.
For all types of studies where organisms are directly dosed, i.e. per injection or gavage, it is assumed that 100% of the administered dose is taken up and distributed evenly over the lipids. For assimilation efficiency in diet studies, a value of roughly 50% is often mentioned in the literature. For studies where dosing takes place via gavage or injection, less information is available. However, assuming 100% availability of the administered dose will not be a gross overestimation.
For studies per diet, the whole-body concentration is estimated using a one-compartment bioaccumulation model.
The question if different dosing methods influence the results cannot be answered for the bird toxicity studies, as all studies available for the extrapolation into MPCs are based on egg injection studies. Injection takes place into the yolk or the air chamber of the eggs. Contaminants first have to pass membranes, and then the transport of the PCBs will probably mainly occur via the circulation of the developing embryo. It is

assumed in our calculations that the PCBs that are injected, partition over the egg (mainly the lipids) and that equilibrium is reached relatively soon after the exposure. The duration of these egg injection studies is in general from 4 to 7 day old eggs until hatching (see appendix I.E. for details). In a study by Näf et al. (1992) is was shown for PAHs that were injected into the yolk on day 4, that at day 18 94% of the PAHs was metabolised. This implicates that the PAHs are available for uptake within this period, as metabolisation will take place in the developing embryo.
For mammals, for these congeners where multiple mammal studies are available (#153, #156, #169) the sensitivities between the different types of studies vary maximal 1.5 log unit (means of probability distributions are compared). Therefore, as far as the available data allow conclusions to be drawn on, the dosing method does not seem to influence the results for a great deal.
Concerning fish, results from different dosing methods for the same congener are available for #77 and #126. The results obtained by fish egg injection are more sensitive in the case of #77, and the opposite is true for #126. Therefore no conclusion is drawn on the influence of the dosing method upon the result from the toxicity study.

8.3.2. Inclusion of several endpoints and several levels of effect
Effects on growth, reproduction, or mortality are integrated in the derivation of MPCs.
For each species and for each compound the most sensitive toxicity test was selected. Not only NOEC values are included but also other percentages of effect such as EC50s. This is evaluated in the below, by studying the steepness of the dose-response curves, and the difference in sensitivity for several endpoints.
We examined several studies in which different levels of effects were reported upon the steepness of the dose-reponse curve (Powell et al., 1996; Brunström, 1989 and 1991; Marks et al., 1981). The steepness of the curve has to be examined within a study, as otherwise differences in sensitivities of the laboratory animals and interlaboratory differences in the experimental set-up obscures the results.
Powell et al. (1996) showed for PCB #77, #105 and #126 that there was a factor of 0.7 to 18 between the LD50 for mortality and the LOAEL for growth in chickens. Brunström (1989) found in their study a factor of 3 between the concentration exerting 36% or 100% mortality in Meleagris g. for congener #126. Brunström (1991) showed that there was a factor of 5 difference in the concentration needed to exert 17% or 100% lethality in chicken embryos exposed *in ovo* to PCB #77. Marks et al. (1981) showed that a factor of 8 increase in dose resulted in the difference between 10% and 60% response for the endpoint reproduction in mice after exposure to congener #169.
These results show that dose-response curves for individual congeners are steep (Brunström, 1989 and 1991; Marks et al., 1981), and that the differences in the concentrations needed to obtain effects on different types of endpoints (mortality, growth) are not too large (Powell et al., 1996). The differences in sensitivity between the species are large (see Figures 5.3 to 5.10), relative to the aforementioned differences within a species between different endpoints or percentages of effects. Therefore, it is believed that the inclusion of diverse endpoints and diverse levels of effects into the extrapolation of an MPCs is acceptable.
An alternative of using different levels of effect in the derivation of one MPC is recalculating LOEC values into NOEC values by applying standard factors. The

standard factors applied within the context of the project 'Setting integrated environmental quality objectives' are (e.g. Van de Plassche, 1994):

1. 10% effect < LOEC<20% effect: NOEC=LOEC/2
2. LOEC≥20% effect, and a distinct concentration-effect relationship; EC10 is calculated and set equal to NOEC
3. LOEC≥20% effect, and no distinct concentration-effect relationship;
   - 20% effect≤LOEC<50% effect: NOEC=LOEC/3
   - LOEC≥50% effect: LOEC/10

However, by applying these standard factors, implicitly an assumption is made on the steepness of the dose-response curve.

8.3.3. Sensitivity of different organism groups

The sensitivity of different groups of organisms (aquatic species, birds and mammals) can be compared directly, as the equivalent effect concentrations are expressed in a comparable unit (i.e. µg/kg o.c.). Although different endpoints, different levels of effect and studies with different dosing methods are combined in the derivation of the MPCs, still patterns is sensitivity of different species groups can be discerned.
For those congeners where information on aquatic species is available (#77, #105, #126), the aquatic organisms (fish and daphnia) are without exception the least sensitive. Avian NOECs on growth or reproduction, expressed as equivalent (no)effect concentrations in the organic carbon, turns out to be the most sensitive parameter (see #77, #105, #126). For congeners #118, #153, #156, #157 and #169, information on avian NOECs for growth or reproduction is lacking, which may result in an underestimation of the MPC. New studies on these endpoints in birds might change the value of the MPC.
The amount of underlying toxicity data for the derivation of the MPCs for the individual congeners varies between two and seven (Table 4.1.). When just two underlying toxicity data are available, the data are on the relative sensitive mammals and/or birds.

8.3.4. Methods for including the risk for secondary poisoning

Toxicity data from studies on aquatic organisms, mammals and birds are recalculated into equivalent toxic concentrations in sediment or soil. Bioconcentration factors, biota-to-sediment concentration factors and biomagnification factors are used in these calculations. In this way all types of studies can be readily compared on one and the same concentration axis, and can be integrated into one MPC. The procedure used deviates from previously used methods to incorporate risks for compounds with a potential for secondary poisoning (as described by Van der Plassche, 1994). We used data from the field for processes as biota-to-sediment-accumulation and biomagnification. In the laboratory, the establishment of a steady state is hard to reach, and it is difficult to simulate all the possible pathways of intake of the contaminants experimentally. Parameters which are difficult to determine for these hydrophobic compounds (BCF and K_{oc}) were avoided where possible. Thereby, we met the criticism of the Health Council (1996) on the use of these parameters in the derivation of MPCs for compounds with a potential for secondary poisoning.
The advantage of using BCF/BSAF instead of the organic carbon normalised sediment/water or soil/water partition coefficient (K_{oc}) is also that possible biotransformation of the compound can be taken into account.

It should be mentioned that the food chains taken into account for this report are relatively simple ones, which are sufficient for the situation in the Netherlands (see Hendriks, 1995). However, there are ecosystems where food-chains have more levels and for that ecosystems the methods used in this study should be extended with multiple steps in the food-chain (Cabana & Rasmussen, 1994).
To be able to use the methods as sketched in this report, a prerequisite is a broad data availability on toxicity as well as environmental behaviour of the compound studied.

8.3.5. Derivation of mixture-MPC
Information of the toxicity and environmental chemistry of the individual congeners was used to derive MPCs, and as a second step a mixture-MPC was derived based on this information. The use of 'TEFs' was avoided in this way, in order to prevent the building-in of circular arguments in the derivation of a (mixture-)MPC as TEFs on their part are also based on toxicity information.

8.3.6. Probabilistic modelling
The variability in the parameters related to environmental fate, which are used in the calculations, is taken into account. Distributions are fitted based on the information gained from the literature, and these distributions are used in the calculations instead of absolute values. Considerable spread in literature data on these parameters can be encountered, because of intra- and interspecies differences, sediment differences, different test methods that were used etc..
Use of probabilistic modelling is an addition to the procedures normally used in the project 'Setting Integrated Environmental Quality Standards'; normally calculations are performed with a mean value or a best estimate.

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Appendix I. Chronic and acute toxicity data

Data that are used for deriving the MPC are underlined, data that are used to derive critical levels in organic carbon related to histopathological or biochemical effects are given in italics.

I.a. Acute toxicity of PCB congeners to aquatic organisms

Data that are used for deriving the MPC are underlined, data that are used to derive critical levels in organic carbon related to histopathological or biochemical effects are given in italics.

Organism	Test sub puity	Test water	pH	Hardness in mg CaCOs/l	Exp. time	Criterion	Result µg/L	Reference
PCB 153								
Daphnia magna, <24 h	99%	tap	7.6-8.3	118-158	48 h	NOEC	>1.3a	Dillon & Burton, 1991
Pimephales promelas, larva <24 h	99%	tap	7.6-8.3	118-158	96 h	NOEC	>1.3a	Dillon & Burton, 1991
PCB 77								
Daphnia magna, <24 h	99%	tap	7.6-8.3	118-158	48 h	NOEC	>0.3a	Dillon & Burton, 1991
Pimephales promelas, larva <24 h	99%	tap	7.6-8.3	118-158	96 h	NOEC	>0.3a	Dillon & Burton, 1991
Salvelinus namaycush, eggs	>97%	-	-	-	48 h	LD50	**29b**	Zabel et al., 1995
PCB 105								
Zebra fish, larvae	>99%				168 h	LC50	6.53	Petersen et al. (subm.)
Zebra fish, larvae	>99%				168 h	EC50b	**1.3**	Petersen et al. (subm.)

a only 1 testconc: saturation; no mortality occurred
b LD50 in µg/kg egg; renewal every 12 h; acetone 1.5 ml/l; waterconcs. not reported

References, appendix 1a

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Zabel, E.W., P.M. Cook, and R.E. Peterson (1995) Potency of 3,3',4,4',5-pentachlorobiphenyl (PCB 126), alone and in combination with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), to produce lake trout early life-stage mortality. Environ. Toxicol. Chem., 14, 2175-2179.

I.b. Chronic toxicity of PCB congeners to aquatic organisms

Data that are used for deriving the MPC are underlined, data that are used to derive critical levels in organic carbon related to histopathological or biochemical effects are given in italics.

Organism	Test type	Test sub. purity	Test water	pH	Hardness in mg CaCO3/l	Exp. time	Criterion	Result µg/l	Reference
PCB 118									
Daphnia magna, < 24 h	R	99%	rw	8.3	170	21 d	NOEC	>1	Dillon et al. 1990
PCB 153									
Daphnia magna, < 24 h	R	99%	rw	8.3	170	21 d	NOEC	>1	Dillon et al. 1990
PCB 77									
Daphnia magna, < 24 h	R	99%	rw	8.3	170	21 d	NOECa	**0.1**	Dillon et al. 1990
Hyalella azteca, 0-1 w	R	-	tap		130	10 w	NOECb	>3200	Borgmann et al. 1990
Oryzias latipes, 1-2 h eggs	S	>99%	am	-	-	17 d	LC50	>250	Harris et al., 1994
Oryzias latipes, 1-2 h eggs	S	>99%	am	-	-	17 d	NOECc	>250	Harris et al., 1994
Oryzias latipes, 1-2 h eggs	S	>99%	am	-	-	17 d	EC50d	>250	Harris et al., 1994
Oryzias latipes, 1-2 h eggs	S	>99%	am	-	-	17 d	NOECd	125	Harris et al., 1994
PCB 126									
Oryzias latipes, 1-2 h eggs	S	>99%	am	-	-	17 d	LC50	0.22	Harris et al., 1994
Oryzias latipes, 1-2 h eggs	S	>99%	am	-	-	17 d	NOECce	**0.018**	Harris et al., 1994
Oryzias latipes, 1-2 h eggs	S	>99%	am	-	-	17 d	EC50d	0.18	Harris et al., 1994
Oryzias latipes, 1-2 h eggs	S	>99%	am	-	-	17 d	NOECde	0.055	Harris et al., 1994

a growth, only 2 concs. of 0.1 and 1 µg/l tested, with methanol 0.01 and 0.1 µg/l, resp.
b mortality, reproduction and growth; DMSO up to 4 ml/l
c mortality
d swim bladder inflation
e NOEC calculated as EC10 from effect concs

References, appendix 1b.

Borgmann, U., W.P. Norwood, and K.M. Ralph (1990) Chronic toxicity and bioaccumulation of 2,5,2',5'- and 3,4,3',4'-tetrachlorobiphenyl and Aroclor 1242 in the amphipod Hyalella azteca. Arch. Environ. Contam. Toxicol., 19, 558-564.

Dillon, T.M., W.H. Benson, A. Stackhouse, and A.M. Crider (1990) Effects of selected PCB congeners on survival, growth, and reproduction in Daphnia magna. Environ. Toxicol. Chem., 9, 1317-132Harris, G.E., Y. Kiparissis, and C.D. Metcalfe (1994) Assessment of the toxic potential of PCB congener 81 (3,4,4',5-tetrachlorobiphenyl) to fish in relation to other non-ortho-substituted PCB congeners. Environ. Toxicol. Chem., 13, 1405-1413.

I.c. Toxicity of PCB congeners to aquatic organisms in injection experiments

Data that are used for deriving the MPC are underlined, data that are used to derive critical levels in organic carbon related to histopathological or biochemical effects are given in italics.

Organism	Purity	Affected stage or parameter	Criterion	Result, per kg w.w.	Reference
PCB 118					
Cyprinus carpio, 54 g	>99%	EROD	ED50	3.948 µmol/kg	Van der Weiden et al., 1994
Cyprinus carpio, 54 g	>99%	P450 1A	ED50	11.95 µmol/kg	Van der Weiden et al., 1994
Oncorhynchus mykiss, eggs	>99%	sac fry	LD50	>6970 ng/g egg	Walker & Peterson, 1991
Oncorhynchus mykiss, 150-270 g	>99%	EROD, AHH	EDa	30 mg/kg	Skaare et al., 1991
PCB 153					
Oncorhynchus mykiss, eggs	>99%	sac fry	LD50	>6200 ng/g egg	Walker & Peterson, 1991
PCB 77					
Morone saxatilis, adult	NR	sex hormones	NoED	3 x 1 mg/kg	Monosson et al., 1996
Oncorhynchus mykiss, eggs	>99%	sac fry	LD50	**1348 ng/g egg**	Walker & Peterson, 1991
Oncorhynchus mykiss, 300-400 g	NR	AHH ind.	ED50	2.2 µmol/kg	Janz & Metcalfe, 1991
PCB 126					
Cyprinus carpio, 54 g	>99%	EROD	ED50	<2.84 µmol/kg	Van der Weiden et al., 1994
Oncorhynchus mykiss, eggs	>99%	sac fry	LD50	**74 ng/g egg**	Walker & Peterson, 1991
Oncorhynchus mykiss, sac fry	NR	EROD	ECa	1.3 ng/embryo	Engwall et al., 1994
Oncorhynchus mykiss, 300-400 g	NR	AHH act.	ED50	1.0 µmol/kg	Janz & Metcalfe, 1991
PCB 105					
Oncorhynchus mykiss, eggs	>99%	sac fry	LD50	>6970 ng/g egg	Walker & Peterson, 1991

a only 1 dose applied 3 times in 9 weeks; concs in eggs was 0.96 mg/kg (0.69-1.41); development of the eggs was not included in the study.

References included in appendix I.c

Engwall, M.; Brunstroem, B.; Brewer, A.; Norrgren, L. (1994) Cytochrome P4501A induction by a coplanar PCB, a PAH mixture, and PCB-contaminated sediment extracts following microinjection of rainbow trout sac-fry. Aquat. Toxicol., 30:311-324

Janz, D.M.; Metcalfe, C.D. (1991) Relative induction of aryl hydrocarbon hydroxylase by 2,3,7,8-TCDD and two coplanar PCBs in rainbow trout (Oncorhynchus mykiss). Environ. Toxicol. Chem. 10,:917-923

Monosson , E.; Hogson, R.G.; Fleming, W.J.; Sullivan, C.V. (1996) Blood plasma levels of sex steroid hormones and vitellogenin in striped bass (Morone saxatilis) exposed to 3,3',4,4'-tetrachlorobiphenyl (TCB). Bull. Environ. Contam. Toxicol., 56:782-787

Skaare, J.U.; Jensen, E.G.; Goksyor, A.; Egaas, E. (1991) Response of xenobiotic metabolizing enzymes of rainbow trout (Oncorhynchus mykiss) to the mono-ortho substituted polychlorinated PCB congener 2,3',4,4',5-pentachlorobiiphenyl, PCB-118, detected by enzyme activities and immunochemical methods. Arch. Environ. Contam. Toxicol. 20:349-352

Van der Weiden, M.E.J.; De Vries, L.P.; Fase, K.; Celander, M.; Seinen, W.; Van den Berg, M. (1994) Relative potencies of polychlorinated dibeno-p-dioxins (PCDDs), dibenzofurans (PCDFs) and biphenyls (PCBs), for cytochrome P450 1A induction in the mirror carp (Cyprinus carpio). Aquat. Tox. 29:163-182

Walker, M.K.; Peterson, R.E. (1991) Potencies of polychlorinated dibenzo-p-dioxin, dibenzofuran, and biphenyl congeners, relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin, for producing early life stage mortality in rainbow trout (Oncorhynchus mykiss). Aquat. Tox. 21:219-23

I.d. Toxicity of PCBs to mammalian species

Data that are used for deriving the MPC are underlined, data that are used to derive critical levels in organic carbon related to histopathological or biochemical effects are given in italics.

species/age	exp. time	parameter	criterion	value (mg/kg w.w. or food)	remark	reference
PCB 77						
mouse (10-d, NMRI) 0.41 or 41 mg/kg bw	single oral dose	muscarinic cholinergic receptor binding in brain		0.41	decreased binding sites	Eriksson, 1988
mouse (13 days pregnant) 6, 9, 12 or 15 mg/kg bw	single i.p. injection	number of oocytes and follicles in 28 days old offspring		15	48% decrease in total number of germ cells	Rönnbäck, 1991
		reproductive capacity of offspring during 5 months	NOEC	>= 15		Rönnbäck, 1991
mouse (female C57BL/6J,) 7, 14, or 21 mg/kg	5 days oral gavage on day 1, 6 or 11 of pregnancy	maternal weight, pup weight, litter size, pup crown rump length	NOEC	>= 21		Rodrigues, 1997
mouse (CD-1, pregnant) 1, 2, 4, 8, 16, 32 or 64 mg/kg	day 6-15 of gestation, oral gavage	weight gain during pregnancy	NOEC	8		Marks et al., 1989
		signs of intoxication (vaginal bleeding, premature delivery, resorptions)	NOEC	1		Marks et al., 1989
		number of dead implantations	NOEC	4		Marks et al., 1989
		average of live fetuses per dam	NOEC	8		Marks et al., 1989
		average malformed fetuses per litter	NOEC	<u>2</u>		Marks et al., 1989
mouse (C57BL/6, male, 6-8 w old) 0.0012, 0.012, 0.12, 0.24 or 1.2 mg/kg	single i.p.	splenic immuno-suppression (a)	ED50	***0.0057-0.028***		Mayura et al., 1993
			NOEC	< 0.0012	34-84% decrease	Mayura et al., 1993
mouse (B6C3F1, female, 7-8 w old) 0, 0.024, 0.12, 0.24 or 1.2 mg/kg, immunization after 2 days with TNP-LPS	i.p injection	splenic immunosuppression (a)	ED50	0.164		Harper et al., 1995
			NOEC	0.024	51%, 55% and 60% decrease at 0.12, 0.24 and 1.2 mg/kg, resp.	Harper et al., 1995
		anti-TNP IgM titer	ED50	0.245		Harper et al., 1995
			NOEC	0.024	31% and 36% decrease at 0.12 and 0.24 mg/kg, resp.	Harper et al., 1995
		EROD activity	NOEC	>= 1.2	no significant changes	Harper et al., 1995
rat (Sprague-Dawley, pregnant) 2 or 8 mg/kg	oral, day 10-16 of gestation	serum thyroxine conc. in pups at day 21		8	15% decrease in females	Seo et al., 1995
		maternal weight gain maternal liver weight duration of gestation litter size	NOEC	> 8		Seo et al., 1995

species/age	exp. time	parameter	criterion	value (mg/kg w.w. or food)	remark	reference
		% live births				
		pup weight d 0 and 21				
		thyroid, liver and brain weight	NOEC	> 8		Seo et al., 1995
		thymus weight		8	15% decrease	Seo et al., 1995
rat (immature, 20 d) 0.16 mg	2 i.p. injections	uterine weight		0.16 mg/rat	no significant differences	Jansen et al., 1993
rat (Sprague-Dawley, male+female) 0, 0.10, 0.1, 1, or 10 mg/kg	oral diet, 13 w	body weight gain	NOEC	>=10	no significant changes in both males and females	Chu et al., 1995
		EROD activity, males	NOEC	1	375% increase at 10 mg/kg	Chu et al., 1995
		EROD activity, females	NOEC	*0.1*	120% and 500% increase at 1 and 10 mg/kg, resp.	Chu et al., 1995
		relative spleen weight. males	NOEC	0.1	23% and 31% increase at 1 and 10 mg/kg, resp.	Chu et al., 1995
		relative spleen weight, females	NOEC	>= 10	no significant changes	Chu et al., 1995
		relative kidney weight males	NOEC	< 0.01	18%, 14% and 14% increase at 0.01, 1, and 10 mg/kg, resp.	Chu et al., 1995
		relative kidney weight females	NOEC	>= 10	no significant changes	Chu et al., 1995
		hepatic vitamin A males and female	NOEC	1	37% and 44% decrease at 10 mg/kg for males and females, resp.	Chu et al., 1995
		renal and pulmonal vitamin A, females	NOEC	>= 10	no signnificant changes	Chu et al., 1995
		renal vitamin A, males	NOEC	1	314% increase at 10 mg/kg	Chu et al., 1995
		pulmonal vitamin A, males	NOEC	1	68% decrease at 10 mg/kg	Chu et al., 1995
		3,4-dihydroxylphenyl acetic acid in brain, (m)	NOEC	1	50% increase at 10 mg/kg	Chu et al., 1995
guinea pig	single oral	mortality	LD50	< 1		EHC 140, 1993
mink (female, 4-7 months) one dose level 50 mg/kg	3 days i.p. injection	clinical signs of illness		50	anorexia, depression and melena	Gilette et al., 1987
		gross and microscopic lesions		50	epithelial necrosis, atrophic and fused villi in the intestines	Gilette et al., 1987
		liver, spleen, kidney and brain weight		50	no significant differences	Gilette et al.,1987a
rat (Sprague-Dawley, female, 45 d) one dose level 50 mg/kg	3 days i.p. injection	clinical signs of illnees		50	no findings	Gilette et al., 1987
		gross and microscopic lesions		50	no findings	Gilette et al., 1987
		spleen and liver weight		50	significantly increased	Gilette et al., 1987a
rat (Sprague-Dawley, male), 0.01, 0.1, 1, or 10 mg/kg	13 w, diet	hepatic vitamin A		3.5	25% reduction	Håkansson et al., 1994
monkey (Macaca mulatta, pregnant) 3.15 or 6.3 mg/kg bw	9 oral doses by gavage	number of abortions, maternal toxicity	NOEC	< 3.15		McNulty, 1985

species/age	exp. time	parameter	criterion	value (mg/kg w.w. or food)	remark	reference
	during day 20 to 40					
monkey (Callithrix jacchus, young adult female) 0.1, 1 or 3 mg/kg, twice per week, for 18 to 23 weeks	oral	serum thyroxine (T4)		0.1	35% decrease (99.9 and 81% at 3 and 1 mg/kg, resp)	van den Berg et al., 1988
		free thyroxine index	NOEC	***0.1***	(97% and 80% decrease at 3 and 1 mg/kg, resp.)	van den Berg et al., 1988
		serum triiodothyronine (T3)	NOEC	1	60% decrease at 3 mg/kg	van den Berg et al., 1988
		T3 resin uptake	NOEC	1	11% increase at 3 mg/kg	van den Berg et al., 1988
		serum thyrotropin (TSH)	NOEC	1	80% and 535% increase at 3 mg/kg after 2 and 18 weeks, resp.	van den Berg et al., 1988
PCB 105						
rat (immature male), dose levels not reported		body weight gain	ED50	**245**		Safe, 1987
rat (immature male), dose levels not reported		thymus atrophy	ED50	336		Safe, 1987
rat (Holtzman, pregnant) day 6 of gestation to postnatal day 21 0.4, 1.0, or 2.5 mg/kg	oral	plasma thyroxine in pups at postnatal day 21	ED50	***0.5***	no effects in dams	Moore et al., 1996
mouse (adult male, C57BL/6J) 245 or 490 mg/kg bw (c)	single i.p. injection	body weight		490	6% decrease	Robertson et al., 1984
		rel. liver weight		490	34% increase	Robertson et al., 1984
		rel. thymus weight		490	35% decrease	Robertson et al., 1984
		rel spleen weight		490	no significant changes	Robertson et al., 1984
mouse (adult male, DBA/2J) 245 or 490 mg/kg bw (c)		body weight		490	3% increase	Robertson et al., 1984
		rel. liver, thymus or spleen weight		490	no significant changes	Robertson et al., 1984
mouse (B6C3F1, female) 25, 50, 100, or 150 mg/kg	single i.p. injection	EROD activity	ED50	***148***		Connor et al., 1995
PCB 118						
rat (immature male), dose levels not reported		body weight gain	ED50	366		Safe, 1987
rat (immature male), dose levels not reported		thymus atrophy	ED50	506		Safe, 1987
rat (Sprague-Dawley, male) 0.01, 0.1, 1, or 10 mg/kg	13 w, diet	hepatic vitamin A	NOEC	>=10		Håkansson et al., 1994
rat (Sprague-Dawley, female)	13 w, diet	hepatic vitamin A	NOEC	>=2		Håkansson et al., 1994

species/age	exp. time	parameter	criterion	value (mg/kg w.w. or food)	remark	reference
0.002, 0.02. 0.2 or 2 mg/kg rat (Sprague-Dawley) male: 0.01, 0.1, 1, or 10 mg/kg	13 w, diet	liver cell abnormalities	LOEC	0.01	proliferation of smooth endoplasmatic reticulum mitochondrial aberration increase of lipofuscin granules and lipid droplets	MacLellan et al., 1994
rat (Sprague-Dawley) female: 0.002, 0.02, 0.2, or 2 mg/kg	13 w, diet	liver cell abnormalities	LOEC	***0.002***	proliferation of smooth endoplasmatic reticulum	MacLellan et al., 1994
rat (Sprague-Dawley) female: 0.002, 0.02, 0.2, or 2 mg/kg	13 w, diet	liver cell abnormalities	LOEC	0.02	mitochondrial aberration increase of lipofuscin granules	MacLellan et al., 1994
rat (Sprague-Dawley, male) 0, 0.10, 0.1, 1, or 10 mg/kg	oral diet, 13 w	body weight gain	NOEC	**1**	12% increase at 10 mg/kg	Chu et al., 1995
		EROD activity	NOEC	1	520% increase at 10 mg/kg	Chu et al., 1995
		relative liver weight	NOEC	>= 10	no significant changes	Chu et al., 1995
		aminopyrine demethylase	NOEC	>=10	no significant changes	Chu et al., 1995
rat (Sprague-Dawley, female) 0, 0.002, 0.02, 0.2 or 2 mg/kg	oral diet, 13 w	body weight gain	NOEC	>=2	no significant changes	Chu et al., 1995
		EROD activity	NOEC	0.2	180% increase at 2 mg/kg,	Chu et al., 1995
		relative liver weight	NOEC	>=2	no significant changes	Chu et al., 1995
		aminopyrine demethylase	NOEC	>= 2	no significant changes	Chu et al., 1995
		dopamine in brain	NOEC	0.2	8% and 34% decrease at 2 mg/kg in caudate nucleus and substantia nigra, resp.	Chu et al., 1995
rat (Sprague-Dawley, pregnant 4 or 16 mg/kg	oral gavage d 10-16 of gestation	serum thyroxine (T4) in pups 21 days old		4	significant decreased	Ness et al., 1993
		maternal weight gain maternal liver weight litter size % live births	NOEC	> 16	no significant changes	Ness et al., 1993
		pup weight at d 7, 14 and 21		16	27%, 22% and 26% decrease in both sexes at day 7, 14 and 21, resp.	Ness et al., 1993
		pup weight at d 7, 14 and 21		4	ca 8% decrease in females at day 7, 14 and 21.	Ness et al., 1993
		pup birth weight		16	13% and 15% decrease in males and females, resp.	Ness et al., 1993
mouse (B6C3F1, female) 0, 3, 7.5, 15 or 30 mg/kg	oral gavage 13 w	hepatic porphyrin	NOEC	*3*	significant increase at higher doses	van Birgelen et al., 1995
mouse (adult male, C57BL/6J) 245 or 490 mg/kg bw (c)	single i.p. injection	body weight, rel. liver, thymus or spleen weight		490	no significant changes	Robertson et al., 1984
mouse (adult male, DBA/2J)		body weight, rel. liver, thymus or		490	no significant changes	Robertson et al., 1984

species/age	exp. time	parameter	criterion	value (mg/kg w.w. or food)	remark	reference
245 or 490 mg/kg bw (c)		spleen weight				
mouse (B6C3F1, female) 25, 50, 100, or 150 mg/kg PCB 126	single i.p. injection	EROD activity	ED50	4785		Connor et al., 1995
rat (immature male), dose levels not reported		body weight gain	ED50	1.1		Safe, 1987
rat (immature male), dose levels not reported		thymus atrophy	ED50	0.3		Safe, 1987
rat (Sprague-Dawley, male) 0.0001. 0.001, 0.01 or 0.1 mg/kg	13 w, diet	relative liver weight	NOEC	0.001	16% and 38% increase at 0.01 and 0.1 mg/kg, resp.	Chu et al., 1994
		body weight	NOEC	**0.01**	12% decrease at 0.1 mg/kg	Chu et al., 1994
		relative weight of heart, spleen, kidney, or brain	NOEC	0.01	13-29% increase at 0.1 mg/kg	Chu et al., 1994
+female		haematological changes (d)	NOEC	0.01		Chu et al., 1994
		cholesterol, glucose	NOEC	0.01	69% increase and 16% decrease, resp. at 0.1 mg/kg	Chu et al., 1994
		AP, ASAT, bilirubin	NOEC	>= 0.1		Chu et al., 1994
+female		EROD activity	NOEC	=< 0.0001	increase at all dose levels	Chu et al., 1994
+ female		uroporphyrin	NOEC	0.001		Chu et al., 1994
+female		hepatic vitamin A	NOEC	0.001		Chu et al., 1994
+ female		renal vitamin A	NOEC	0.01		Chu et al., 1994
rat (Sprague-Dawley, female) 0.0001. 0.001, 0.01 or 0.1 mg/kg	13 w, diet	body weight, relative weight of liver, heart, spleen, kidney or brain	NOEC	0.01		Chu et al., 1994
		cholesterol	NOEC	0.001	30% and 35% increase at 0.01 and 0.1 mg/kg, resp.	Chu et al., 1994
		AP, glucose	NOEC	0.01	60% increase and 29% decrease resp. at 0.1 mg/kg	Chu et al., 1994
		ASAT, bilirubin	NOEC	>= 0.1		Chu et al
rat (Wistar, 4 w)	single i.p. injection	hepatic G6PDH activity		**25**	significantly increased	Hori et al., 1997
rat (Sprague-Dawley, male) 0.0001, 0.001. 0.01 or 0.1 mg/kg	13 w, diet	hepatic vitamin A		0.006	25% reduction	Håkansson et al., 1994
		hepatic vitamin A	NOEC	0.01		Håkansson et al., 1994
rat (Sprague-Dawley, female) 0.0001, 0.001, 0.01 or 0.1 mg/kg	13w, diet	hepatic vitamin A	NOEC	0.01		Håkansson et al., 1994
rat (Sprague-Dawley, female) 0.007, 0.050, or 0.18 mg/kg diet	13 w, diet	hepatic porphyrin	NOEC	0.007	ca. 70% increase at 0.050 and 0.18 mg/kg	van Birgelen et al., 1996
rat (Sprague-Dawley, male and female) 0.0001, 0.001, 0.01, or 0.1 mg/kg	13 w, diet	liver cell abnormalities	LOEC	***0.0001***	proliferation of smooth endoplasmatic reticulum mitochondrial aberration increase of lipid droplets	MacLellan et al., 1994

species/age	exp. time	parameter	criterion	value (mg/kg w.w. or food)	remark	reference
rat (Sprague-Dawley, pregnant) 0.00025 or 0.001mg/kg	oral, day 10-16 of gestation	serum thyroxine conc. in pups at day 21	NOEC	> 0.001		Seo et al., 1995
		maternal weight gain maternal liver weight duration of gestation litter size % live births pup weight d 0 and 21	NOEC	> 0.001		Seo et al., 1995
		thymus, thyroid and brain weight	NOEC	>0.001		Seo et al., 1995
		liver weight		0.00025	13% increase	Seo et al., 1995
		liver weight		0.001	17% increase	Seo et al., 1995
mouse (adult male, C57BL/6J) 245 or 490 mg/kg bw (c)	single i.p. injection	body weight		245	18% decrease	Robertson et al., 1984
		rel. liver weight		245	23% increase	Robertson et al., 1984
		rel thymus or spleen weight		245	no significant changes	Robertson et al., 1984
mouse (adult male, DBA/2J) 245 or 490 mg/kg bw (c)		rel. liver weight		490	65% increase	Robertson et al., 1984
		body weight, rel. thymus or spleen weight		490	no significant changes	Robertson et al, 1984
mouse (male DBA/2N, 4 w) 25, 50, 75, 100 or 150 mg/kg	single i.p. injection	hepatic G6PDH activity	NOEC	> 150		Hori et al., 1997
mouse (male C57BL/6J, 4 w) 0.5, 5, 15, 25, or 50 mg/kg	single i.p. injection	hepatic G6PDH activity	NOEC	15		Hori et al., 1997
mouse (B6C3F1, female, 7-8 w old) 0, 0.00012, 0.0012, 0.0024 or 0.012 mg/kg, immunization after 2 days with TNP-LPS	i.p injection	splenic immunosuppression (a)	ED50	0.0047		Harper et al., 1995
			NOEC	***0.00012***	28%, 51% and 58% decrease at 0.0012, 0.0024 and 0.012 mg/kg, resp.	Harper et al., 1995
		anti-TNP IgM titer	ED50	0.0056		Harper et al., 1995
			NOEC	0.00012	40%, 42% and 44% decrease at 0.0012, 0.0024 and 0.012 mg/kg resp.	Harper et al., 1995
		EROD activity	NOEC	0.0024	293% increase at 0.012 mg/kg	Harper et al., 1995
mouse (C57BL/6, pregnant) 0.13, 0.26 or 0.52 mg/kg bw	single oral day 10 of gestation	number of implants/litter, resorptions or deaths/litter live fetuses/litter fetal body weight maternal weight gain litter weight	NOEC	> 0.52	no significant changes	Mayura et al., 1993
		rel. maternal liver weight	NOEC	< 0.13	significant increased at all doses	Mayura et al., 1993
		fetuses with cleft palate	NOEC	0.13	13.5% and 92.3% at 0.26 and 0.52 mg/kg,	Mayura et al., 1993

species/age	exp. time	parameter	criterion	value (mg/kg w.w. or food)	remark	reference
		fetuses with hydronephrosis	NOEC	< 0.13	resp. 48.3%, 98.6% and 100% at 0.13, 0.26 and 0.52 mg/kg, resp.	Mayura et al., 1993
mouse (C57BL/6, male, 6-8 w old) 0.00012, 0.0012, 0.012, 0.024 or 0.12 mg/kg	singel i.p.	splenic immuno-suppression (a)	ED50	0.001-0.0014		Mayura et al., 1993
guinea pig (male Hartley) 0.5 mg/kg PCB 153	single i.p. injection	hepatic G6PDH activity		0.5	significantly decreased	Hori et al., 1997
guinea pig	single oral	mortality	LD50	> 10		EHC 140, 1993
rat (Sprague-Dawley, male) 0.05, 0.5, 5 or 50 mg/kg	13 w, diet	hepatic vitamin A		26	25% reduction	Håkansson et al., 1994
rat (Sprague-Dawley, male, female) 0.05, 0.5, 5 or 50 mg/kg	13 w, diet	hepatic vitamin A	NOEC	*5*		Håkansson et al., 1994
mouse (NMRI, pregnant) one dose level 0.05 mg/kg	day 5-19 of gestation, oral	reproductive capacity		0.05	not affected	Örberg, 1977
mouse, (C57BL/6N, pregnant) 25 or 50 mg/kg bw	oral gavage day 10-13 of gestation	maternal weight gain rel. maternal liver weight, number of live fetuses/litter, live fetal weight, placenta weight, % fetal mortality, % fetuses with cleft palate		50	no significant changes	Birnbaum et al., 1985
rat (female), 10, 30 or 100 mg/kg	13 w diet	body weight gain	NOEC	>=100	factor 10?	van der Kolk et al., 1992
		relative liver weight			increase	van der Kolk et al., 1992
rat (Sprague-Dawley, female) 10, 30, 100 mg/kg diet	13 w, diet	hepatic porphyrin	NOEC	>= 100	no significant changes	van Birgelen et al., 1996
rat ((SprageDawley, female, 20 d old) 8, 11, 25, 51, 59 mg/kg (total dose: 16, 22, 50, 102, 118 mg/kg)	i.p injection on d 20 and 21	uterine weight	NOEC	22	63% and 46% increase at 50 and 102 mg/kg (no changes at 118 mg/kg	Li, et al., 1994
		EROD activity	NOEC	>= 118	no significant changes	Li et al., 1994
		PROD activity	NOEC	=< 16	increase at all dose levels (223-765%)	Li et al., 1994
		serum total T4 conc.	NOEC	>= 118	no significant changes	Li et al., 1994
rat (WAG/Rij, female, 4 w old) one dose of 30 mg/kg bw vit. K3 deficient casein diet	oral gavage single dose	blood coagulation time, 1, 2, or 3 weeks after dosing		30	no effect after 1 week, 37% and 149% increase after 2 and 3 weeks, resp.	Bouwman et al., 1992
rat (Sprague-Dawley, pregnant 16 or 64 mg/kg	oral gavage d 10-16 of gestation	serum thyroxine (T4) in pups 21 days old		16	significant decreased	Ness et al., 1993
		maternal weight gain maternal liver weight	NOEC	> 64	no significant changes	Ness et al., 1993

species/age	exp. time	parameter	criterion	value (mg/kg w.w. or food)	remark	reference
		litter size				
		% live births				
		pup birth weight				
		pup weight at d 7, 14 and 21		**64**	ca 9% decrease in females after d 7, 14 and 21 and in males at d 21	Ness et al., 1993
mink (anestrous, immature) 20 mg/kg, livers and uteri collected after 6 h 3 d or 14 d	single i.p. injection	uterine nuclear estrogen receptor concentration (b)		20	31% and 42% decrease at 6 h and 3 d after injection, resp.	Patnode & Curtis, 1994
		uterine cytosolic estrogen receptor concentration (b)		20	no changes	Patnode & Curtis, 1994
		liver cytochrome P450		20	49% and 46% decrease at 6 h and 14 d after injection, resp.	Patnode & Curtis, 1994
		liver EROD activity		20	no significant effect	Patnode & Curtis, 1994
mink (pregnant) 20 mg/kg, embryo and tissue collection 3, 7, or 14 d after injection	single i.p. injection	uterine nuclear estrogen receptor concentration		20	no significant effect	Patnode & Curtis, 1994
		uterine cytosolic estrogen receptor concentration		20	229% increase 14 d after injection	Patnode & Curtis, 1994
		liver cytochrome P450		20	no significant effect	Patnode & Curtis, 1994
		liver EROD activity		20	no significant effect	Patnode & Curtis, 1994
		number of implantation sites		20	no significant effect	Patnode & Curtis, 1994
		number of embryos		20	significantly reduced (24%)	Patnode & Curtis, 1994
		embryonal weight, crown-rump and head lenght		20	significantly reduced	Patnode & Curtis, 1994
		uterine progesterone receptor concentration		20	265% increase at day 14	Patnode & Curtis, 1994
		progesterone receptor dissociation constant		20	100% increase at day 14	Patnode & Curtis, 1994
mink (female) dosed before and during mating and during pregnancy until day 88 or 102; 2.5 or 5 mg/kg	12.5-14.5 w oral diet	body weight gain at week 8		**2.5**	significant weight loss at both dose levels; transient at week 12.5	Aulerich et al., 1985
		mortality	NOEC	>= 5	10% and 0% mortality at 2.5 and 5 mg/kg, resp.	Aulerich et al., 1985
		weight of brain, liver, spleen, kidneys, lungs, heart or adrenals		5	no significant changes	Aulerich et al., 1985
		reproductive performance		5	no significant changes	Aulerich et al., 1985
		plasma 17ß-estradiol or progesterone		5	no significant changes	Aulerich et al., 1985
		Cytochrome P-450 conc.		2.5	111% increase at both dose levels	Aulerich et al., 1985
		aminopyrine-N-demethylase activity		5	75% increase at 5 mg/kg	Aulerich et al., 1985
		benzo[a]pyrene hydroxylase activity		2.5	95% and 59% increase at 2.5 and 5 mg/kg,	Aulerich et al., 1985

species/age	exp. time	parameter	criterion	value (mg/kg w.w. or food)	remark	reference
					resp.	
		EROD activity		5	no significant changes	Aulerich et al., 1985
		norepinephrine conc. in medulla		5	20% increase, no effect in other brain portions	Aulerich et al., 1985
		dopamine conc. in brain		5	no significant changes in all brain portions	Aulerich et al., 1985
		serotonin conc. in brain		5	no significant changes in all brain portions	Aulerich et al., 1985
PCB 156						
mouse (B6C3F1, female) 25, 50, 100, or 150 mg/kg	single i.p. injection	EROD activity	ED50	107		Connor et al., 1995
rat (immature male), dose levels not reported		body weight gain	ED50	65		Safe, 1987
rat (immature male), dose levels not reported		thymus atrophy	ED50	65		Safe, 1987
mouse (6-8 w, B6), 12.7 or 127 mg	single i.p. injection	immune response suppression (a)	loec	127	70% decrease	Silkworth et al., 1984
		rel. spleen weight		127	20% decrease	Silkworth et al., 1984
		rel. thymus weight		127	no significant changes	Silkworth et al., 1984
mouse (adult male, C57BL/6J) 272 or 543 mg/kg bw (c)	single i.p. injection	body weight	loec	543	11% decrease	Robertson et al., 1984
		rel. liver weight		543	21% increase	Robertson et al., 1984
		rel. thymus weight		543	38% decrease	Robertson et al., 1984
		rel. spleen weight		543	no significant changes	Robertson et al., 1984
mouse (adult male, DBA/2J) 272 or 543 mg/kg bw (c)		body weight, rel. liver, thymus or spleen weight		543	no significant changes	Robertson et al., 1984
mouse (male C57BL/6N) 0.36, 1.8, 9.1, or 36.2 mg/kg	single i.p. injection	splenic immune response (a)	ED50	**0.7**	decrease	Davis & Safe, 1990
			NOEC	***0.36***	47% decrease ?????	Davis & Safe, 1990
mouse, (C57BL/6N, pregnant) 40 or 80 mg/kg bw	oral gavage once at day 11 of gestation	maternal weight gain number of live fetuses/litter, live fetal weight, placenta weight, % fetal mortality, % fetuses with cleft palate		80	no significant changes	Birnbaum et al., 1985
		rel. maternal liver weight		40	15% increase (21% increase at 80 mg/kg)	Birnbaum et al., 1985
mouse, (C57BL/6N, pregnant) 10 or 20 mg/kg bw	oral gavage day 10-13 of gestation	maternal weight gain rel. maternal liver weight, number of live fetuses/litter, live fetal weight, placenta weight, % fetal mortality, % fetuses with cleft palate		20	no significant changes	Birnbaum et al., 1985
rat (Sprague-Dawley, female) 1.2, 6, or 12 mg/kg diet	13 w, diet	hepatic porphyrin	NOEC	1.2	94% and 137% increase at 6 and 12 mg/kg, resp.	van Birgelen et al., 1994
		body weight	NOEC	**1.2**	16% and 20% decrease at 6 and 12 mg/kg, resp.	van Birgelen et al, 1994

species/age	exp. time	parameter	criterion	value (mg/kg w.w. or food)	remark	reference
		relative liver weight	NOEC	1.2	36% and 58% increase at 6 and 12 mg/kg, resp.	van Birgelen et al., 1994
		relative thymus weight	NOEC	1.2	30% decrease at 6 and 12 mg/kg	van Birgelen et al., 1994
		relative kidney weight	NOEC	1.2	18% and 23% increase at 6 and 12 mg/kg, resp.	van Birgelen et al., 1994
		relative spleen weight	NOEC	>= 12		van Birgelen et al., 1994
PCB 157						
rat (immature male), dose levels not reported		body weight gain	ED50	**80**		Safe, 1987
rat (immature male), dose levels not reported		thymus atrophy	ED50	81		Safe, 1987
mouse (adult male, C57BL/6J) 272 or 543 mg/kg bw (c)	single i.p. injection	rel liver weight		543	33% increase	Robertson et al., 1984
		body weight, rel. thymus or spleen weight		543	no significant changes	Roberson et al., 1984
mouse (adult male, DBA/2J) 272 or 543 mg/kg bw (c)		rel liver weight		543	13% increase	Robertson et al., 1984
		body weight, rel thymus or spleen weight		543	no significant changes	Robertson et al., 1984
PCB 169						
mouse (CD-1, pregnant) 0, 0.1, 1, 2, 4, 8 or 16 mg/kg bw/day	oral gavage day 6-15 of gestation	maternal weight gain	NOEC	4	(11% decrease at 8 mg/kg bw)	Marks et al., 1981
		number of live fetuses/dam	NOEC	2	(20% decrease at 4 mg/kg bw)	Marks et al., 1981
		average number of implantations/dam	NOEC	4	(10% decrease at 8 mg/kg bw	Marks et al., 1981
		% resorptions	NOEC	4	10% increase at 8 mg/kg bw	Marks et al., 1981
		average fetal weight	NOEC	4	8% decrease at 8 mg/kg bw	Marks et al., 1981
		average % malformed fetuses	NOEC	1	11%, 36%,65% and 60% increase at 2, 4, 8, and 16 mg/kg, resp.	Marks et al., 1981
mouse (B6C3F1, female) 0, 0.03 or 0.09 mg/kg	oral gavage 13 w	hepatic porphyrin	LOEC	0.03	significant increase	van Birgelen et al., 1995
mouse (B6C3F1, female, 7-8 w old) 0, 0.00024, 0.0024, 0.012, 0.024 mg/kg, immunization after 2 days with TNP-LPS	i.p injection	splenic immunosuppression (a)	ED50	0.0111		Harper et al., 1995
			NOEC	***0.00024***	47%, 48% and 52% decrease at 0.0024, 0.0012 and 0.024 mg/kg, resp.	Harper et al., 1995
		anti-TNP IgM titer	ED50	0.0094		Harper et al., 1995
			NOEC	0.00024	39%, 41% and 41% decrease at 0.0024,	Harper et al., 1995

species/age	exp. time	parameter	criterion	value (mg/kg w.w. or food)	remark	reference
					0.0012 and 0.024 mg/kg, resp.	
		EROD activity	NOEC	>= 0.024	no significant changes	Harper et al., 1995
mouse (C57BL/6, male, 6-8 w old) 0.00024, 0.0024, 0.024, 0.048 or 0.24 mg/kg	singel i.p.	splenic immuno-suppression (a)	ED50	0.0007-0.0027		Mayura et al., 1993
			NOEC	< 0.00024	38-79% decrease	Mayura et al., 1993
rat (day 1 of gestation) 0.2, 0.6, or 1.8 mg/kg bw	single dose oral gavage	pup weight, duration of gestation, mean litter size	NOEC	**0.6**	2-generation reproduction study	Smits-van Prooije et al., 1993
rat (F1 generation)		mating succes, pregnancy rate	NOEC	0.6	2-generation reproduction study	Smits-van Prooije et al., 1993
rat (immature male), dose levels not reported		body weight gain	ED50	5.4		Safe, 1987
rat (immature male), dose levels not reported		thymus atrophy	ED50	3.2		Safe, 1987
guinea pig	single oral	mortality	LD50	**0.5**		EHC 140, 1993
mink (female) 0.01 or 0.05 mg/kg	135 days, diet	mortality		0.05		Aulerich et al.,1987
		body weight loss		**0.05**	23% weight loss	Aulerich et al., 1987
		liver weight		0.05	64% increase	Aulerich et al., 1987
		brain weight		0.05	22% increase	Aulerich et al., 1987
		kidneys weight		0.05	55% increase	Aulerich et al., 1987
		heart weight		0.05	40% increase	Aulerich et al., 1987
		adrenals weight		0.05	198% increase	Aulerich et al., 1987
		spleen weight		0.05	no significant increase	Aulerich et al., 1987
		serum thyroxine, triiodothyronine		0.05	55-66% decrease	Aulerich et al., 1987
mink (anestrous, immature) 0.4 mg/kg, livers and uteri collected after 6 h, 3 d or 14 d	single i.p. injection	uterine nuclear estrogen receptor concentration (b)		0.4	25% and 39% decrease at 6 h and 3 d after injection, resp.	Patnode & Curtis, 1994
		uterine cytosolic estrogen receptor concentration (b)		0.4	no significant changes	Patnode & Curtis, 1994
		liver cytochrome P450 (b)		0.4	50% decreaseand 76% increase at 6 h and 14 d after injection, resp.	Patnode & Curtis, 1994
		liver EROD activity (b)		0.4	990% increase 14 d after injection	Patnode & Curtis, 1994
mink (pregnant) 0.4 or 0.8mg/kg, embryo and tissue collection 3, 7, or 14 d after injection	single i.p. injection	uterine nuclear estrogen receptor concentration		0.8	no significant effect	Patnode & Curtis, 1994
		uterine cytosolic estrogen receptor concentration		0.8	40% increase 14 d after injection	Patnode & Curtis, 1994
		liver cytochrome P450		0.8	96% and 194% increase	Patnode & Curtis, 1994

species/age	exp. time	parameter	criterion	value (mg/kg w.w. or food)	remark	reference
					at 7 and 14 d after injection, resp.	
		liver EROD activity		0.8	100% increase at day 7	Patnode & Curtis, 1994
				0.4	275% increase at day 14	Patnode & Curtis, 1994
		number of implantation sites	NOEC	0.8		Patnode & Curtis, 1994
		number of embryos		0.4	no embryos at 0.4 mg/kg and 33% decrease at 0.8 mg/kg)	Patnode & Curtis, 1994
		embryonal weight, crown-rump and head lenght		0.4	significantly reduced	Patnode & Curtis, 1994
		uterine progesterone receptor concentration		0.8	98% increase at day 14	Patnode & Curtis, 1994
		progesterone receptor dissociation constant		0.4	265% increase at day 14	Patnode & Curtis, 1994
mink (female) dosed before and during mating and during pregnancy until day 88 or 102; 0.1 or 0.5 mg/kg	12.5-14.5 w oral diet	body weight gain	NOEC	<0.1	significant weight loss during whole treatment period at both doses	Aulerich et al., 1985
		mortality		0.1	100% mortality at 0.5 mg/kg	Aulerich et al., 1985
		weight of brain, liver, or lungs		0.5	no significant changes	Aulerich et al., 1985
		weight of spleen and kidneys		0.5	46% decrease and 27% increase, resp. at 0.5 mg/kg	Aulerich et al., 1985
		weight of heart or adrenals		0.1	in heart 19 %decrese in adrenals 123% increase	Aulerich et al., 1985
		reproductive performance		0.1	no kits whelped	Aulerich et al., 1985
		plasma 17ß-estradiol		0.1	no significant changes	Aulerich et al., 1985
		plasma progesterone		0.1	168% increase	Aulerich et al., 1985
		Cytochrome P-450 conc.		0.1	270% increase	Aulerich et al., 1985
		aminopyrine-N-demethylase activity		0.1	no significant changes	Aulerich et al., 1985
		benzo[a]pyrene hydroxylase activity		0.1	173% increase	Aulerich et al., 1985
		EROD activity		0.1	no significant changes	Aulerich et al., 1985
		norepinephrine conc. in brain		0.1	no significant changes in any brain portion	Aulerich et al., 1985
		dopamine conc. in brain		0.1	74% decrease and 66% increase in midbrain and cerebral hemispheres, resp.	Aulerich et al., 1985
		serotonin conc. in brain		0.1	no significant changes in all brain portions	Aulerich et al., 1985

a: as measured by splenic antibody plaque-forming cells
b: 24 h before uterine tissue collection the animals received 100 µg estradiol by s.c injection
c: results are given for only one concentration
d: Hb, Ht, RBC, MCH, MCV and platelets

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I.e. Toxicity of PCBs to avian species

Data that are used for deriving the MPC are underlined, data that are used to derive critical levels in organic carbon related to histopathological or biochemical effects are given in italics.

species/age	exp. time	parameter	criterion	value (µg/kg w.w.)	remark	reference
PCB 77						
Anas platyrhynchos, 5-d old eggs	19 days	embryo mortality	LD0	5000	injected into yolk	Brunström, 1991b
Anas platyrhynchos, eggs	hatch time	hatching rate	ED0	100	injected into yolk	Bosveld & van den Berg, 1995
Anser anser , eggs	hatch time	hatching rate	ED0	1000	injected into yolk	Bosveld. 1995
Anser anser, 5-d old eggs	20 days	embryo mortality	LD0	1000	injected into yolk	Brunström, 1988
Bucephala clangula, eggs	hatch time	hatching rate	ED0	1000	injected into yolk	Bosveld & van den Berg 1995
Chick, 17-d old eggs	24 h	AHH activity	ED50	0.934 ug/egg		Rifkind et al., 1984
Chick, 17-d old eggs	24 h	EROD activity	ED50	0.934 ug/egg		Rifkind et al., 1984
Falco sparverius, 4-d-old eggs	hatch time	embryo mortality	LD50	**688**	injected into air sac	Hoffman et al, 1996
Gallus gallus, day 0 eggs	24 days	embryo mortality	LD50	8.8	injected into yolk	Powell et al., 1996a
Gallus gallus, day 0 eggs	24 days	embryo abnormality	ED15	27	injected into yolk	Powell et al., 1996a
Gallus gallus, day 0 eggs	24 days	posthatch body weight	LOAEL	3	injected into yolk	Powell et al., 1996a
Gallus gallus, 4-d-old eggs	14 days	embryo mortality	LD100	50	injected into yolk	Brunström, 1991b
Gallus gallus, 4-d-old eggs	14 days	embryo mortality	LD17	10	injected into yolk	Brunström, 1991b
Gallus gallus, 7-d-old eggs	3 days	EROD activity	ED50	2	injected into air sac	Brunström, 1991a,b
Gallus gallus, 7-d-old eggs	3 days	embryo mortality	LD50	8.6	injected into air sac	Brunström & Andersson, 1988
Gallus gallus, 13-d-old eggs	5 days	lymphoid development inhibition	ED50	14.6	injected into air sac	Brunström et al., 1990
Gallus gallus, 5-d old eggs	48 h	AHH activity	ED50	***0.6***	injected into air sac	Brunström & Lund, 1988
Larus ridibundus, eggs	hatch time	hatching rate	ED0	1000	injected into yolk	Bosveld & van den Berg, 1995
Larus argentatus, eggs	hatch time	hatching rate	ED0	1000	injected into yolk	Bosveld & van den Berg, 1995
Larus argentatus, 5-d old eggs	19 days	embryo mortality	LD13	1000	injected into yolk	Brunström, 1988
Meleagris gallopova, 5-d old eggs	19 days	embryo mortality	LD60	**1000**	injected into yolk	Brunström, 1991b
Meleagris gallopova, 7-d old eggs	48 h	AHH activity	ED50	***6***	injected into air sac	Brunström & Lund, 1988
Phasianus colchicus, eggs	hatch time	hatching rate	ED0	100	injected into yolk	Bosveld & van den Berg, 1995
Phasianus colchicus, eggs	hatch time	hatching rate	ED100	1000	injected into yolk	Bosveld & van den Berg 1995
Gallus gallus, 4 d old eggs	hatch time	survival day 11-18, hatching succes, malformed or edema	NOEC	**1.2**	injected into air sac	Hoffman et al., 1998
Somateria mollissima, 5-d old eggs	19 days	embryo mortality	LD20	1000	injected into yolk	Brunström, 1991b
Falco sparverius, 6 d old eggs	hatch time	survival day 11-18, hatching succes, malformed or edema	NOEC	<100	injected into air sac	Hoffman et al., 1998
PCB 105						
Falco sparverius, 1-y old females	28 days	APND activity	ED61	3000	3 oral doses/week	Elliott et al., 1991
Gallus gallus, day 0 eggs	24 days	embryo mortality	LD50	5592	injected into yolk	Powell et al., 1996a
Gallus gallus, day 0 eggs	24 days	embryo abnormality	ED22	8100	injected into yolk	Powell et al., 1996a
Gallus gallus, day 0 eggs	24 days	posthatch body weight	LOAEL	**300**	injected into yolk	Powell et al., 1996a
Gallus gallus, 4-d old eggs	14 days	embryo mortality	LD85	2500	injected into yolk	Brunström, 1991b
Gallus gallus, 7-d old eggs	3 days	EROD activity	ED50	150	injected into air sac	Brunström, 1991b
Gallus gallus, 7-d old eggs	3 days	embryo mortality	LD50	2200	injected into air sac	Brunström, 1990

species/age	exp. time	parameter	criterion	value (µg/kg w.w.)	remark	reference
Gallus gallus, 13-d-old eggs	5 days	lymphoid development inhibition	ED50	***1.14***	injected into air sac	Brunström et al., 1990
PCB 118						
Gallus gallus, 4-d old eggs	14 days	embryo mortality	LD45	**5000**	injected into yolk	Brunström, 1991b
Gallus gallus, 7-d old eggs	3 days	EROD activity	ED50	2200	injected into air sac	Brunström, 1991b
Gallus gallus, 7-d old eggs	3 days	embryo mortality	LD50	>4000	injected into air sac	Brunström et al., 1990
Gallus gallus, 13-d-old eggs	5 days	lymphoid development inhibition	ED50	***2.55***	injected into air sac	Brunström et al., 1990
PCB 126						
Falco sparverius, 1-y old females	28 days	EROD activity	ED46	50	3 oral doses/week	Elliott et al., 1991
Falco sparverius, 1-y old females	28 days	AE activity	ED80	50	3 oral doses/week	Elliott et al., 1991
Falco sparverius, 4-d-old eggs	hatch time	embryo mortality	LD50	65	injected into air sac	Hoffman et al, 1996
Gallus gallus, day 0 eggs	24 days	embryo mortality	LD50	0.6	injected into yolk	Powell et al., 1996a
Gallus gallus, day 0 eggs	24 days	embryo abnormality	ED22	0.9	injected into yolk	Powell et al., 1996a
Gallus gallus, day 0 eggs	24 days	posthatch body weight	LOAEL	0.9	injected into yolk	Powell et al., 1996a
Gallus gallus, 4-d old eggs	14 days	embryo mortality	LD90	2	injected into yolk	Brunström, 1991b
Gallus gallus, 7-d old eggs	3 days	EROD activity	ED50	***0.1***	injected into air sac	Brunström, 1991b
Gallus gallus, 13-d-old eggs	5 days	lymphoid development inhibition	ED50	1.3	injected into air sac	Brunström et al., 1990
Gallus gallus, 7-d old eggs	3 days	embryo mortality	LD50	3.1	injected into air sac	Brunström & Andersson, 1988
Gallus gallus, eggs	24 days	embryo mortality	LD50	2.3	injected into yolk	Powell et al, 1996b
Maleagris gallopova, 5-d old eggs	19 days	embryo mortality	LD100	60	injected into yolk	Brunström , 1989
Meleagris gallopova, 5-d old eggs	19 days	embryo mortality	LD36	**20**	injected into yolk	Brunström , 1989
Gallus gallus, 4 d old eggs	hatch time	Survival day 11-18, hatching succes	NOEC	**0.3**	injected into air sac	Hoffman et al., 1998
Gallus gallus, 4 d old eggs	hatch time	malformed or edema in embryos	NOEC	<0.3	injected into air sac	Hoffman et al., 1998
Falco sparverius, 6 d old eggs	hatch time	hatching succes	NOEC	23	injected into air sac	Hoffman et al., 1998
Falco sparverius, 6 d old eggs	hatch time	embryos with malformation or edema	NOEC	**0.23**	injected into air sac	Hoffman et al., 1998
Sterna hirundo, 4 d old eggs	hatch time	Survival day 11-18, hatching succes	NOEC	<44	injected into air sac	Hoffman et al., 1998
Sterna hirundo, 4 d old eggs	hatch time	embryos with malformation or edema	NOEC	44	injected into air sac	Hoffman et al., 1998
PCB 153						
Falco sparverius, 1-y old females	28 days	APND activity	ED56	4000	3 oral doses/week	Elliott et al., 1991
Falco sparverius, 1-y old females	28 days	AE activity	ED52	***4000***	3 oral doses/week	Elliott et al., 1991
Gallus gallus, 4-d old eggs	14 days	embryo mortality	LD0	50000	injected into yolk	Brunström, 1991b
Gallus gallus, 7-d old eggs	3 days	EROD activity	ED50	>40000	injected into air sac	Brunström, 1991b
PCB 156						
Gallus gallus, 4-d old eggs	14 days	embryo mortality	LD95	2500	injected into yolk	Brunström, 1991b
Gallus gallus, 7-d old eggs	3 days	EROD activity	ED50	140	injected into air sac	Brunström, 1991b
Gallus gallus, 7-d old eggs	3 days	embryo mortality	LD50	**1500**	injected into air sac	Brunström, 1990
Gallus gallus, 13-d-old eggs	5 days	lymphoid development inhibition	ED50	***1.23***	injected into air sac	Brunström et al., 1990
PCB 157						

species/age	exp. time	parameter	criterion	value (µg/kg w.w.)	remark	reference
Gallus gallus, 4-d old eggs	14 days	embryo mortality	LD90	2500	injected into yolk	Brunström, 1991b
Gallus gallus, 7-d old eggs	3 days	EROD activity	ED50	200	injected into air sac	Brunström, 1991b
Gallus gallus, 7-d old eggs	3 days	embryo mortality	LD50	**2500**	injected into air sac	Brunström, 1990
Gallus gallus, 13-d-old eggs PCB 169	5 days	lymphoid development inhibition	ED50	*1.77*	injected into air sac	Brunström et al., 1990
Gallus gallus, 4-d old eggs	14 days	embryo mortality	LD80	**100**	injected into yolk	Brunström, 1991b
Gallus gallus, 7-d old eggs	3 days	EROD activity	ED50	*14*	injected into air sac	Brunström, 1991b
Gallus gallus, 7-d old eggs	3 days	embryo mortality	LD50	170	injected into air sac	Brunström & Andersson, 1988
Gallus gallus, 13-d-old eggs	5 days	lymphoid development inhibition	ED50	109	injected into air sac	Brunström et al., 1990

Hepatic mixed funtion oxidases:
EROD: 7-ethoxyresorufin-O-deethylase
APND: aminopyrine-N-demethylase
AE: aldrin epoxidase
AHH: arylhydrocarbon hydroxylase

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Appendix II. Bioconcentration factors, biota-to-sediment accumulation factors

II.a.1. Bioconcentration factors (BCFs), laboratory studies, excluding crustaceae

Organism	Exp. period	Cwater (µg/l)	calc. method	fract lipid	BCF wet	BCF fat	Reference
PCB 77							
Dreissena polymorpha, adult	6 h	0.004-1 ng/l	k1/k2	nr	4.7E+4	4.3E+6	Fisher et al. 1992
Brachydanio rerio, 0.243 g	30 d	0.48	k1/k2	0.0292	2.3E+5	7.8E+6	Fox et al. 1994
Poecilia reticulata, 215 mg female	8 d	sat.	k1/k2	0.05	4.2E+4	8.4E+5	Opperhuizen & Voors 1987
Poecilia reticulata, 1 yr male	16 d		k1/k2	nr	5.6E+4	1.3E+6	Opperhuizen & Jongeneel, 1986
PCB 105							
Brachydano rerio, larvae	144 h	0.135	k1/k2			3388441	Petersen & Kristensen (1998)
Gadus morhua, larvae	138 h	0.175	k1/k2			5248074	Petersen & Kristensen (1998)
PCB 118							
Poecilia reticulata, 1 yr males	16 d		k1/k2	nr	5.9E+4		Opperhuizen & Jongeneel 1986
PCB 126							
Brachydanio rerio, 0.243 g	30 d	0.15	k1/k2	0.0292	6.5E+5	2.2E+7	Fox et al. 1994
PCB 153							
Crassostrea virginica					4.8E+4	4.4E+6	Hawker & Connell 1986 (rev)
Dreissena polymorpha, adult	6 h	0.004-1 ng/l	k1/k2	nr	5.6E+5a	5.1E+7	Fisher et al. 1992
Dreissena polymorpha, adult	6 h	0.004-1 ng/l	k1/k2	nr	4.2E+4b	3.8E+6	Fisher et al. 1992
Mytilus edulis	40 d	*27-31			5.0E+5	4.6E+7	Aquapol/Haenen/Pruell et al.1993
Strophitis rugosus (clam)	30d	0.048	Co/Cw	nr	5.8E+3	5.3E+5	Lynch et al. 1982
Brachydanio rerio, 0.243 g	30 d	0.12	k1/k2	0.0292	4.5E+5	1.5E+7	Fox et al. 1994
Cottus bairdi (fish)	30d	0.048	Co/Cw	nr	4.3E+4	9.8E+5	Lynch et al. 1982
Cyprinodon variegatus, 0.1-1 g	30 d	0.58, *20	Co/Cw	nr	2.0E+5	4.5E+6	Lores et al. 1993
Oncorhynchus mykiss, fry	4-5 d	0.081	k1/k2	nr	6.9E+4	1.6E+6	Muir et al. 1985
Pimephales promelas, 0.22 g	5 d	0.046	k1/k2	0.0916	1.5E+6	1.7E+7	Sijm & v.d. Linde 1995

Organism	Exp. period	Cwater (µg/l)	calc. method	fract lipid	BCF wet	BCF fat	Reference
Pimephales promelas, 0.41 g	5 d	0.087	k1/k2	0.0741	1.3E+6	1.7E+7	Sijm & v.d. Linde 1995
Pimephales promelas,1.17 g	5 d	0.14	k1/k2	0.0147	2.2E+4	1.5E+6	Sijm & v.d. Linde 1995
Pimephales promelas, 0.67 g	5 d	0.14	k1/k2	0.0358	4.8E+5	1.3E+7	Sijm & v.d. Linde 1995
Poecilia reticulata, 0.1 g	6 d					6.0E+6	Aquapol/Bruggeman 1984
Poecilia reticulata, 0.1 g	7-13 d		k1/k2	nr	4.5E+5	1.0E+7	Opperhuizen & Schrap, 1987
Poecilia reticulata, 0.1 g	12 d	12	k1/k2	nr		1.4E+6	Aquapol/Gobas 1989
Poecilia reticulata, males 0.1 g	6 d	sat.	k1/k2	0.035	2.1E+5	6.0E+6	Bruggeman 1983 (thesis)
Poecilia reticulata, 1 yr males	16 d		k1/k2	nr	1.0E+5	2.3E+6	Opperhuizen & Jongeneel 1986
PCB 169							
Brachydanio rerio, 0.243 g	30 d	0.052	k1/k2	0.0292	9.3E+5	3.2E+7	Fox et al. 1994

Bruggeman 1983 (thesis): ca. 50% mortality occurred
Hawker & Connell 1986: review, data uit Vreeland et al. 1974
Lynch et al. (1982): model stream ecosystem
When no % fat was reported, conversion factors from SAWES-database/Haenen et al. were used: fish FW=>fat=/0.044 and bivalves FW=>fat=/0.011

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II.a.2. Bioconcentration factors (BCFs), laboratory studies, crustaceae

Organism	Exp. period	Cwater (µg/l)	calc. method	BCF WW	Reference
PCB 77					
Daphnia magna, <24 h	21 d	0.1	Co/Cw	13,200	Dillon et al. 1990
Daphnia magna, <24 h	21 d	1	Co/Cw	11,000	Dillon et al. 1990
Hyalella azteca, 0-1 w	10w	1.34		3,500	Borgmann et al. 1990
Hyalella azteca, 0-1 w	10w	4.8		3,800	Borgmann et al. 1990
Hyalella azteca, 0-1 w	10w	16	Co/Cw	2,400	Borgmann et al. 1990
Hyalella azteca, 0-1 w	10w	55	Co/Cw	2,600	Borgmann et al. 1990
PCB 118					
Daphnia magna, <24 h	21d	0.1	Co/Cw	7,000	Dillon et al. 1990
Daphnia magna, <24 h	21d	1	Co/Cw	26,600	Dillon et al. 1990
PCB 153					
Selenastrum capricornutum				30,200	Richer & Peters,1993
Daphnia magna, <24 h	21d	0.1	Co/Cw	13,000	Dillon et al. 1990
Daphnia magna, <24 h	21d	1	Co/Cw	14,400	Dillon et al. 1990
Mysis relicta	6h		k1/k2	440,000	Evans & Landrum, 1989
Pontoporeia hoyi	6h		k1/k2	100,000	Evans & Landrum, 1989

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II.b. Biomagnification factors (BMFs), laboratory studies

Organism	Exp. period	Cfood, mg/kg	calc. method	BMF, lw	Reference
PCB 77					
Oncorhynchus kisutch, 2.9 g	108 d	3.3	Co/Cfood	4.21	Gruger et al. 1975
PCB 118					
Oncorhynchu mykiss, juv 2-4 g	30 d	0.02	Ef*feedrate/kd	6.00	Fisk et al 1998
PCB 126					
Gasterosteus aculeatus, juv. 2 g	83 d	0.008	Co,fat/Cfood,fat	4.94	Van Bavel et al. 1996
PCB 153					
Dreissena polymorpha, adult	6 h		k1/k2		Fisher et al. 1992
Gasterosteus aculeatus, juv. 2 g	83 d	0.008	Co,fat/Cfood,fat	12.03	Van Bavel et al. 1996
Oncorhynchus kisutch, 2.9 g	108 d	3.3	Co/Cfood	9.21	Gruger et al. 1975
Oncorhynchus kisutch, 11-20 g	72 d	0.018-3.82	Co/Cfood		Gruger et al. 1976
Poecilia reticulata, adult	30 w	6.14	Ef/(k2+km+g)	46.56	Sijm et al. 1992
Poecilia reticulata, adult	30 w	6.14	Co/Cfood	22.46	Sijm et al. 1992
Oncorhynchu mykiss, juv 2-4 g	30 d	0.022	Ef*feedrate/kd	16.00	Fisk et al 1998
PCB 169					
Gasterosteus aculeatus, juv. 2 g	83 d	0.008	Co,fat/Cfood,fat	4.65	Van Bavel et al. 1996

Gruger et al. 1976: 4 concs. in food were applied; BMF decreased with increasing conc. in food; lowest conc. is control with untreated food.
Sijm et al. 1992: BMF is corrected for growth dilution

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II.c. Biota-to-sediment/soil accumulation factors (BSAFs), laboratory studies

Species	Exp. period	conc. in sed/soil (µg/kg dw)	calc. method	fract. oc	fract. lipid	BSAF ww/dw	BSAF lipid/oc	Reference
DATA IN SEDIMENT								
PCB 105								
Macoma nasuta, 25-32 mm	42 d	52.6	(Co/fat)/(Csed/oc)	0.8	0.046		1.63	Boese et al. 1995
Macoma nasuta, 25-32 mm	42 d	43.2	(Co/fat)/(Csed/oc)	1.29	0.046		2.87	Boese et al. 1995
Macoma nasuta, 25-32 mm	42 d	48.8	(Co/fat)/(Csed/oc)	2.48	0.046		3.85	Boese et al. 1995
Macoma nasuta	28 d	1.26	(Co/fat)/(Csed/oc)	0.0084	0.0552		0.22	Ferraro et al. 1991
Macoma nasuta	28 d	70	(Co/fat)/(Csed/oc)	0.0737	0.0552		0.39	Ferraro et al. 1991
PCB 118								
Macoma nasuta, 25-32 mm	42 d	44.2	(Co/fat)/(Csed/oc)	0.8	0.046		2.02	Boese et al. 1995
Macoma nasuta, 25-32 mm	42 d	36.2	(Co/fat)/(Csed/oc)	1.29	0.046		3.28	Boese et al. 1995
Macoma nasuta, 25-32 mm	42 d	41.6	(Co/fat)/(Csed/oc)	2.48	0.046		4.74	Boese et al. 1995
Macoma nasuta	28 d	2.5	(Co/fat)/(Csed/oc)	0.0084	0.0552		0.73	Ferraro et al. 1991
Macoma nasuta	28 d	162	(Co/fat)/(Csed/oc)	0.0737	0.0552		0.54	Ferraro et al. 1991
PCB 153								
Macoma nasuta, 25-32 mm	42 d	46	(Co/fat)/(Csed/oc)	0.8	0.046		1.57	Boese et al. 1995
Macoma nasuta, 25-32 mm	42 d	38.5	(Co/fat)/(Csed/oc)	1.29	0.046		2.66	Boese et al. 1995
Macoma nasuta, 25-32 mm	42 d	44.5	(Co/fat)/(Csed/oc)	2.48	0.046		4.05	Boese et al. 1995
Macoma nasuta	28 d	4	(Co/fat)/(Csed/oc)	0.0084	0.0552		0.71	Ferraro et al. 1991
Macoma nasuta	28 d	112	(Co/fat)/(Csed/oc)	0.0737	0.0552		0.40	Ferraro et al. 1991
Macoma nasuta, 1.9 g	120 d	80.7	(Co/fat)/(Csed/oc)	0.097	0.0152	1.7	1.75	Pruell et al. 1993
Cyprinodon variegatus, 0.1-1 g	30 d	18	Co/Csed	0.06	nr	5.4		Lores et al. 1993
Ictalurus nebulosus, 7-11 g	10 d		k1/k2	0.009	0.098	2.55	0.23	Dabrowska & Fisher, 1993
Ictalurus nebulosus, 7-11 g	10 d		k1/k2	0.009	0.057	5.91	0.93	Dabrowska & Fisher, 1993
Ictalurus nebulosus, 7-11 g	10 d		k1/k2	0.031	0.098	2.41	0.76	Dabrowska & Fisher, 1993

Species	Exp. period	conc. in sed/soil (µg/kg dw)	calc. method	fract. oc	fract. lipid	BSAF ww/dw	BSAF lipid/oc	Reference
Ictalurus nebulosus, 7-11 g	10 d		k1/k2	0.031	0.057	10.92	5.94	Dabrowska & Fisher, 1993
PCB 156								
Macoma nasuta	28 d	0.48	(Co/fat)/(Csed/oc)	0.0084	0.0552		0.61	Ferraro et al. 1991
Macoma nasuta	28 d	34	(Co/fat)/(Csed/oc)	0.0737	0.0552		0.16	Ferraro et al. 1991
DATA IN SOIL								
PCB 118								
Eisenia andrei	110 d	128	Co,ss/Csoil	0.12	0.01	0.37	4.30	Belfroid et al. 1995
PCB 153								
Eisenia andrei	100 d	571	Co,ss/Csoil	0.12	0.01	0.35	4.10	Belfroid et al. 1995
PCB 156								
Eisenia andrei	100 d	38.6	Co,ss/Csoil	0.12	0.01	0.34	3.90	Belfroid et al. 1995

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II.d. Bioconcentration factors (BCFs), field studies

Organism	Exp. period	Cwater (µg/l)	calc. method	BCF ww	BCF lw	Reference
PCB 118						
Mytilus edulis	-	*0.5			1.6E+7	Aquapol/Duursma 1989
Mytilus edulis	-	*19.5			1.3E+7	Aquapol/Duursma 1989
Mytilus edulis	-	*27.5			5.0E+6	Aquapol/Duursma 1989
Mytilus edulis	-	*29.5			4.0E+6	Aquapol/Duursma 1989
PCB 153						
Mytilus edulis	56 d	*30-35	calibr.	7.4E+4	6.6E+6	Aquapol/Haenen 1993
Mytilus edulis	-	*15-35	Co/Cw	4.0E+5	4.2E+7	Aquapol/Haenen 1993
Oncorhynchus mykiss				1.0E+7		Oliver & Niimi, 1985
Platichthys flesus		*15-35	Co/Cw	3.2E+6	7.1E+7	Haenen et al., 1993

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II.e. Biomagnification factors (BMFs), field studies

Organism	BMF	Reference
PCB 77		
fish to otter	2.5	Smit et al. (1996a)
fish diet to otter	1.4	Leonards et al. (1997)
mammal/amphibians to weasel	17	Leonards et al. (subm)
mammal/amphibians to stoat	6	Leonards et al. (subm)
mammal/amphibians to polecat	4	Leonards et al. (subm)
PCB 105		
fish to otter	7.9	Smit et al. (1996a)
fish diet to otter	12	Leonards et al. (1997)
mammal/amphibians to weasel	10	Leonards et al. (subm)
mammal/amphibians to stoat	38	Leonards et al. (subm)
mammal/amphibians to polecat	31	Leonards et al. (subm)
fish to herring gull	27.7	Braune & Norstrom, 1989
PCB 118		
fish to otter	35	Smit et al. (1996a)
fish diet to otter	15	Leonards et al. (1997)
mammal/amphibians to weasel	7	Leonards et al. (subm)
mammal/amphibians to stoat	25	Leonards et al. (subm)
mammal/amphibians to polecat	20	Leonards et al. (subm)
fish to herring gull	22	Braune & Norstrom, 1989
PCB 126		
fish to otter	130	Smit et al. (1996a)
fish diet to otter	70	Leonards et al. (1997)
mammal/amphibians to weasel	20	Leonards et al. (subm)
mammal/amphibians to stoat	112	Leonards et al. (subm)
mammal/amphibians to polecat	31	Leonards et al. (subm)
PCB 153		
fish to otter	28	Smit et al. (1996a)
fish diet to otter	15	Leonards et al. (1997)
mammal/amphibians to weasel	5	Leonards et al. (subm)
mammal/amphibians to stoat	26	Leonards et al. (subm)
mammal/amphibians to polecat	180	Leonards et al. (subm)
Zebra mussel to avocet	9	Hendriks, 1995
zebra mussel to pochard	5	Hendriks, 1995
perch to common tern	6	Hendriks, 1995
zebramussel to pochard	3	Hendriks, 1995
roach to cormorant	32	Hendriks, 1995
PCB 156		
fish to otter	37	Smit et al. (1996a)
fish diet to otter	30	Leonards et al. (1997)
mammal/amphibians to weasel	7	Leonards et al. (subm)
mammal/amphibians to stoat	37	Leonards et al. (subm)
mammal/amphibians to polecat	64	Leonards et al. (subm)
PCB 157		
fish to otter	84	Smit et al. (1996a)
fish diet to otter	19	Leonards et al. (1997)
mammal/amphibians to weasel	3	Leonards et al. (subm)
mammal/amphibians to stoat	22	Leonards et al. (subm)

Organism	BMF	Reference
mammal/amphibians to polecat	58	Leonards et al. (subm)
PCB 169		
fish to otter	108	Smit et al. (1996a)
fish diet to otter	348	Leonards et al. (1997)

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II.f. Biota-to-sediment/soil accumulation factors (BSAFs), field studies

Species	% oc	% fat	BSAF, ww/dw	BSAF, lipid/oc	Reference
PCB 77					
Dreissena polymorpha, 0.15 g	24	1.6		3.32	Leonards et al. (subm.)
Pseudanodonta complanata, 3.8 g	24	0.8		3.21	Leonards et al. (subm.)
pooled fish				1.10	Smit et al. (1996a)
PCB 105					
Dreissena polymorpha, 0.15 g	24	1.6		5.09	Leonards et al. (subm.)
Pseudanodonta complanata, 3.8 g	24	0.8		4.56	Leonards et al. (subm.)
pooled fish				5.50	Smit et al. (1996a)
PCB 118					
Anodonta cygnea	39.8	0.38		3.80	Van Hattum et al. 1992
Pseudanodonta complanata	19.6	0.47		0.51	Van Hattum et al. 1992
Dreissena polymorpha, 0.15 g	24	1.6		7.02	Leonards et al. (subm.)
Pseudanodonta complanata, 3.8 g	24	0.8		3.55	Leonards et al. (subm.)
Mercenaria mercenaria	4.4	nr		1.32	Lake et al. 1990
Yoldia limatula	4.4	nr		4.26	Lake et al. 1990
Yoldia limatula	2.5	nr		5.73	Lake et al. 1990
Anguila anguila	19.6	3.62		4.57	Van Hattum et al. 1992
Anguila anguila	33.6	6.19		4.88	Van Hattum et al. 1992
Anguila anguila	34.7	5.56		5.88	Van Hattum et al. 1992
Abramis brama	33.6	0.43		5.47	Van Hattum et al. 1992
Abramis brama	34.7	0.44		4.00	Van Hattum et al. 1992
Esox lucius	33.6	0.15		5.06	Van Hattum et al. 1992
Esox lucius	34.7	0.08		4.75	Van Hattum et al. 1992
Rutilus rutilus, ca. 5 y	33.6	0.59		4.71	Van Hattum et al. 1992
Rutilus rutilus, ca. 5 y	34.7	0.48		6.63	Van Hattum et al. 1992
Rutilus rutilus, ca.1 y	34.7	1.67		1.75	Van Hattum et al. 1992
Rutilus rutilus, ca. 1 y	30.3	1.6		7.00	Van Hattum et al. 1992
pooled fish				7.10	Smit et al. (1996a)
PCB 126					
Dreissena polymorpha, 0.15 g	24	1.6		4.17	Leonards et al. (subm.)
pooled fish				5.70	Smit et al. (1996a)
PCB 153					
Anodonta cygnea	39.8	0.38		6.75	Van Hattum et al. 1992
Pseudanodonta complanata	19.6	0.47		0.78	Van Hattum et al. 1992
Dreissena polymorpha	9.7	1.74		4.95	Van der Oost et al. 1988
Dreissena polymorpha, 0.15 g	24	1.6		0.00	Leonards et al. (subm.)
Pseudanodonta complanata, 3.8 g	24	0.8		6.36	Leonards et al. (subm.)
Mercenaria mercenaria	4.4	nr		1.49	Lake et al. 1990
Yoldia limatula	4.4	nr		5.10	Lake et al. 1990
Yoldia limatula	2.5	nr		8.25	Lake et al. 1990
Anguila anguila	9.7	14.9		14.34	Van der Oost et al. 1988
Anguila anguila	19.6	3.62		5.97	Van Hattum et al. 1992
Anguila anguila	33.6	6.19		5.87	Van Hattum et al. 1992
Anguila anguila	34.7	5.56		7.69	Van Hattum et al. 1992
Abramis brama	33.6	0.43		9.23	Van Hattum et al. 1992
Abramis brama	34.7	0.44		5.38	Van Hattum et al. 1992

Species	% oc	% fat	BSAF, ww/dw	BSAF, lipid/oc	Reference
Esox lucius	33.6	0.15		4.20	Van Hattum et al. 1992
Esox lucius	34.7	0.08		3.92	Van Hattum et al. 1992
Rutilus rutilus, ca. 5 y	33.6	0.59		6.43	Van Hattum et al. 1992
Rutilus rutilus, ca. 5 y	34.7	0.48		10.08	Van Hattum et al. 1992
Rutilus rutilus, ca.1 y	34.7	1.67		2.69	Van Hattum et al. 1992
Rutilus rutilus, ca. 1 y	30.3	1.6		11.33	Van Hattum et al. 1992
pooled fish				10.00	Smit et al. (1996a)
PCB 156					
Dreissena polymorpha, 0.15 g	24	1.6		5.00	Leonards et al. (subm.)
Pseudanodonta complanata, 3.8 g	24	0.8		3.22	Leonards et al. (subm.)
pooled fish				6.10	Smit et al. (1996a)
PCB 157					
Dreissena polymorpha, 0.15 g	24	1.6		20.00	Leonards et al. (subm.)
Pseudanodonta complanata, 3.8 g	24	0.8		10.35	Leonards et al. (subm.)
pooled fish				3.00	Smit et al. (1996a)
PCB 169					
pooled fish				2.50	Smit et al. (1996a)
Remarks					
DATA IN SOIL					
PCB 118					
Lumbricus rubellus	5	1.19	0.21		Hendriks et al., 1995
PCB 153					
Lumbricus rubellus	5	1.19	0.16		Hendriks et al., 1995
Lumbricus rubellus	9	1.23	0.05		Hendriks et al., 1995
Remark					

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II.g. Impression on extractable lipid content in various species and their eggs as percentage of wet weight

Species	lipid % whole body, mean±std	lipid % egg, mean±std	reference
Birds			
Larus argentatus	10.3±2.2	7.7±0.8	Braune& Norstrom, 1989
Larus argentatus		8.2-9.9	Koslowski et al. 1994
Aythya fuligula	3.6±0.7		Zimmerman et al. 1997
Podiceps cristatus	4.2±0.5		Zimmerman et al. 1997
Ardea cinerea	2.7±1.5		Zimmerman et al. 1997
Phalacrocorax carbo sinensis	3.5±0.5		Zimmerman et al. 1997
Phalacrocorax carbo sinensis		3.5-4.5	Dirksen et al. 1995, values are means for different colonies
Birds	8.9±9.0		Jongbloed et al. 1994[a]
Altricials		5.9±1.5	Carey et al., 1980[a]
Semi-altricials		6.3±0.7	Carey et al., 1980[a]
Semi-precocials		9.5±2.3	Carey et al., 1980[a]
Precocials		10.3±1.4	Carey et al., 1980[a]
Mammals			
Martes Martes (muscular tissue)	3.4±1.8		Bremle et al., 1997
Stoat (liver)	4.1±0.7		Leonards, 1997b
Weasel (liver)	3.2±0.6		Leonards, 1997b
Polecat (liver)	5.4±2.0		Leonards, 1997b
Red vole	2.9±0.5		Leonards, 1997b
Wood mouse	4.6		Leonards, 1997b
Common vole	2.7		Leonards, 1997b
Shrew	2.7		Leonards, 1997b
Common hare	0.95		Leonards, 1997b
Mammals	9.4±7.7		Jongbloed et al. 1994[a]
Fish			
Silver bass		5.5	Koslowski et al. 1994
Smallmouth bass		11.4	Koslowski et al. 1994
Walleye		10.1±0.2	Fisk et al. 1997

[a]Based on compilation of literature data

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Appenidix III. Physico-chemical data

III.A. WATER SOLUBILITY
(measured values, derived from AQUAPOL)

compound	value mg/l	water system	method	remark	reference
PCB77	4.35 E-4	F	generator column		Dickhut, 1986
	5.50 E-4	F	generator column		Dunnivant, 1988
	1.80 E-3	F	generator column		Opperhuizen, 1989
	7.50 E-4	F	generator column		von Weil., 1974
	9.80 E-4	F	RP-HPLC-column	average of 4 meas.	Brodsky, 1988
PCB105	9.80 E-3	F	RP-HPLC-column	average of 4 meas.	Brodsky, 1988
PCB118	1.28 E-3	M	shake-flask	Aroclor 1254 used	Wiese, 1978
	1.34 E-2	F	shake-flask	Aroclor 1254 used	Murphy, 1987
	1.50 E-2	F	RP-HPLC-column	average of 4 meas.	Brodsky, 1988
PCB153	8.60 E-4	F	generator column		Dunnivant , 1988
	1.15 E-3	F	generator column		Opperhuizen, 1989
	9.53 E-4	F	shake-flask		Haque, 1975
	1.20 E-3	F	generator column		von Weil, 1974
	7.9 E-4	M	shake-flask	Aroclor 1254 used	Wiese, 1978
	9.14 E-3	F	shake flask	Aroclor 1254 used	Murphy, 1987
	9.50 E-4	F	shake-flask		Chiou, 1977
	1.20 E-3	F	RP-HPLC-column	average of 4 meas..	Brodsky, 1988
PCB169	5.10 E-4	F	RP-HPLC-column	average of 4 meas.	Brodsky, 1988

III.B. Logarithms of the *n*-octanol/water partition voefficients
(measured values, derived from AQUAPOL)

compound	value	water system	method	remark	reference
PCB77	6.63	F	slow-stirring		de Bruijn, 1991
	5.62	F	RP column	IPC-18 column	Rapaport, 1984
	6.21	F	generator column		Hawker, 1988
	6.12	F	RP column	C18-waters column	de Kock, 1987
	6.37	F	RP column	sepralyte diphenyl column	Brodsky, 1988
	6.40	F	RP column	sepralyte diphenyl column	Brodsky, 1988
PCB105	5.82	F	generator column		Hawker, 1988
	7.14	F	HPLC	sepralyte diphenyl column	Brodsky, 1988
	6.41	F	HPLC	nucleosil SC18 column	Brodsky, 1988
	6.93	F	HPLC	sepralyte diphenyl column	Brodsky, 1988
	6.68	F	HPLC	nucleoside SC18 column	Brodsky, 1988
PCB118	7.12	F	HPLC	IPC18-column	Rapaport, 1984
	6.57	F	HPLC	nucleosil 5C18 column	Brodsky, 1988
PCB153	6.90	F	generator column		Doucette, 1987
	6.68	F	HPLC	ambient temp. ultrasphere ODS-column used	Burkhard, 1985
	7.07	F	HPLC	supercosil LC-18 column	Risby, 1990
	7.12	F	HPLC	VYDAC 204 TPS column	Risby, 1990
	7.75	F	HPLC	IPC 18 column	Rapaport, 1984
	6.72	F	shake-flask		Chiou, 1977
	7.71	F	HPLC	C18 waters column	de Kock, 1987
	6.71	F	HPLC	sepralyte diphenyl column	Brodsky, 1988
	6.84	F	HPLC	nucleosil 5C18 column	Brodsky, 1988
	6.74	F	HPLC	sepralyte diphenyl column	Brodsky, 1988
	6.90	F	HPLC	nucleosil 5C18 column	Brodsky, 1988
	6.75	F	HPLC	apolane-87 GC-column, based upon distribution constant data with capillary GLC	Risby, 1990
	6.75	F	HPLC	apolane-87 GC-column, based upon relative retention data with capillary GLC	Risby, 1990
PCB 169	7.41	F	slow-strirring		de Bruijn, 1991
	7.55	F	RP-HPLC column	VYDAC 201 TPS-column	Risby, 1990
	7.39	F	RP-HPLC-column	supercosilLC-18 column	Risby, 1990
	7.54	F	RP-HPLC column	nucleosil SC18- column	Brodsky, 1988
	7.61	F	RP-HPLC-column	sepralyte diphenyl column	Brodsky, 1988
	7.42	F	RP-HPLC column	nucleosil SC18- column	Brodsky, 1988
	7.62	F	RP-HPLC-column	sepralyte diphenyl column	Brodsky, 1988
	7.42	F	HPLC	apolane-87 GC-column, based upon distribution constant data with capillary GLC	Risby, 1990
	7.46	F	HPLC	apolane-87 GC-column, based upon relative corrected retention data with capillary GLC	Risby, 1990

III.C. Logarithms of the organic carbon/water partition coefficient
(measured values, derived from AQUAPOL)

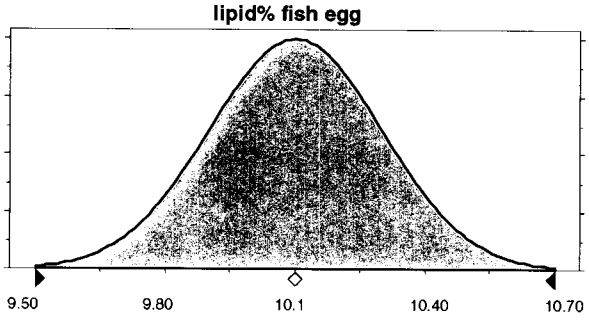
compound	value	water system	method	remark	reference
PCB77	5.78	F	batch, soil	1.87% oc	Cortes, 1991
	6.31	F	apparent part. coeff. sediment		Brannon, 1991
PCB105	5.81	F	batch, soil	1.87% oc	Paya-Perez, 1991
	6.10	M	batch, sediment	4.00% oc	Duursma, 1989
	6.10	B	batch, sediment	3.00% oc	Duursma, 1989
	6.40	M	batch, sediment	2.10% oc	Duursma, 1989
	8.09	M	co-solvent, sediment	3.01% oc	Evers, 1993
	6.62	M	batch, sediment	3.09% oc	Evers, 1993
PCB118	5.81	F	batch, soil	1.87% oc	Paya-Perez, 1991
	6.50	M	batch, sediment	4.00% oc	Duursma, 1989
	6.20	M	batch, sediment	2.10% oc	Duursma, 1989
	7.00	F	batch, sediment	3.40% oc	Duursma, 1989
	6.40	B	batch, sediment	3.00% oc	Duursma, 1989
	7.69	M	co-solvent, sediment	3.09% oc	Evers, 1993
	6.64	M	batch, sediment	3.09% oc	Evers, 1993
PCB126	5.92	F	batch, soil	0.03-1.87% oc	Cortes, 1991
PCB153	6.76	F	field, susp. matter	3.20 % oc	Eadie, 1990
	5.86	F	batch, soil	0.03-1.87 % oc	Paya-Perez, 1991
	6.48	F	batch, susp. matter		Swackhamer, 1987
	5.86	F	batch, soil		Cortes, 1991
	6.80	F	batch, susp. matter		Lau, 1989
	6.61	F	batch, sediment	2.80% oc	Di Toro, 1985
	6.27	F	batch, sediment	4.80% oc	Oliver, 1985
	6.30	F	batch, sediment	4.80% oc	Oliver, 1985
	6.03	F	batch, sediment	4.80% oc	Oliver, 1985
	6.04	F	apparent part. coeff. sediment		Brannon, 1991
	6.18	M	batch, sediment	0.50% oc	Lara, 1990
	6.13	M	batch, sediment	1.47% oc	Lara, 1990
	5.99	M	batch, sediment	2.23% oc	Lara, 1990
	6.31	F	batch, sediment	3.00% oc	Opperhuizen, 1989
	8.08	M	co-solvent, sediment	3.09% oc	Evers, 1993
	7.72	F	gas purge, sediment	1.21% oc	Coates, 1986
	6.81	M	batch, sediment	0.31% oc	Lara, 1990
	6.54	M	batch, sediment	3.09% oc	Evers, 1993
PCB169	5.92	F	batch, soil	0.03-1.87% oc	Cortes, 1991

In Bockting et al. (1993) 40 experimental log Koc-values for PCB153 are given ranging from 4.25 to 6.90 (average 5.65)

Appenidx IV. Details of the simulations

IV.a. Distributions that were used in the calculations for all congeners

Lipid% of fish eggs

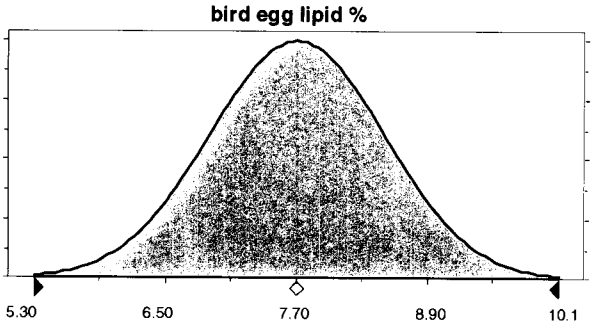


Normal distribution with parameters:

Mean	10.10
Standard Dev.	0.20

Selected range is from -0.00 to +Infinity
Mean value in simulation was 10.10

Lipid% of bird eggs



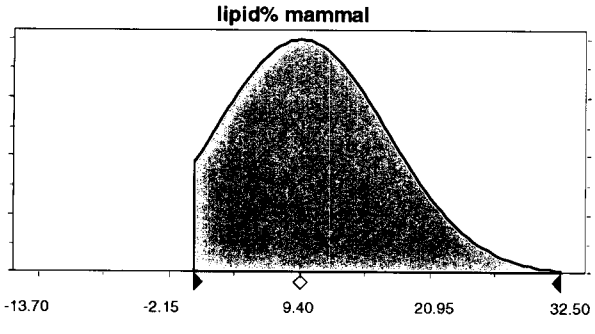
Normal distribution with parameters:

Mean	7.70
Standard Dev.	0.80

Selected range is from -0.00 to +Infinity
Mean value in simulation was 7.70

Lipid% of mammals

Two alternative distributions were used in the calculations:

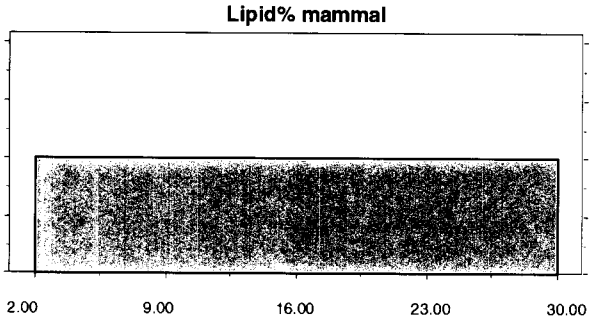


Normal distribution with parameters:

Mean	9.40
Standard Dev.	7.70

Selected range is from 0.00 to +Infinity
Mean value in simulation was 11.04

or

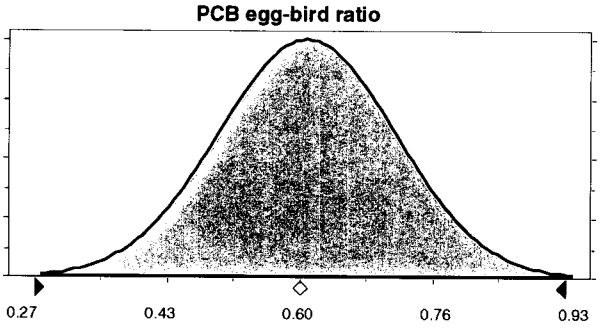


Uniform distribution with parameters:

Minimum	2.00
Maximum	30.00

Mean value in simulation was 16.00

Bird egg-to-whole body ratio

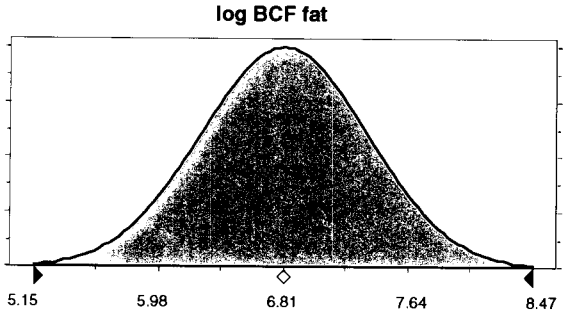


Normal distribution with parameters:

Mean	0.60
Standard Dev.	0.11

Selected range is from -0.00 to +Infinity
Mean value in simulation was 0.60

Bioconcentration factor



Normal distribution with parameters:

Mean	6.81
Standard Dev.	0.55

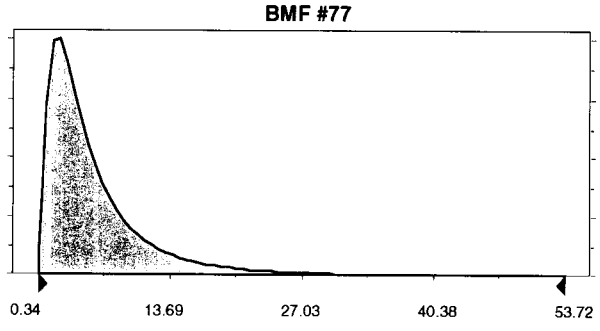
Selected range is from 0.00 to +Infinity
Mean value in simulation was 6.81

IV.b. PCB 77

Toxicity data

Species	Criterion	Endpoint		Unit	Reference	Formula for translation to ωg/kg o.c.
Salvelinus namaycush, eggs	LD50		29	mg/kg egg	Zabel et al., 1995	(LD50/fish egg lipid%/BSAF)*1000
Daphnia magna, < 24 h	NOEC	growth	0.1	ωg/l	Dillon et al. 1990	(BCF/BSAF)*NOEC
Oncorhynchus mykiss, eggs	LD50		1348	ng/g egg	Walker & Peterson, 1991	1000*LD50/(fish egg lipid%*BSAF)
mouse	NOEC	average malformed fetuses per litter	6.86	mg/kg	Marks et al., 1989	NOEC/(mammal lipid%*BMF*BSAF) *1000
Falco sparverius, 4-d-old eggs	LD50	embryo mortality	688	ωg/kg	Hoffman et al, 1996	LD50/(bird egg lipid%*EBR*BMF*BSAF)
Meleagris gallopova, 5-d old eggs	LD60	embryo mortality	1000	ωg/kg	Brunström, 1991	LD60/(bird egg lipid%*EBR*BMF*BSAF)
Gallus gallus, 4 d old eggs	NOEC	Survival day 11-18, hatchin succes, molformed or edema	1.2	ωg/kg	Hoffman et al. 1998	NOEC/(bird egg lipid%*EBR*BMF*BSAF)

Distribution for BMF, PCB 77



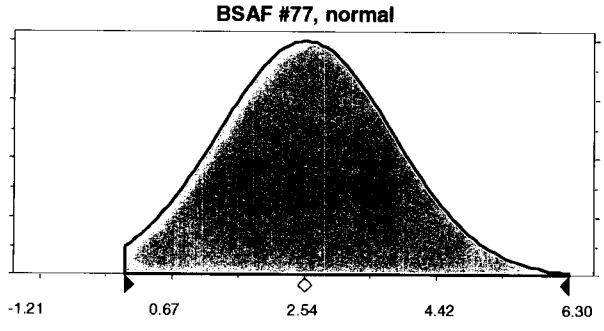
Lognormal distribution with parameters:

Mean	6.10
Standard Dev.	6.22

Selected range is from 0.00 to +Infinity
Mean value in simulation was 6.12

Distribution for BSAF, PCB 77

Two alternatives were used:

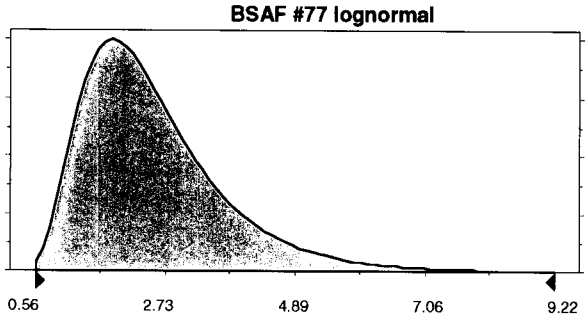


Normal distribution with parameters:

Mean	2.54
Standard Dev.	1.25

Selected range is from -0.00 to +Infinity
Mean value in simulation was 2.61

or



Lognormal distribution with parameters:

Mean	2.54
Standard Dev.	1.25

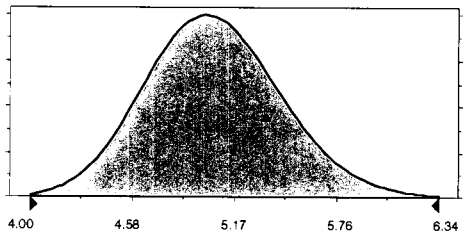
Selected range is from 0.00 to +Infinity
Mean value in simulation was 2.54

IV.c. PCB 105

Toxicity data

Species	Criterion	Endpoint		Unit	Reference	Formula for translation to ωg/kg o.c.
Zebra fish, larvae	EC50		1.3	µg/l	Petersen et al. (subm.)	(CF/BSAF)*EC50
rat (immature male)	ED50	body weight gain	245	mg/kg	Safe 1987	ED50/(mammal lipid%*BMF*BSAF)*1000
Gallus gallus, day 0 eggs	LOAEL	posthatch body weight	300	ug/kg	Powell et al., 1996	LOAEL/(bird egg lipid%*EBR*BMF*BSAF)

Distribution for BMF, PCB 105

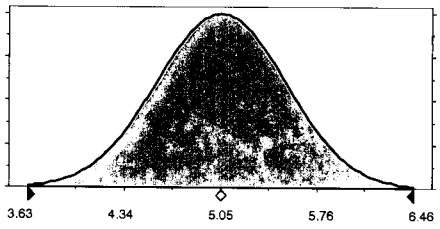


Lognormal distribution with parameters:

Mean	21.33
Standard Dev.	14.30

Selected range is from 0.00 to +Infinity
Mean value in simulation was 21.29

Distribution for BSAF, PCB 105

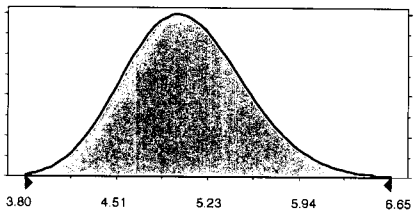


Normal distribution with parameters:

Mean	5.05
Standard Dev.	0.47

Selected range is from 0.00 to +Infinity
Mean value in simulation was 5.05

or



Lognormal distribution with parameters:

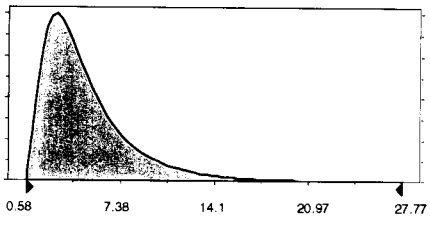
Mean	5.05
Standard Dev.	0.47

Selected range is from 0.00 to +Infinity
Mean value in simulation was 5.05

IV.d. PCB 118

Species	Criterion	Endpoint		unit	Reference	formula
rat (Sprague-Dawley, male)	NOEC	body weight gain	0.67	mg/kg	Chu et al. 1995	NOEC/(mammal lipid%*BMF*BSAF)*1000
Gallus gallus, 4-d old eggs	LD45	embryo mortality	5000	µg/kg	Brunström, 1991	LD45/(bird egg lipid%*EBR*BMF*BSAF)

Distribution for BMF, PCB 118

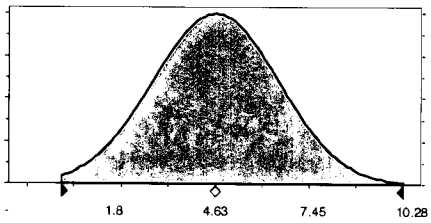


Lognormal distribution with parameters:

Mean	4.94
Standard Dev.	3.55

Selected range is from 0.00 to +Infinity
Mean value in simulation was 4.94

Distribution for BSAF, PCB 118

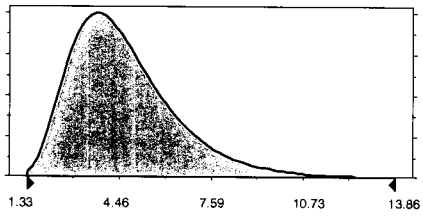


Normal distribution with parameters:

Mean	4.63
Standard Dev.	1.88

Selected range is from -0.00 to +Infinity
Mean value in simulation was 4.67

or



Lognormal distribution with parameters:

Mean	4.63
Standard Dev.	1.88

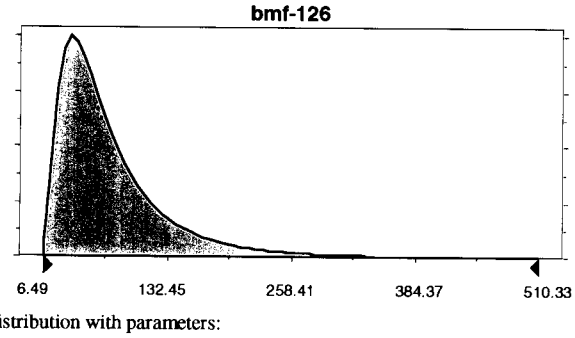
Selected range is from 0.00 to +Infinity
Mean value in simulation was 4.63

IV.e. PCB 126

Toxicity data

Species	Criterion	Endpoint		Unit	Reference	formula
Oryzias latipes, 1-2 h eggs	NOEC	mortality	0.018	ug/l	Harris et al., 1994	(BCF/BSAF)*NOEC
Oncorhynchus mykiss, eggs	LD50	mortality	74	ng/g egg	Walker & Peterson, 1991	(LD50/fish egg lipid%/BSAF)*1000
rat (Sprague-Dawley, male)	NOEC	body weight	0.079	mg/kg	Chu et al., 1994	NOEC/(mammal lipid%*BMF*BSAF)*1000
Meleagris gallopova, 5-d old eggs	LD36	embryo mortality	20	ug/kg	Brunström, 1989	LD36/(bird egg lipid%*EBR*BMF*BSAF)
Gallus gallus, 4 d old eggs	NOEC	Survival day 11-18, hatchin succes	0.3	ug/kg	Hoffman et al. 1998	NOEC/(bird egg lipid%*EBR*BMF*BSAF)
Falco sparverius, 6 d old eggs	NOEC	embryos with malformation or edema	0.23	ug/kg	Hoffman et al. 1998	NOEC/(bird egg lipid%*EBR*BMF*BSAF)

Distribution for BMF, PCB 126



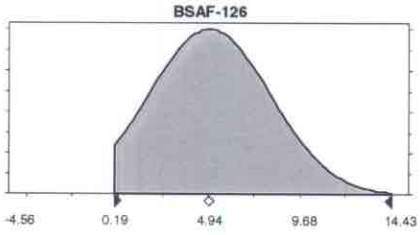
Lognormal distribution with parameters:

Mean	74.99
Standard Dev.	62.63

Selected range is from 0.00 to +Infinity

Mean value in simulation was 74.73

Distribution for BSAF, PCB 126

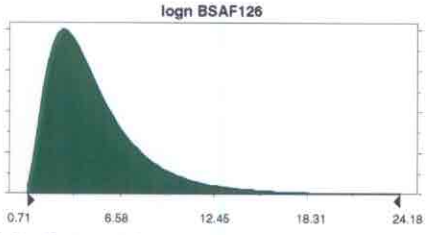


Normal distribution with parameters:

Mean	4.94
Standard Dev.	3.17

Selected range is from -0.00 to +Infinity
Mean value in simulation was 5.33

or



Lognormal distribution with parameters:

Mean	4.94
Standard Dev.	3.17

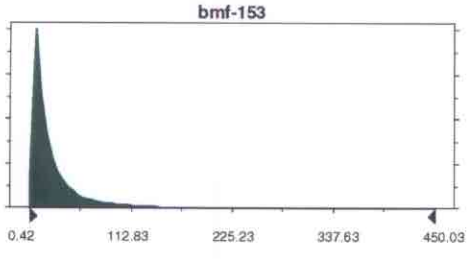
Selected range is from 0.00 to +Infinity
Mean value in simulation was 4.93

IV.f. PCB 153

Toxicity data

Species	Criterion	Endpoint		Unit	Reference	formula
rat (Sprague-Dawley)	LOEC	pup weight	339	mg/kg	Ness et al., 1993	LOEC/(mammal lipid%*BMF*BSAF)*1000
mink (female)	LOEC	body weight gain	9.62	mg/kg	Aulerich et al., 1985	LOEC/(mammal lipid%*BMF*BSAF)*1000

Distribution for BMF, PCB 153

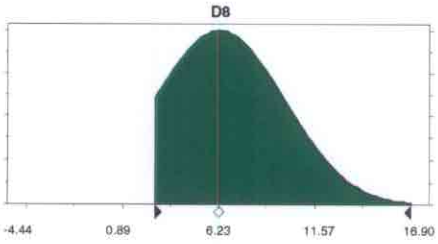


Lognormal distribution with parameters:

Mean	27.12
Standard Dev.	45.78

Selected range is from 0.00 to +Infinity
Mean value in simulation was 27.09

Distribution for BSAF, PCB 153

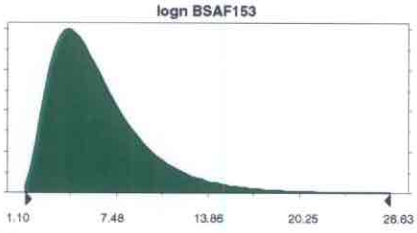


Normal distribution with parameters:

Mean	6.23
Standard Dev.	3.56

Selected range is from 2.67 to +Infinity
Mean value in simulation was 7.25

or



Lognormal distribution with parameters:

Mean	6.23
Standard Dev.	3.56

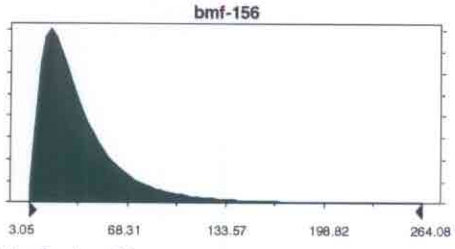
Selected range is from 0.00 to +Infinity
Mean value in simulation was 6.23

IV.g. PCB 156

Toxicity data

Species	Criterion	Endpoint		Unit	Reference	formula
mouse	LOEC	number of live fetuses	40	mg/kg	Birnbaum et al. 1985	ED50/(mammal lipid%*BMF*BSAF)*1000
rat (Sprague-Dawley)	NOEC	body weight	4.87	mg/kg	van Birgelen et al, 1994	NOEC/(mammal lipid%*BMF*BSAF)*1000
Gallus gallus, 7-d old eggs	LD50	embryo mortality	1500	ug/kg	Brunström, 1990	LD50/(bird egg lipid%*EBR*BMF*BSAF)

Distribution for BMF, PCB 156

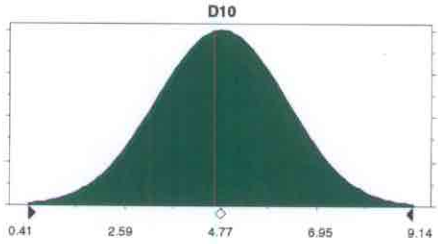


Lognormal distribution with parameters:

Mean	37.41
Standard Dev.	32.15

Selected range is from 0.00 to +Infinity
Mean value in simulation was 37.32

Distribution for BSAF, PCB 156

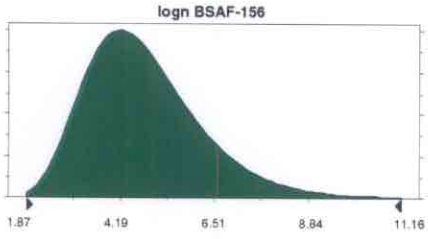


Normal distribution with parameters:

Mean	4.77
Standard Dev.	1.45

Selected range is from -0.00 to +Infinity
Mean value in simulation was 4.78

or



Lognormal distribution with parameters:

Mean	4.77
Standard Dev.	1.45

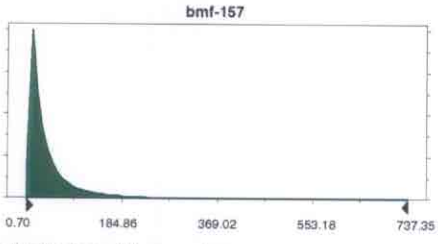
Selected range is from 0.00 to +Infinity
Mean value in simulation was 4.77

IV.h. PCB 157

Toxicity data

Species	Criterion	Endpoint		Unit	Reference	formula
rat (immature male)	ED50	body weight gain	80	mg/kg	Safe, 1987	NOEC/(mammal lipid%*BMF*BSAF)*1000
Gallus gallus, 7-d old eggs	LD50	embryo mortality	2500	ug/kg	Brunström, 1990	LD50/(bird egg lipid%*EBR*BMF*BSAF)

Distribution for BMF, PCB 157

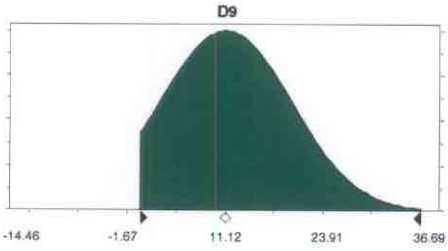


Lognormal distribution with parameters:

Mean	44.57
Standard Dev.	75.04

Selected range is from 0.00 to +Infinity
Mean value in simulation was 44.60

Distribution for BSAF, PCB 157

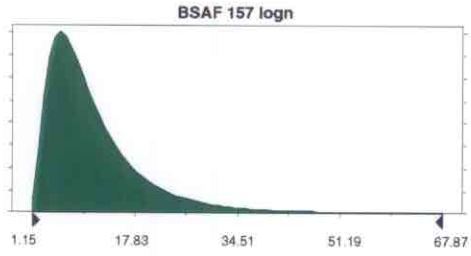


Normal distribution with parameters:

Mean	11.12
Standard Dev.	8.53

Selected range is from 0.00 to +Infinity
Mean value in simulation was 12.73

or



Lognormal distribution with parameters:

Mean	11.12
Standard Dev.	8.53

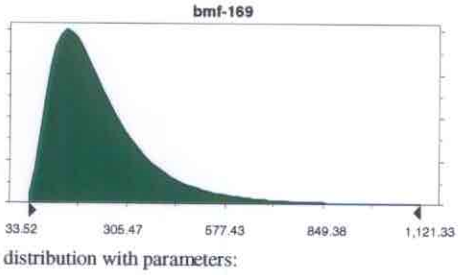
Selected range is from 0.00 to +Infinity
Mean value in simulation was 11.12

IV.i. PCB 169

Toxicity data

Species	Criterion	Endpoint		Unit	Reference	formula
mouse	NOEC	number of live fetuses	1	mg/kg	Marks et al., 1981	NOEC/(mammal lipid%*BMF*BSAF)*1000
rat	NOEC	pup weight, mean litter size	0.6	mg/kg bw/day	Smits-van Prooije et al., 1993	NOEC/(mammal lipid%*BMF*BSAF)*1000
guinea pig	LD50	mortality	0.5	mg/kg	EHC 140, 1993	LD50/(mammal lipid%*BMF*BSAF)*1000
mink (female)	LOEC	body weight loss	0.241	mg/kg	Aulerich et al., 1987	LOEC/(mammal lipid%*BMF*BSAF)*1000
Gallus gallus, 4-d old eggs	LD80	embryo mortality	100	ug/kg	Brunström, 1991	LD80/(bird egg lipid%*EBR*BMF*BSAF)

Distribution for BMF, PCB 169

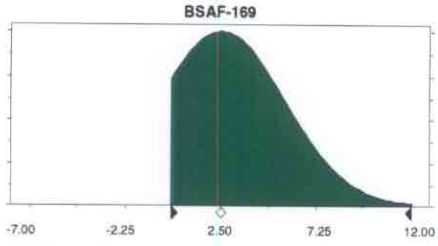


Lognormal distribution with parameters:

Mean	230.05
Standard Dev.	146.97

Selected range is from 0.00 to +Infinity
Mean value in simulation was 229.71

Distribution for BSAF, PCB 169

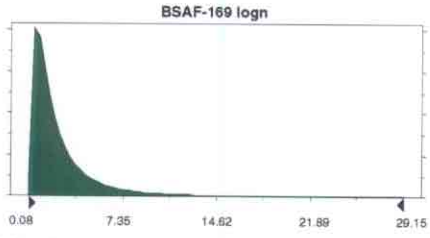


Normal distribution with parameters:

Mean	2.50
Standard Dev.	3.17

Selected range is from -0.00 to +Infinity
Mean value in simulation was 3.68

or



Lognormal distribution with parameters:

Mean	2.50
Standard Dev.	3.17

Selected range is from 0.00 to +Infinity
Mean value in simulation was 2.49

Appenidix V: Monitoring data in sediments from fresh and marine aquatic ecosystems in the Netherlands compared with MPCs for PCB congeners #105, #118, #153 and the mixture-MPC.

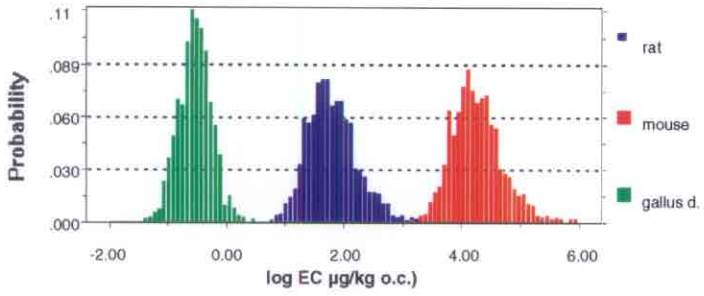
Ameland 20 km	960718	4.88	0.2	4.10	0.51	0.7	14.34	0.36	0.8	16.39	0.41	26.44
Amsterdam NZKanaal	961015	4.88		0.00		4	81.97	2.05	9	184.43	4.61	297.46
Appelzak 4km	960618	0.9	0.3	33.33	4.12	0.6	66.67	1.67	1	111.11	2.78	179.21
Balgzand wetwal zuid	961023	4.28	0.7	16.36	2.02	1.9	44.39	1.11	3.6	84.11	2.10	135.66
Ballumerbocht	960910	2.12	0.5	23.58	2.91	1	47.17	1.18	1.6	75.47	1.89	121.73
Boontjes oostoever	961009	1.8	0.4	22.22	2.74	1	55.56	1.39	1.8	100.00	2.50	161.29
Borndiep zuidwest	960925	3.74	0.6	16.04	1.98	1.4	37.43	0.94	2.2	58.82	1.47	94.88
Bovensluis	911010	4.4		0.00		12	272.73	6.82	38	863.64	21.59	1392.96
Dantziggat kwelder buiten	961009	1.3	0.3	23.08	2.85	0.7	53.85	1.35	1.3	100.00	2.50	161.29
Dantziggat zuid	960910	3.01	0.4	13.29	1.64	1.2	39.87	1.00	2	66.45	1.66	107.17
Den Oever sluis buiten	961024	4.55	1.2	26.37	3.26	3.8	83.52	2.09	7.2	158.24	3.96	255.23
De Gijster inlaat	911010	4.5		0.00		9	200.00	5.00	32	711.11	17.78	1146.95
Griend Kwelder	960911	2.65	0.4	15.09	1.86	1.3	49.06	1.23	2.7	101.89	2.55	164.33
Hammen Oost	961015	2.3	0.4	17.39	2.15	1.4	60.87	1.52	2.2	95.65	2.39	154.28
Hansweert	961017	2.35	0.5	21.28	2.63	2.2	93.62	2.34	4.8	204.26	5.11	329.44
Haringvliet Boei	911010	2.52		0.00		4	158.73	3.97	11	436.51	10.91	704.05
Haringvliet 2km	961010	2.6	1.2	46.15	5.70	4.9	188.46	4.71	12	461.54	11.54	744.42
Ijmuiden 1	960703	3.16	0.8	25.32	3.13	2.6	82.28	2.06	4.8	151.90	3.80	245.00
Koffiebonenplaat	960925	3.43	0.3	8.75	1.08	1	29.15	0.73	1.5	43.73	1.09	70.54
Kornwerderzand Buiten sluis	961021	4.06	0.7	17.24	2.13	2.2	54.19	1.35	3.8	93.60	2.34	150.96
Lauwersoog	961018	1.95	0.4	20.51	2.53	1.4	71.79	1.79	2	102.56	2.56	165.43
Lobith ponton	961009	3.14		0.00		20	636.94	15.92	32	1019.11	25.48	1643.72
Malzwin zuidwal	961024	2.85	0.3	10.53	1.30	1	35.09	0.88	2	70.18	1.75	113.19
Oestergronden 19	960716	2.75 <	0.2	7.27	0.90 <	0.2	7.27	0.18	0.2	7.27	0.18	11.73
Oestergronden 21	960716	2.07 <	0.2	9.66	1.19	0.2	9.66	0.24	0.3	14.49	0.36	23.38

Rottemerplaat kwelder zuid	960905	2.72		0.3	11.03	1.36		0.9	33.09	0.83		1	36.76	0.92	59.30
Rottemerplaat 3	960717	3.89		0.4	10.28	1.27		1	25.71	0.64		1.8	46.27	1.16	74.63
Schiermonnikoog 40	960717	2.69		0.2	7.43	0.92		0.4	14.87	0.37		0.9	33.46	0.84	53.96
Schouwen 10	960618	3.38	<	0.2	5.92	0.73		0.6	17.75	0.44	<	0.2	5.92	0.15	9.54
Steenbergen	961008	4.9			0.00		<	5	102.04	2.55		3	61.22	1.53	98.75
Terschelling 100	960627	1.24	<	0.2	16.13	1.99	<	0.2	16.13	0.40		0.2	16.13	0.40	26.01
Terschelling 135	960627	1.91	<	0.2	10.47	1.29	<	0.2	10.47	0.26	<	0.2	10.47	0.26	16.89
Terschelling 178	960627	2.05	<	0.2	9.76	1.20		0.2	9.76	0.24		0.2	9.76	0.24	15.74
Terschelling 20	960627	2.8		0.3	10.71	1.32		0.7	25.00	0.63		1	35.71	0.89	57.60
Terschelling 4	960627	4.39		0.3	6.83	0.84		1.1	25.06	0.63		2	45.56	1.14	73.48
Terschelling	961206	1.58	<	0.2	12.66	1.56		0.5	31.65	0.79		0.8	50.63	1.27	81.67
Texel 1	960711	3.27		0.3	9.17	1.13		1	30.58	0.76		1.9	58.10	1.45	93.72
Vlakte van Kerken Schorren noord	961023	2.16		0.3	13.89	1.71		1	46.30	1.16		1.8	83.33	2.08	134.41
Vlakte van Oostbierum	961009	4.67		0.5	10.71	1.32		1.8	38.54	0.96		3	64.24	1.61	103.61
Voordelta 4	960822	2.22		0.5	22.52	2.78		1.8	81.08	2.03		3.9	175.68	4.39	283.35
Walcheren 30	960625	3.71		1	26.95	3.33		2.8	75.47	1.89		3.2	86.25	2.16	139.12
Wolderwijd	961016	0.47			0.00		<	1	212.77	5.32	<	1	212.77	5.32	343.17
Zuidhaven	911010	2.02			0.00			7	346.53	8.66		14	693.07	17.33	1117.85

Appenidx VI. Information on levels in organic carbon related to biochemical and histopathological effects in mammals and birds

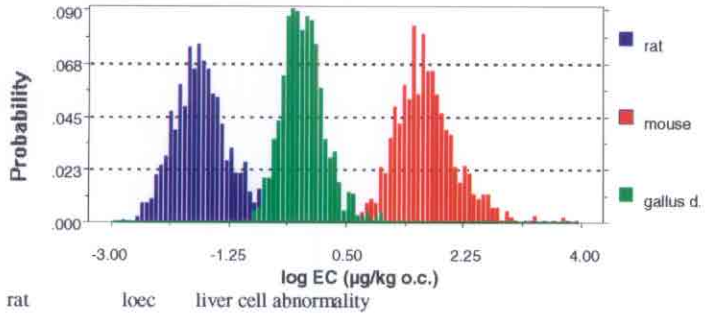
See main text (7.3) for explanation.

PCB 105



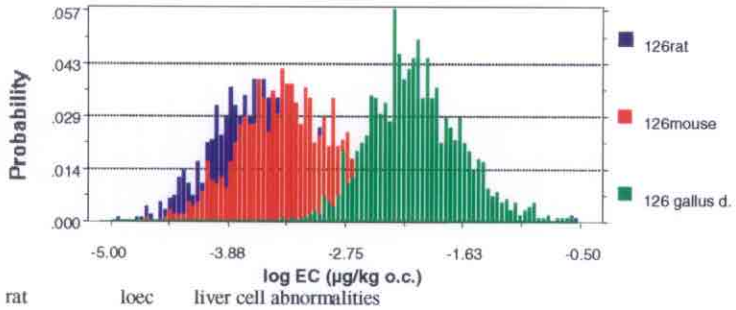
rat	ed50	plasma thyroxin level in pup
mouse	ed50	EROD
gallus d.	ed50	lympoid development

PCB 118

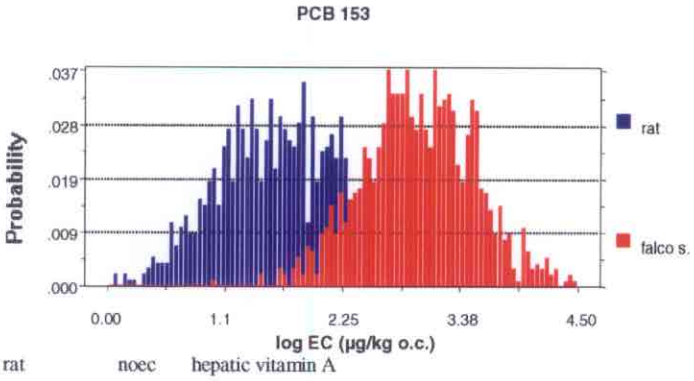


rat	loec	liver cell abnormality
mouse	noec	hepatic porphyrin
gallus d.	ed50	lympoid developmetn

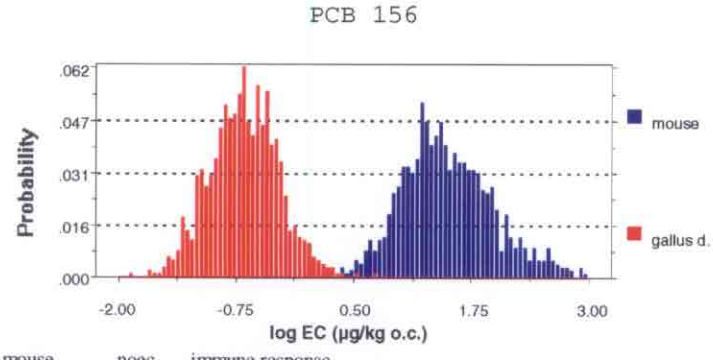
PCB 126



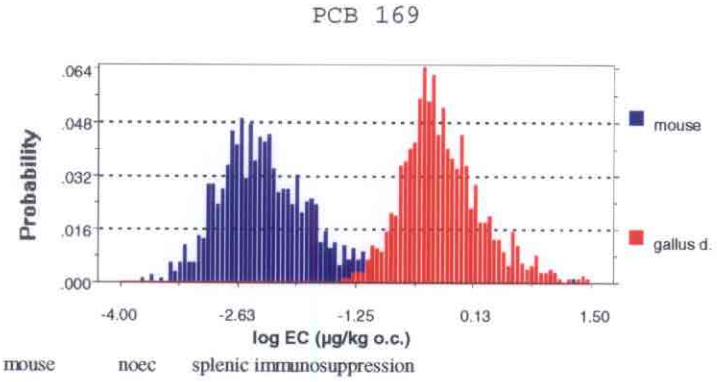
rat	loec	liver cell abnormalities
mouse	noec	splenic immunosuppression
gallus d.	ed50	erod



rat	noec	hepatic vitamin A
falco s.	ed50	AEactivity



mouse	noec	immune response
gallus d.	ed50	lympoid development



mouse	noec	splenic immunosuppression
gallus d.	ed50	erod