MILJØstyrelsen

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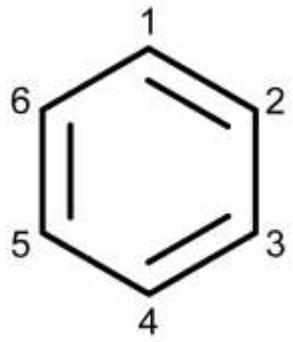
hcl/Kemikalier

Benzen (CAS nr. 71-43-2). Fastsættelse af kvalitetskriterier

Strukturformel



Vandkvalitetskriterie, ferskvand: 10 µg/l *(VRD)*
Vandkvalitetskriterie, saltvand: 8 µg/l *(VRD)*

Korttidsvandkvalitetskriterie(*fersk- og saltvand****): 50 µg/l*** *(VRD)*

Sundhed taget i betragtning:
Sedimentkvalitetskriterie, ferskvand: 0,70 µg/kg tørvægt
Sedimentkvalitetskriterie, saltvand: 0,70 µg/kg tørvægt

Sundhed ej taget i betragtning:
Sedimentkvalitetskriterie, ferskvand: 83 µg/kg tørvægt
Sedimentkvalitetskriterie, saltvand: 68 µg/kg tørvægt

Biotakvalitetskriteriet: 1,1 mg/ kg føde, vådvægt

English Summary

Data are given in the EU fact-sheet from December 2010, which is attached to this document as an appendix ("Bilag").

The environmental quality standards (EQS) and maximum acceptable concentrations (MAC) for fresh- and saltwater are given in the Water Framework Directive (WFD). These values do not take health effects into account, in which case the EQS would have been: $EQS_{freshw} = EQS_{saltw} = EQS_{water,\ human\ health} = 0{,}084\ \mu g/l$*, where the value has been "back-calculated" from the* $biota_{human\ health}$ *QS to a concentration in water.*

No data on toxicity to sediment dwelling organisms was found, and the EQS was calculated with equilibrium partitioning method (EqP).

Quality standards (QS) for sediment were calculated based on $EQS_{water,\ human\ health}$ *and on the WFD EQSes.*

The biota quality standard is based on human health effects, and is taken from the EU fact-sheet.

The quality standards are:
$EQS_{freshwater} = 10\ \mu g/l$ *(WFD)*
$EQS_{saltwater} = 8\ \mu g/l$ *(WFD)*
$MAC_{freshwater} = MAC_{saltwater} = 50\ \mu g/l$
EQSsediment:
Human health taken into account:
$EQS_{sediment,\ freshwater} = EQS_{sediment,\ saltwater} = 0{,}70\ \mu g/kg\ dw$
Human health not taken into account:
$EQS_{sediment,\ freshwater} = 83\ \mu g/kg\ dw$
$EQS_{sediment,\ saltwater} = 68\ \mu g/kg\ dw$
$EQS_{biota,\ human\ health} = 1{,}1\ \mu g/kg\ ww$

Oplysningerne om stoffet er hentet fra EU databladet fra december 2010, som er vedhæftet dette dokument som bilag.

Brug af stoffet: Se bilaget

Opløselighed i vand: Se bilaget

Giftighed overfor vandorganismer (EC_{50}, NOEC, EC_x, PNEC osv.):
Se bilag

Giftighed overfor pattedyr og fugle (NOEC, NOAEL, $PNEC_{oral}$ ($PNEC_{føde}$), hormonforstyrrende effekter osv.):
Se bilag

Giftighed overfor mennesker (ADI, TDI, hormonforstyrrende effekter, klassificering for kræft, reproduktionsskader og mutagenicitet):
Se bilag

Afsmag i fisk, skaldyr o.l.:
Ingen oplysninger

Nedbrydelighed:
Let nedbrydeligt. Se bilag

Bioakkumulering (log Kow, BCF, BMF):
BCF: Fisk: 13
Se bilag

Naturlig forekomst:
I afsnit 6.2 i bilaget er angivet nogle målte værdier, som dog næppe kan betragtes som naturlige baggrundsværdier. For ferskvand generelt er der noteret værdier mellem <0,1 µg/l – 31,7 µg/l.

For havvand har man målte værdier mellem <0,005 µg/l – 0,02 µg/l, men værdierne for fjorde og kystnære områder er på <1 – 89 µg/l.

I sediment har man for kornstørrelser <2 mm målt 39,5 µg/kg tørstof og for 20µm 174 µg/kg tørstof.

Vandkvalitetskriterier, inkl. argumentation og kvalitetsvurdering af udslagsgivende undersøgelse:

Data og beregninger er taget fra EU databladet (december-2010), der ligger til grund for vandkvalitetskriterierne (VKK) og korttidsvandkvalitetskriterierne (KVKK) i Vandrammedirektivet. EU databladet er vedlagt dette datablad som bilag.

Vandkvalitetskriterier:

VKK og KVKK er fra Vandrammedirektivet:

$VKK_{ferskvand}$	10 µg/l
$VKK_{saltvand}$	8 µg/l
$KVKK_{fersk\ og\ saltvand}$	50 µg/l

Biotakvalitetskriterier, BKK:

Benzen er klassificeret som kræftfremkaldende og mutagent, så selvom bioakkumuleringspotentialet er lille skal der fastsættes et BKK baseret på sundhedseffekter.

I EU databladet (se bilag) er BKK beregnet både for sekundær forgiftning af rovdyr (ekskl. kræft) og for sundhed.

Kvalitetskriteriet i biota til sikring mod sekundær forgiftning i fødekæden er 2306 µg/kg føde, vådvægt (fisk eller skaldyr), mens kvalitetskriteriet i biota baseret på beskyttelse af mennesker er 1,1 µg/kg føde, vådvægt. Da sidstnævnte er lavest, sættes biota kvalitetskriteriet til:

BKK = 1,1 µg/kg føde, vådvægt.

Omregnet til vandkoncentrationer fås:

$BKK_{vand,\ sekundær\ forgift} = BKK_{sek.forg.}/(BCF*BMF) = 2306\ µg/kg:(13\ l/kg*1) = 177\ µg/l$

$BKK_{vand,\ sundhed} = BKK_{sundhed.}/(BCF*BMF) = 1,1\ µg/kg:(13\ l/kg*1) = 0,0846\ µg/l$

$BKK_{vand,\ sundhed}$ er betydeligt lavere end VKK fastsat i Vandrammedirektivet, og normalt ville man vælge BKK_{vand} som overordnet VKK, hvis værdien er mindre end de almindelige VKK, men EU databladet har ikke beregnet BKK_{vand}, og Vandrammedirektivet har ikke fastsat BKK og tager ikke hensyn til denne værdi.

Sedimentkvalitetskriterier, SKK:

Der er ingen testresultater med effekter på sedimentlevende organismer, og den eneste mulighed er derfor at bruge ligevægtsfordelingsmetoden (Equilibrium Partitioning, EqP).

$SKK_{vådvægt} = K_{sediment\text{-}vand}*VKK*1000/RHO_{sed}$

$K_{sediment\text{-}vand}$ = 4,2 (se bilag)

RHO_{sed} = 1300 (standardværdi i EU vejledningen)

Baseret på et overordnet VKK = BKK_{vand} = 0,0846 µg/l fås:

$SKK_{vådvægt}$ = 4,2*0,0846 µg/l*1000:1300 kg/m^3 = 0,27 µg/kg vådvægt

Baseret på $VKK_{ferskvand}$ = 10 µg/l og VKKsaltvand = 8 µg/l fås:

Ferskvand: $SKK_{vådvægt}$ = 4,2*10 µg/l*1000:1300 kg/m^3 = 32 µg/kg vådvægt

Saltvand: $SKK_{vådvægt}$ = 4,2*8 µg/l*1000:1300 kg/m^3 = 26 µg/kg vådvægt

$SKK_{tørvægt}$ = $SKK_{vådvægt}$ *konverteringsfaktor

Konverteringsfaktor = RHO_{sed} /F_{solid}* RHO_{solid} = 1300/0,2*2500 = 2,6

RHO_{solid} =2500 (standardværdi i EU vejledningen)

F_{solid} = 0,2 (standardværdi i EU vejledningen)

Baseret på et overordnet VKK = BKK_{vand} = 0,0846 µg/l fås:

$SKK_{tørvægt}$ = 0,27 µg/kg*2,6 = 0,70 µg/kg tørvægt

Baseret på $VKK_{ferskvand}$ = 10 µg/l og VKKsaltvand = 8 µg/l fås:

Ferskvand: $SKK_{tørvægt}$ = 32 µg/kg*2,6 = 83 µg/kg

Saltvand: $SKK_{tørvægt}$ = 26 µg/kg*2,6 = 68 µg/kg

Kvalitetskriterierne bliver således:

$VKK_{ferskvand}$: 10 µg/l *(vandrammedirektivet)*

$VKK_{saltvand}$: 8 µg/l *(vandrammedirektivet)*

KVKK = 50 µg/l *(vandrammedirektivet)*

Sundhed taget i betragtning:

SKK_{fersku} = $SKK_{saltvand}$ = 0,70 µg/kg tørvægt

Sundhed ikke taget i betragtning:
SKK_{ferskv} = 83 µg/kg tørvægt
$SKK_{saltvand}$ = 68 µg/kg tørvægt

BKK = 1,1 µg/kg føde, vådvægt

BILAG

EU datablad fra 11/12 2010

BENZENE

The Environmental Quality Standard (EQS) fact sheet addressing benzene issued in 2005 is not totally consistent with the draft TGD on EQS derivation (E.C., 2010) and does not include latest ecotoxicological and toxicological data contained in the final version of the European Union Risk Assessment Report (E.C., 2008a) performed in the context of assessment of existing chemicals (Regulation 793/93/EEC). The EQS for benzene in the present document was updated based on this new document and on its corresponding Summary Risk Assessment Report (E.C., 2007a).
Comments in response to the Questionnaire on existing priority substances issued to WG E in 2010 included the concern that benzene is carcinogenic and that the EQS for benzene should be based on the most sensitive endpoint, i.e. human health. For this reason (noting particularly the high potential toxicity of benzene, its mutagenicity and carcinogenicity), and despite the low bioaccumulation attributed to benzene, the dossier presents the proposal that the $QS_{biota,\ hh}$ (derived from epidemiological data), as the lowest standard derived, be deemed the "critical QS" for derivation of an EQS for all surface water bodies. This would equate to a water EQS of 0.08 $\mu g.l^{-1}$, compared with the existing standard of 10 $\mu g.l^{-1}$. The latter was based on direct toxicity combined with the expert judgment of the assessors regarding how to account for the other hazardous characteristics of the substance.
Because of the concern expressed about human health, a review of existing published information on threshold values for drinking water was conducted. It did not lead to the conclusion that the quality standard for protection of human health via the consumption of drinking water itself should be changed. It is noted, though, that the provisional drinking water standard calculated on the basis of the epidemiological data referred to above is lower than the EC standard. Derivation of standards for water bodies intended for the abstraction of drinking water

($QS_{dw, hh}$) is presented at the end of section 7.3. Data gathered in 2010 on benzene removal efficiency are referred to.

1 CHEMICAL IDENTITY

Common name	Benzene
Chemical name (IUPAC)	Benzene
Synonym(s)	Cyclohexatriene; Benzol
Chemical class (when available/relevant)	Polyaromatic hydrocarbons (PAH)
CAS number	71-43-2
EC number	200-753-7
Molecular formula	C6H6
Molecular structure	
Molecular weight ($g.mol^{-1}$)	78.11

2 EXISTING EVALUATIONS AND REGULATORY INFORMATION

Legislation	
Annex III EQS Dir. (2008/105/EC)	Not Included
Existing Substances Reg. (793/93/EC)	Priority List 1 / Final RAR, final approved version of 2008
Pesticides(91/414/EEC)	Not applicable
Biocides (98/8/EC)	Not applicable
PBT substances	Not investigated by EU PBT groups
Substances of Very High Concern (1907/2006/EC)	Not assessed
POPs (Stockholm convention)	No
Other relevant chemical regulation (veterinary products, medicament, ...)	No
Endocrine disrupter (Groshart and Okkerman, 2000; E.C., 2004; E.C., 2007b; Petersen *et al.*, 2007)	Not assessed

3 PROPOSED QUALITY STANDARDS (QS)

3.1 ENVIRONMENTAL QUALITY STANDARD (EQS)

Although benzene has a low bioaccumulation potential, protection of human health from consumption of fishery products via derivation of $QS_{biota, hh}$ is deemed of relevance because benzene is a confirmed mutagenic and carcinogenic substance. $QS_{biota, hh}$ is the lowest standard and is deemed the "critical QS" for derivation of an Environmental Quality Standard for other water bodies. However, as benzene does not have a high potential for bioaccumulation, the proposed AA-EQS is expressed in the water compartment, in $\mu g.l^{-1}$.

The uncertainty associated to the derivation of $QS_{biota, hh}$ is linked to the route-to-route extrapolation from an inhalation value used for the determination of the threshold level (oral cancer risk value). This extrapolation was agreed by US-EPA experts who have derived this threshold value, in particular on the basis of the consistency of the target cells affected by benzene toxicity, independently of the exposure route. Moreover, it is consistent with the value proposed by EU experts.

	Value	**Comments**
Proposed AA-EQS for [freshwaters and saltwaters] [$\mu g.l^{-1}$] based on AA-EQS for [biota] [$\mu g.kg^{-1}_{biota\ ww}$]	**0.08** 1.1	**Critical QS is $QS_{biota, hh}$** **See section 7**
Proposed MAC-EQS for [freshwater] [$\mu g.l^{-1}$] **Proposed MAC-EQS for [marine waters] [$\mu g.l^{-1}$]**	**80** **8**	**See section 7.1**

3.2 SPECIFIC QUALITY STANDARD (QS)

Protection objective[1]	**Unit**	**Value**	**Comments**
Pelagic community (freshwater)	[$\mu g.l^{-1}$]	80	See section 7.1
Pelagic community (marine water)	[$\mu g.l^{-1}$]	8	
Benthic community (freshwater)	[$\mu g.kg^{-1}$ dw]	Not relevant	See section 7.1
Benthic community (marine)	[$\mu g.kg^{-1}$ dw]	Not relevant	
Predators (secondary poisoning)	[$\mu g.kg^{-1}_{biota\ ww}$]	2 306	See section 7.2
	[$\mu g.l^{-1}$]	177.3	

[1] Please note that as recommended in the Technical Guidance for deriving EQS (E.C., 2010), "EQSs […] are not reported for 'transitional and marine waters', but either for freshwater or marine waters". If justified by substance properties or data available, QS for the different protection objectives are given independently for transitional waters or coastal and territorial waters.

Human health via consumption of fishery products	[$\mu g.kg^{-1}_{biota\ ww}$]	1.1	See section 7.3
	[$\mu g.l^{-1}$]	0.084	
Human health via consumption of water	[$\mu g.l^{-1}$]	1	
Water intended for abstraction for human consumption	[$\mu g.l^{-1}$]	1-1.67	

4 MAJOR USES AND ENVIRONMENTAL EMISSIONS

Data reported hereunder are extracted from the Summary RAR on Benzene (E.C., 2007a).

4.1 USES AND QUANTITIES

"*The natural sources of benzene are crude oil and, to a lesser extent, condensate from natural gas production. Benzene is produced by different petroleum conversion processes in petroleum refinery and chemical plant processes, primarily by catalytic reforming, steam cracking and dealkylation. Benzene is recovered during production of coal-derived chemicals, primarily from coke oven by-products. Benzene is extracted from these sources and purified for industrial use.*

Based on the available information the estimated annual production of benzene as a chemical intermediate in the European Union (EU) [was] 7 247 kt/a [in 2008]. These figures, however, can overestimate production, because for some companies IUCLID figures were used.

In the EU benzene [was] produced or imported [in 2008] by 14 companies, 22 sites were identified at which both production and processing take place, and 12 sites where benzene is only processed.

The major uses of benzene in the EU are the production of ethylbenzene (52 %), cumene (20 %), cyclohexane (13 %), nitrobenzene (9 %), alkylbenzene (3 %), maleic anhydride and other (2 %) and chlorobenzene (1 %). Benzene used in petrol is in addition to the benzene of chemical intermediate production. The quantity of benzene present in petrol [was] estimated at 1.41 million t for the EU in 2000.

Very small quantities are also used as a laboratory reagent and solvent.

Since benzene is a natural component of crude oil, it is an intrinsic constituent of certain refinery fractions, or it is formed during the refining process in use today. As a result, benzene as a component of refinery products also ends up in consumer products."

4.2 ESTIMATED ENVIRONMENTAL EMISSIONS

"*Benzene is released from a number of man-made sources. The primary sources of environmental benzene are automobile exhaust emissions, evaporative losses and refuelling emissions. Benzene in automotive exhaust is a mixture of incompletely burned benzene and benzene produced in the motor during combustion through dealkylation of toluene and xylenes. From industrial sources, it primarily enters the environment as fugitive emissions from industrial intermediate production and processing operations and through air emissions from waste water treatment plants.*

Natural sources of benzene emissions such as vulcanos and forest fires exist.

Benzene is used and emitted in large quantities. Because benzene is a volatile organic compound, it is mainly emitted to the air and emissions to soil and water partly lead to emission to the air. As a result the emission most of the benzene is found in the air compartment.

Based on the available and traceable exposure data and the default values used, an emission to waste water treatment plants (WWTP) of 25 821 t/a and an emission to air (direct) of 60 787 t/a is calculated for the industrial production and processing sites.
In addition, further environmental point source releases occur from oil refineries, coking plants, stationary combustion of fossil fuels for energy production, offshore platforms, road traffic. Disperse source releases include evaporative losses from petrol distribution, combustion of fossil fuels for commercial and residential heating, WWTP, laboratory reagent and solvent at laboratories, landfill sites, accidental releases (not considered in this report), natural sources and environmental tobacco smoke (ETS). A total emission to air of 193 909 t/a is estimated for the releases from all these sources, including industrial production and processing."

5 ENVIRONMENTAL BEHAVIOUR

5.1 ENVIRONMENTAL DISTRIBUTION

		Master reference
Water solubility ($mg.l^{-1}$)	1 800 at 25°C	Freier, 1976 *in* E.C., 2008a
Volatilisation	**Benzene is a substance very likely to volatilise.**	
Vapour pressure (Pa)	99 700 at 20°C	Folkins, 1985 *in* E.C., 2008a
Henry's Law constant($Pa.m^3.mol^{-1}$)	432.6 at 20°C (*calculated*)	E.C., 2008a
Adsorption	**The calculated value 134 is used for derivation of QS**	
Organic carbon – water partition coefficient (K_{OC}) ($l.kg^{-1}$)	18.2 – 1 023 (*measured*) 134 (*TGD calculated*, used in EU-RAR)	Chiou *et al.*, 1983 Uchrin and Mangels, 1987 *in* E.C., 2008a
Sediment – water partition coefficient ($K_{sed\text{-}water}$) ($m^3.m^{-3}$)	4.2	*Calculated from K_{OC}*
Bioaccumulation	**The BCF value of 13 is used for derivation of $QS_{biota\ secpois}$. Thus, $BMF_1 = BMF_2 = 1$ (E.C., 2010).**	
Octanol-water partition coefficient (log Kow)	2.13 (*measured – HPLC method*)	Sangster, 1989 *in* E.C., 2008a
BCF fish	The RAR specifies: "*different experiments available show that benzene has a low to moderate bioaccumulation potential. In all but one available test conducted with fish BCF were clearly below 100. The uptake of benzene was followed by a fast depuration when the test organisms were placed into clean medium. In one test conducted with northern anchovies a BCF of 8 450 was measured in the gallbladder. As only ^{14}C-analysis was conducted not only accumulated benzene but also possible metabolites were detected. Moreover, bioaccumulation in certain organs of fish is difficult to interprete as it is not possible to calculate the BCF for the whole fish. Therefore, for the assessment of the bioaccumulation potential only BCFs that are related to the whole fish are used.*" The highest available measured BCFs related to whole fish were equal	E.C., 2008a

	or lower than 11. "*These values are supported by the BCF of 13 that can be estimated from the log Kow of 2.13* [...]. *In the further assessment a BCF of 13 is used. It has to be kept in mind that aquatic invertebrates serving as food source for fish may accumulate benzene to a high degree if they are not able to discharge or metabolize it.*"	

5.2 ABIOTIC AND BIOTIC DEGRADATIONS

		Master reference
Hydrolysis	Hydrolysis at environmental conditions is not likely due to the lack of reactive functional groups in the molecule.	E.C., 2008a
Photolysis	Direct photolysis (surface water) is of minor importance due to low absorbance of UV light.	E.C., 2008a
Biodegradation	Benzene has to be classified as readily biodegradable. Corresponding default values (E.C., 2003) are: Surface water: $k_{biodegradation}$ = 0.047 d^{-1} and $t_{1/2}$ = 15 d Aerated sediment: $k_{biodegradation}$ = 0.0023 d^{-1} and $t_{1/2}$ = 300 d	E.C., 2008a

6 AQUATIC ENVIRONMENTAL CONCENTRATIONS

6.1 ESTIMATED CONCENTRATIONS

Compartment		**Predicted environmental concentration (PEC)**	**Master reference**
Freshwater (µg.l^{-1})	C_{local} – production and processing – generic approach	0.29 – 4 732	E.C., 2007a
	C_{local} – use as laboratory reagent	unknown	
	C_{local} – use in refinery processes	<0.02 – 0.19	
	$PEC_{regional}$	0.275	
Marine waters (µg.l^{-1})		No data available	E.C., 2007a
Freshwater sediment (µg.kg^{-1} dw)		No accumulation assumed	E.C., 2007a E.C., 2008a
Marine sediment (µg.kg^{-1} dw)			
Biota (freshwater)		No accumulation assumed	E.C., 2007a E.C., 2008a
Biota (marine)			
Biota (marine predators)			

6.2 MEASURED CONCENTRATIONS

Compartment		**Measured environmental concentration (MEC)**	**Master reference**
Freshwater (µg.l^{-1})		PEC 1: 3 PEC 2: 1	James *et al.*, 2009*(1)*
		<0.1 – 31.7	E.C., 2007a
Marine waters (coastal and/or transitional) (µg.l^{-1})		No data available	James *et al.*, 2009*(1)*
		Marine : <0.005 – 0.02 Transitional: <1 – 89.4	E.C., 2007a
WWTP effluent (µg.l^{-1})		No data available	
Sediment(µg.kg^{-1} dw)	Sed < 2 mm	PEC 1: no data PEC 2: 39.5	James *et al.*, 2009*(1)*
	Sed 20 µm	PEC 1: 174	

		PEC 2: 89.5	
	Sed 63µm	No data available	
Biota(µg.kg^{-1} ww)	Invertebrates	No data available	James *et al.*, 2009[(1)]
	Fish	No data available	
	Marine predators	No data available	

[(1)] data originated from EU monitoring data collection

7 EFFECTS AND QUALITY STANDARDS

The data reported in this section hereafter correspond to the most relevant effect data extracted from EU-RAR (E.C., 2008a).

7.1 ACUTE AND CHRONIC AQUATIC ECOTOXICITY

The data considered as valid in the RAR were not further assessed. The RAR specifies: "*For the* [effects] *assessment those tests are preferred that were conducted in flow-through systems with analytical monitoring of the benzene concentration because of the high volatility of the substance. If nominal concentrations are reported it has to be considered that the effect values may be significantly lower due to volatilization.*"(E.C., 2007a).

In the tables below, the following legend is used:

- Effects concentrations reported as based on nominal and measured concentrations are referred to as "(n)" and "(m)", respectively
- Studies conducted in closed or open vessels are reported as "(c)" and "(o)", respectively

ACUTE EFFECTS			Reliability	Master reference
Micro-organisms (mg.l^{-1})	**Freshwater**	*Pseudomonas putida* 16h – $EC_{3\ -\ cell\ multiplication\ inhib.}$ = 92 (n)	Valid according to EU-RAR (E.C., 2008a)	Bringmann and Kühn, 1980
		Nitrosomonas sp. 24h – $IC_{50\ -\ NH3\ consum.\ inhib.}$ = 13 (n, c)		Blum and Speece, 1991
		Tetrahymena pyriformis 24h – $EC_{0\ -\ ciliary\ immobilisation}$ = 391 (n, c)		Rogerson *et al.*, 1983
	Marine	No information available		
Algae & aquatic plants (mg.l^{-1})	**Freshwater**	*Ankistrodesmums falcatus* 4h – $EC_{50-\ 14C\ uptake\ inhib.}$ = 310 (n, c)	Valid according to EU-RAR (E.C., 2008a)	Wong and Raabe, 1995
		Chlamydomonas angulosa 3h – $EC_{50-\ 14Co2\ uptake\ inhib.}$ = 461 (n, c)		Hutchinson *et al.*, 1980
		Chlorella vulgaris 3h – $EC_{50-\ 14Co2\ uptake\ inhib.}$ = 312.5 (n, c)		Hutchinson *et al.*, 1980
		Selenastrum capricornutum 72h – $E_bC_{50\ -\ growth\ inhib.}$ = 28 (m, c) 72h – $E_rC_{50\ -\ growth\ inhib.}$ = 100 (m, c) 72h – $EC_{50\ -\ growth\ inhib.}$ = 29 (m, c) 8d – $E_bC_{50\ -\ growth\ inhib.}$ = 41 (n, c)		TNO, 2000 TNO, 2000 Galassi *et al.*, 1988 Herman *et al.*, 1990
	Marine	*Phaeodactylum tricornutum* 24h – $LOEC_{photosynthesis\ inhib.}$ = 50 (n, c) 175 <2h – $EC_{50\ -\ photos.\ inhib.}$< 350 (n, c)		Kusk, 1981 Kusk, 1980

ACUTE EFFECTS			Klimisch code	Master reference
Invertebrates **(mg.l^{-1})**	**Freshwater** Cnidarians	*Hydra oligactis* 48h – LC_{50} = 34 (n, c)	Valid according to EU-RAR (E.C., 2008a)	Sloof and al., 1983
	Plathelmin-thes	*Dugesia cf. lugubris* 48h – LC_{50} = 74 (n, c)		Sloof and al., 1983
	Annelids	*Erpobdella octoculata* 48h – LC_{50} > 320 (n, c)		Sloof and al., 1983
		Limnodrilus sp. and Tubifex sp. 48h – LC_{50} > 320 (n, c)		Sloof and al., 1983
	Crustaceans	*Asellus aquaticus* 48h – LC_{50} = 120 (n, c)		Sloof and al., 1983
		Daphnia magna 24h, 48h – $EC_{50\ -\ immobilisation}$ = 10 (n) 24h – $EC_{50\ -\ immobilisation}$ = 18 (m, c)		Janssen and Persoone, 1993 Galassi *et al.*, 1988
		Daphnia pulex 96h – LC_{50} = 15 (m, c)		Trucco *et al.*, 1983
		Ceriodaphnia dubia 48h – LC_{50} = 17.2 (m, c)		Niederlehner *et al.*, 1998
		Gammarus pulex 48h – LC_{50} = 42 (n, c)		Sloof and al., 1983
	Insects	*Aedes aegypti* / 4th instar larvae 24h – LC_0 = 12.9 24h – LC_{50} = 59		Berry and Brammer, 1977
		Chironomus gr. thummi 48h – LC_{50} = 100 (n, c)		Sloof and al., 1983
		Cloëon dipterum 48h – LC_{50} = 34 (n, c)		Sloof and al., 1983
		Corixa punctata 48h – LC_{50} = 48 (n, c)		Sloof and al., 1983
		Ischnura elegans 48h – LC_{50} = 10 (n, c)		Sloof and al., 1983
		Nemoura cinerea 48h – LC_{50} = 130 (n, c)		Sloof and al., 1983
	Marine Crustaceans	*Artemia salina* 48h – LC_{50} = 21 (n)		Price *et al.*, 1974
		Cancer magister 96h – LC_{50} = 108 (n)		Caldwell and al., 1977
		Nitocra spinipes 24h – $LC_{50\ -\ 1.5\%\ salinity}$ = 82 (n) 24h – $LC_{50\ -\ 2.5\%\ salinity}$ = 111.5 (n)		Potera, 1975
		Palaemonetes pugio 24h – $LC_{50\ -\ 1.5\%\ salinity}$ = 38 (n) 24h – $LC_{50\ -\ 2.5\%\ salinity}$ = 33.5 (n) 96h – LC_{50} = 27 (n, o)		Potera, 1975 Potera, 1975

				Tatem, 1978
	Sediment	No information available		

ACUTE EFFECTS			Klimisch code	Master reference
Fish **(mg.l^{-1})**	**Freshwater**	*Carassius auratus* 96h – LC_{50} = 34.42 (n, s)	Valid according to EU-RAR (E.C., 2008a)	Pickering and Henderson, 1966
		Cottus cognatus 96h – LC_{50} = 13.5 (n, s)		Moles and al., 1979
		Gasterosteus aculeatus 96h – LC_{50} = 21.8 (n, s)		Moles and al., 1979
		Lepomis macrochirus / (n, s) 48h – LC_{50} = 20 (n, s) 96h – LC_{50} = 22.49 (n, s)		Turnbull *et al.*, 1954 Pickering and Henderson, 1966
		Oncorhynchus gorbuscha Freshwater – 96h – LC_{50} = 15 (n, s) Saltwater – 96h – LC_{50} = 7.4 (n, s)		Moles and al., 1979
		Oncorhynchus mykiss 96h – LC_{50} = 5.3 (m, ft) 96h – LC_{50} = 5.9 (m, ss) 96h – LC_{50} = 21.6 (m, ft)		DeGraeve *et al.*, 1982 Galassi *et al.*, 1988 Hodson *et al.*, 1984
		Oncorhynchus nerka Freshwater – 96h – LC_{50} = 9.5 (n, s) **Saltwater – 96h – LC_{50} = 4.9* (n, s)**		Moles and al., 1979
		Oncorhynchus tschawytscha 96h – LC_{50} = 10.3 (n, s)		Moles and al., 1979
		Pimephales promelas 96h – LC_{50} = 15.6 (m, ft, larvae) 96h – LC_{30} = 15.1 (m, ft) 96h – $LC_{50\ -\ hard\ water}$ = 32 (n, s) 96h – $LC_{50\ -\ soft\ water}$ = 33.5 (n, s) 7d – LC_{50} = 14.02 (m, ft, larvae) 7d – $NOEC_{growth}$ = 10.02 (m, ft, larvae)		Marchini *et al.*, 1992 DeGraeve *et al.*, 1982 Pickering and Henderson, 1966 Pickering and Henderson, 1966 Marchini *et al.*, 1992 Marchini *et al.*, 1992
		Poecilia reticulata 96h – LC_{50} = 28.6 (m, ss) 96h – LC_{50} = 36.6 (n, s) 14d – LC50 = 63.5 (n, ss)		Galassi *et al.*, 1988 Pickering and Henderson, 1966 Koenemann, 1981
		Thymallus arcticus 96h – LC_{50} = 12.9 (n, s)		Moles and al., 1979

	Fresh, brackish and marine water	*Oncorhynchus kisutsch* 96h – LC_{50} = 12.4 (n, s)		Moles and al., 1979
	Marine	*Salvelinus malma* Freshwater – 96h – LC_{50} = 10.5 (n, s) Saltwater – 96h – LC_{50} = 5.5 (n, s)		Moles and al., 1979
		Morone saxatilis 96h – LC_{50} = 9.58 (m, ft)		Meyerhoff, 1975
	Sediment	No information available		

***** "*LC50 determined from the initial benzene concentration while the authors found a decrease of benzene concentration to 75 % after 24 hours and to 10 % after 96 hours. Therefore, the real effect value may be significantly lower than the nominal value reported above.*" (E.C., 2008a)

CHRONIC EFFECTS			**Valid according to**	**Master reference**
Algae & aquatic plants **(mg.l^{-1})**	**Freshwater**	*Selenastrum capricornutum* 72h – $E_bC_{10\ –\ growth\ inhib.}$ = 8.3 (m, c) 72h – $E_rC_{10\ –\ growth\ inhib.}$ = 34 (m, c)	Valid according to EU-RAR (E.C., 2008a)	TNO, 2000
	Marine	*Phaeodactylum tricornutum* 96h – $LOEC_{growth\ inhib.}$ = 50 (n, c)		Kusk, 1981
Invertebrates **(mg.l^{-1})**	**Freshwater**	*Ceriodaphnia dubia* 7 d – $NOEC_{reproduction}$ = 3 (m, ss, c)		Niederlehner *et al.*, 1998
	Marine	No information available		
	Sediment	No information available		
Fish **(mg.l^{-1})**	**Freshwater**	*Oncorhynchus mykiss* / eggs 23-27d – N OEC = 0.0035* (m, ft)	Valid according to EU-RAR (E.C., 2008a)	Black *et al.*, 1982***
		Pimephales promelas* / ELS / larvae** **32d – $NOEC_{wet\ weight,\ length}$ ≈ 0.8*		Russom and S.J., 1991
	Marine	No information available		
	Sediment	No information available		
Amphibians **(mg.l^{-1})**	**Freshwater**	*Rana pipiens* / embryo-larval Exposure: fertilisation -> 4d after hatching $LC_{1\ –\ repro}$ = 0.0032 (m, ft) $LC_{10\ –\ repro}$ = 0.0756(m, ft)	Valid according to EU-RAR (E.C., 2008a)	Black *et al.*, 1982 ***
		Ambystoma gracile / embryo-larval Exposure: fertilisation -> 4d after hatching $LC_{1\ –\ repro}$ = 0.0682 (m, ft) $LC_{10\ –\ repro}$ = 0.478 (m, ft)		
	Marine	No information available		

* *"No NOEC or EC_{10} was determined by the authors. Therefore an EC10-value was derived by probit analysis on the basis of the available test results. An EC10-value of 3.5 µg/l could be determined that can be regarded as NOEC for 23-27 day exposure."* (E.C., 2008a)

** "*NOEC ≈ 0.8 according to TGD as 32d – $LOEC_{wet\ weight,\ length}$ of 1.6 was in the 10-20% effect range.*" (E.C., 2008a)

*** "*The effect values found by Black et al. (1982) for several substances* […] *are usually very low compared to effect values found by other authors. No explanation for these large discrepancies could be found. However, as it was not possible to reproduce the effect values found by Black and his co-workers, Member State's and industry experts advised not to use these data for a derivation of* [an effects threshold for aquatic organism] *if other valid fish early life stage tests are available.*" (E.C., 2008a).

The EU-RAR (E.C., 2008a) indicates that for algae, crustaceans and fish, the experimental acute and chronic values "*are in general agreement with QSAR estimation[s]* [...] *for non polar narcotic acting substances. However, it should be noted that benzene may not only cause adverse effects due to non-polar narcotic action as the substance is a human carcinogen. Therefore, it cannot be excluded that the substance may cause ecological relevant adverse effects based on specific modes of action.*"
The Draft Guidance Document on EQS derivation (E.C., 2010) states that "*in principle, ecotoxicity data for freshwater and saltwater organisms should be pooled for organic compounds, if certain criteria are met*" and that "*the presumption that for organic compounds saltwater and freshwater data may be pooled must be tested, except where a lack of data makes a statistical analysis unworkable*."
This is the case for benzene. In fact, there are too few data (either freshwater or saltwater) to perform a "*meaningful statistical comparison*" and no further indications of "*a difference in sensitivity between freshwater vs saltwater organisms*". Even if "*specific modes of action can not be excluded*", the main mode of action identified being narcosis, information allowing differentiating between the two media is insufficient.
Therefore, in this case, the data sets may be combined for QS derivation according to the Draft Guidance Document on EQS derivation (E.C., 2010).
Several toxicity tests with aquatic organisms were conducted using benzene as test substance, including a high number of taxonomic groups: micro-organisms, algae, several species of invertebrates (cnidarians, plathelminthes, annelids, crustaceans and insects) and several families of fish, but higher plants are missing in the dataset to apply a statistical approach.

The lowest long term effect value was obtained in an embryo-larval-test conducted with *Oncorhynchus mykiss* but this test was lead by Black et al. (1982), which are deemed not usable by Member State's and industry experts because generating very low inexplicable and non-reproducible values compared to effect values found by other authors. These values are therefore deemed not usable for effects assessment purposes if other valid fish early life stage tests are available. The 32d – NOEC of 0.8 $mg.l^{-1}$ found by Russom and Boderius (1991) in the ELS test with *Pimephales promelas* is thus used as a basis for the $QS_{water, eco}$ derivation.
Short term and long-term tests being available from three trophic levels, assessment factors of 10 and 100 applied on the lowest NOEC value are used for derivation of AA-$QS_{freshwater, eco}$ and AA-$QS_{marine\ water, eco}$, respectively.

With regard to short-term exposure of organisms, the available valid $L(E)C_{50}$ values point to similar susceptibility of sensitive taxa in fish and invertebrates (crustaceans), comparing to a somewhat lower overall sensitivity of algae. The most sensitive species seem to be the salmonids. In seawater a LC_{50} of 4.9 $mg.l^{-1}$ was derived with *Oncorhynchus nerka* in a static system but as the effect value was determined from the initial benzene concentration with a decrease of benzene concentration to 75 % after 24 hours and to 10 % after 96 hours, the real effect value may be significantly lower than the nominal value reported by Moles et al (1979).
With no more specific information on the study, it can be assumed that 96h-LC_{50} based on

measured data would have been in the range of the lowest NOEC of 0.8 $mg.l^{-1}$. Given the assumed non specific mode of action (narcosis), standard assessment factors of 100 and 1000 can be lowered to 10 and 100 to derive $MAC\text{-}QS_{freshwater,\ eco}$ and $MAC\text{-}QS_{marine\ water,\ eco}$, respectively. Applying these assessment factors result in MAC values very close to AA-QS values. Therefore, given the rather low difference of organisms' sensitivity between acute and chronic exposure, and the uncertainty linked to the determination of the acute effects level, it is proposed to set the MAC-QS equal to the $AA\text{-}QS_{water}$.

There are no results from sediment tests with benthic organisms available. According to the physico-chemical properties currently known, there is nothing indicating that benzene accumulates in sediment and derivation of $QS_{sediment}$ values are deemed not necessary.

Tentative QS_{water} Assessment factor method	**Relevant study for derivation of QS**	**AF**	**Tentative QS**
$MAC_{freshwater,\ eco}$	The MAC-QS is set as equal to the $AA\text{-}QS_{water}$	-	80 $\mu g.l^{-1}$
$MAC_{marine\ water,\ eco}$		-	8 $\mu g.l^{-1}$
$AA\text{-}QS_{freshwater,\ eco}$	*Pimephales promelas* / ELS / larvae 32d – $NOEC_{wet\ weight,\ length} \approx 0.8\ mg.l^{-1}$	10	80 $\mu g.l^{-1}$
$AA\text{-}QS_{marine\ water,\ eco}$		100	8 $\mu g.l^{-1}$
$AA\text{-}QS_{freshwater,\ sed.}$	Not triggered according to physico-chemical properties of the substance		
$AA\text{-}QS_{marine\ water,\ sed.}$			

7.2 SECONDARY POISONING

Benzene presents a high toxicological potential and is carcinogenic. Therefore, an effects assessment for protection of top predators from secondary poisoning is triggered although benzene has a low bioaccumulation potential (log K_{OW}=2.13, BCF=13).

The study considered as relevant for the assessment of secondary poisoning is a combined chronic and cancer study, which leads to the lowest non-carcinogenic endpoint reported in the following table for the purpose of $QS_{biota,\ sec.\ pois.}$ derivation. More detailed information on mammals and human toxicity of benzene are reported in the section dedicated to human health protection (see below section 7.3).

Secondary poisoning of top predators		**Master reference**
Mammalian oral toxicity	Oral-gavage / 2-year chronic/cancer study $B6C3F_1$ Mice / 0, 5, 100 and 200 $mg.kg^{-1}_{bw}.d^{-1}$ Female rats / 0, 25, 50 and 100 $mg.kg^{-1}_{bw}.d^{-1}$ Male rats / 0, 50, 100 and 200 $mg.kg^{-1}_{bw}.d^{-1}$ Compound-related non-neoplastic or neoplastic effects on the haematopoietic system, Zymbal gland, forestomach, and adrenal gland effects were seen in both sexes of both species. In addition, the oral cavity and the lung were affected in rats while liver harderian gland, preputial gland, ovary, and mammary gland in mice. LOAEL = 25 $mg.kg^{-1}_{bw}.d^{-1}$ NOAEL = 8.3 $mg.kg^{-1}_{bw}.d^{-1}$ ($CF_{LOAEL->NOAEL}$=3[(1)]) NOEC = 69 $mg.kg^{-1}_{food}$ ($CF_{NOAEL->NOEC}$=8.3)	NTP, 1986 Huff *et al.*, 1989 as cited *in* US-EPA, 2000
Avian oral toxicity	No information available	

[(1)] No recommendation is made about this conversion factor in the Draft Guidance Document on EQS derivation (E.C., 2010) but this value is recommended by REACH guidances (ECHA, 2008).

Tentative $QS_{biota,\ sec.\ pois.}$	**Relevant study for derivation of QS**	**AF**	**Tentative QS**
Biota	NOEC = 69 $mg.kg^{-1}_{feed\ ww}$	30	2 306 $\mu g.kg^{-1}_{biota\ ww}$ corresponding to 177.3 $\mu g.l^{-1}$ (fresh and marine waters)

7.3 HUMAN HEALTH

Benzene presents a high toxicological potential to mammals and humans. It is mutagenic and carcinogenic. Therefore, an effects assessment for protection of human health from consumption of fishery products is deemed triggered although benzene has a low bioaccumulation potential (log K_{OW}=2.13, BCF=13).

The key findings suggest that benzene is absorbed by all routes: inhalation, dermal and oral routes.

Repeated dose toxicity: Irrespective of the exposure route the main and sensitive targets of toxicity in animals and humans after repeated dose application of benzene are the cells of the bone marrow and haematopoietic system. The rapidly proliferating stem cells, myeloid progenitor cells and stromal cells are sensitive targets. Chronic benzene exposure can result in bone marrow depression expressed as leucopenia, anaemia and/or thrombocytopenia, leading to pancytopenia and aplastic anaemia.

Mutagenicity: Benzene is an *in vivo* somatic cell mutagen mutagen for mammals, especially chromosomal aberrations and micronuclei are induced, and for man. Data on germ cell effects are inconsistent. However, due to the clastogenicity to spermatogonia and the toxicokinetic properties it is concluded that benzene has the potential to reach the gonads and induce germ cell mutations.

Carcinogenicity: Animal models were able to identify the carcinogenic potential of benzene. However, the tumour response is different between animals and humans. There is sufficient scientific evidence from the numerous human epidemiological studies to assume a causal relationship between benzene exposure and acute non-lymphatic leukaemia. It is unclear, however, if there exists a threshold level of benzene exposure above which the risk of leukaemia significantly increases.

Reprotoxicity: Evidence from human data for an effect of benzene exposure on female reproduction is not sufficient to demonstrate a causal association due to poorly designed studies and inadequately quantified exposure to benzene as well as to other chemicals. Epidemiological studies in males on effects on fertility are not available. Likewise epidemiological studies implicating benzene as a developmental toxicant have many limitations thus not providing sufficient information to assess the effects on the human fetus. Thus, hazard identification and assessment is primarily based on the data from animal studies.

CMR classification	**Carcinogenic: 1A** – Known to have carcinogenic potential for humans, classification is largely based on human evidence **Mutagenic: 1B –** Substances known to induce heritable mutations or to be regarded as if they induce heritable mutations in the germ cells of humans. Sub-	E.C., 2008b

	stances known to induce heritable mutations in the germ cells of humans. The classification in Category 1B is based on: — positive result(s) from in vivo heritable germ cell mutagenicity tests in mammals; or — positive result(s) from in vivo somatic cell mutagenicity tests in mammals, in combination with some evidence that the substance has potential to cause mutations to germ cells. It is possible to derive this supporting evidence from mutagenicity/genotoxicity tests in germ cells in vivo, or by demonstrating the ability of the substance or its metabolite(s) to interact with the genetic material of germ cells; or — positive results from tests showing mutagenic effects in the germ cells of humans, without demonstration of transmission to progeny; for example, an increase in the frequency of aneuploidy in sperm cells of exposed people. **Reprotoxic: not classified**	

Human health via consumption of fishery products		**Master reference**
Mammalian oral toxicity	Mice / Oral / Long-term exposure / micronucleus tests LOAEL = 25 $mg.kg^{-1}_{bw}.d^{-1}$	E.C., 2008a
	Oral-gavage / 2-year chronic/cancer study - Female rats / 0, 25, 50 and 100 $mg.kg^{-1}_{bw}.d^{-1}$ Male rats / 0, 50, 100 and 200 $mg.kg^{-1}_{bw}.d^{-1}$ Zymbal gland carcinomas, oral cavity squamous cell papilloma and carcinoma, skin squamous cell papilloma and carcinoma in both sexes - $B6C3F_1$ Mice / 0, 5, 100 and 200 $mg.kg^{-1}_{bw}.d^{-1}$ Zymbal gland carcinomas, mammary carcinomas and carcinosarcomas, increased tumour incidences in dosed mice (e.g. haemopoietic system), adrenals, ovary, liver, lung and preputial gland Overall (both species): LOAEL = 25 $mg.kg^{-1}_{bw}.d^{-1}$ US-EPA (2000) proposes $LOAEL_{ADJ}$ = 18 $mg.kg^{-1}_{bw}.d^{-1}$ (1)	NTP, 1986 Huff *et al.*, 1989 as cited *in* US-EPA, 2000
Human toxicological studies	Pliofilm rubber workers exposed to benzene *via* inhalation exposure route. Oral cancer risk estimate based on route-to-route extrapolation (see details in paragraph below).	Crump and Allen, 1984; Paustenbach *et al.*, 1993; Rinsky *et al.*, 1981; Rinsky *et al.*, 1987 as cited *in* US-EPA, 2000
	$1:10^{-6}$ Leukemia Unit Risks associated to an average concentration varying between 0.2 and 20 $\mu g.m^{-3}$	E.C., 1999 as cited *in* Baars *et al.*, 2001

(1) $LOAEL_{ADJ}$ = LOAEL adjusted from 5-day to 7-day exposure period because the value is later compared to an occupational study.

Animal studies: Oral rat and mice studies can be used to define a Threshold Level (TLhh) based on the 25 $mg.kg^{-1}bw.d^{-1}$ LOAEL (oral reference dose (RfD) proposed by US-EPA, 2000).

Human toxicological studies: US-EPA (2000) proposes an oral slope factor of $1.5\ 10^{-5}$ – $5.5\ 10^{-5}$ $(\mu g.kg^{-1}.d^{-1})^{-1}$ based on a cancer unit risk estimate ranging between $2.2\ 10^{-6}$ and $7.8\ 10^{-6}$ $(\mu g.m^{-3})^{-1}$. The resulting Risk Specific Dose (RSD) is comprised between $1.8\ 10^{-4}$ and $6.7\ 10^{-4}$

$mg.kg^{-1}.d^{-1}$ for a $1:10^{-5}$ oral lifetime cancer risk and thus corresponding to $1.8\ 10^{-5}$ – $6.7\ 10^{-5}$ $mg.kg^{-1}_{bw}.d^{-1}$ for a $1:10^{-6}$ oral lifetime cancer risk.

In a 2001 report, Baars et al. (2001) proposed an oral cancer risk (CR_{oral}) of 3.3 $\mu g.kg^{-1}_{bw}.d^{-1}$ for a $1:10^{-4}$ excess lifetime cancer risk intake derived by route-by-route extrapolation from inhalation exposure data on humans following EU working group conclusions (E.C., 1999). This value corresponds to a CR_{oral} of $3.3\ 10^{-5}$ $mg.kg^{-1}_{bw}.d^{-1}$ for a $1:10^{-6}$ excess lifetime cancer risk intake.

Both tentative TL_{hh} derived from human toxicological studies are consistent. The lowest one is chosen.

Tentative $QS_{biota\ hh}$	**Relevant data for derivation of QS**	**AF**	**Threshold Level** ($mg.kg^{-1}_{bw}.d^{-1}$)	**Tentative $QS_{biota,\ hh}$**
Human health	$LOAEL_{ADJ}$ = 18 $mg.kg^{-1}_{bw}.d^{-1}$	3 000 (1)	RfD 0.006	365 $\mu g.kg^{-1}_{biota\ ww}$ corresponding to 28 $\mu g.l^{-1}$ (fresh and marine waters)
	Epidemiological studies: RSD proposed by US-EPA (2000) / $1:10^{-6}$ lifetime cancer risk (oral exposure)		RSD $1.8\ 10^{-5}$	1.1 $\mu g.kg^{-1}_{biota\ ww}$ corresponding to 0.084 $\mu g.l^{-1}$ (fresh and marine waters)

(1) Global assessment factor (AF) validated by US-EPA accounting for : an AF of 10 for extrapolation from LOAEL to NOAEL, an AF of 10 accounting for interspecies variability, an AF of 10 accounting for intraspecies variability and an AF of 3 accounting for database deficiencies.

Tentative $QS_{biota,\ hh}$ based on animal studies are less conservative and epidemiological studies may be preferable to animal studies to derive a $QS_{biota,\ hh}$ in the sense that they avoid extrapolation due to intra and interspecies variability. Moreover, a thorough comparison of supporting studies has been made by US-EPA in their most recent assessment (US-EPA, 2000) leading to the use of epidemiological data. Therefore, **the lower limit based on carcinogenicity risk – as called for by Member States – and epidemiological studies of $1.8\ 10^{-5}$ $mg.kg^{-1}_{bw}.d^{-1}$ proposed by US-EPA should be retained as the TL_{hh} for the derivation $QS_{biota,\ hh}$.**

As regards protection of human health from **consumption of drinking water**, there exists a **regulatory standard** in the Directive 98/83/EC (Drinking Water Directive) which is 1 $\mu g.l^{-1}$. Since the water-equivalent of the above-calculated $QS_{biota,hh}$ is stricter than the EU drinking water standard, the derivation of a QS for water intended for the abstraction of drinking water ($QS_{dw,\ hh}$) could be considered unnecessary. However, the calculation is presented below.

The following treatment achievability values were obtained from different sources:

	EUREAU (2010)	Member State response	WHO, 2008
Simple treatment	< 40%	-	-
Activated carbon	40% – 80%	100%	80% or more
Ozonation	40% – 80%	inadequate technique	80% or more
UV	40% – 80%	-	-

Combination of 2 advanced oxidation techniques	40% – 100%	-	-

Variations in the data provided by EUREAU in 2005 led to a further data-gathering round in 2010. The data received were little changed compared with those provided earlier. The WHO figure (WHO, 2008) is associated with reference to an achievable concentration of 0.01 mg/l (cf the EC drinking water standard of 1 $\mu g.l^{-1}$).
Applying a worst case assumption (simple treatment with <40% removal) and using the equations in the Draft Guidance Document on EQS derivation (E.C., 2010), the $QS_{dw, hh}$ can be calculated as follows:

$$QS_{dw,hh} = \frac{\text{drinking water standard (98/83/EC)}}{F_{\text{not removable by treatment}}} \quad \text{or} \quad QS_{dw,hh} = \frac{MPC_{dw,hh}}{F_{\text{not removable by treatment}}}$$

with $F_{\text{not removable by treatment}}$ = 60% – 100%

where the **"provisional drinking water standard"** ($MPC_{dw, hh}$) may be calculated directly from a calculated TL_{hh} retrieved from the literature, in this case based on the available studies used for derivation of $QS_{biota, hh}$.

$$MPC_{dw,hh} = \frac{0.1 \cdot TL_{hh} \cdot bw}{uptake_{dw}}$$

assuming a human body weight (bw) of 70 kg, a daily uptake of drinking water ($uptake_{dw}$) of 2 litres and a contribution of drinking water to total exposure of 10%.

Human health via consumption of drinking water		**Master reference**
Existing drinking water standards / guidelines	**EC drinking water standard** **1 $\mu g.l^{-1}$**	Directive 98/83/EC
	WHO guideline for drinking water **10 $\mu g.l^{-1}$** History of WHO Water Quality Guideline (WQG) development: - 1st edition WHO WQG (WHO, 1984; WHO, 1993): recommendation of a 10 $\mu g.l^{-1}$ value corresponding to a $1:10^5$ lifetime oral risk cancer based on data for the production of leukaemia after inhalation exposures in humans and using a linear multistage extrapolation model. - 2nd edition WHO WQG (WHO, 1996): As data on the carcinogenic risk to humans following the ingestion of benzene are not available, risk estimates were also carried out on the basis of the 2-year gavage study in rats and mice (NTP, 1986, Huff et al., 1989). The robust linear extrapolation model was used, as there was a statistical lack of fit of some of the data with the linearized multistage model. The estimated range of concentrations in drinking-water corresponding to excess lifetime cancer risks of 10^{-4}, 10^{-5}, and 10^{-6}, based on leukaemia and lymphomas in female mice and oral cavity squamous cell carcinomas in male rats, are 100-800, 10-80, and 1-8 $\mu g.l^{-1}$, respectively. These estimates are similar to those derived from epidemiological data, which formed the basis for the previous guideline value of 10 $\mu g.l^{-1}$ associated with a 10^{-5} excess lifetime cancer risk. Therefore, the recommended WQG remained the same.	WHO, 1984; WHO, 1993 WHO, 1996 WHO, 2008

Human health via consumption of drinking water		Master reference
	- 3rd (current) edition of WHO WQG (WHO, 2008): the recommendation remained the same.	
Calculated provisional drinking water standard $MPC_{dw, hh}$	Based on US-EPA, 2000 and E.C., 2010 using an RSD of 1.8 10^{-5} $mg.kg^{-1}_{bw}.d^{-1}$: **0.063 µg.l^{-1}**	
Standards for raw water intended for abstraction of drinking water $QS_{dw, hh}$	**Existing standard** suggested in EQS datasheet for benzene (2005): **1.7 µg.l^{-1}**	
	Standard calculated according to E.C. 2010 **using EC drinking water standard** and worst-case treatment achievability of <40%**:** $QS_{dw, hh}$ = **1 – 1.67 µg.l^{-1}**	
	Standard calculated according to E.C. 2010 **using provisional drinking water standard** $MPC_{dw, hh}$ and worst-case treatment achievability of <40%**: 0.06 – 0.11 µg.l^{-1}**	

No background document is publicly available that reports the original reasoning behind the regulatory standard of Directive 98/83/EC. Even though supporting studies are reported in the WHO Water Quality Guidelines report, there are very few indications on the calculation of the guideline itself. Parameters such as the contribution of drinking water to total exposure (default value of 10% in TGD EQS calculation, unknown in WHO calculations) or average body weight (70kg in TGD EQS calculation, 60kg in WHO calculations) may account for part of the difference between the EC and WHO values.

Since the existing EC drinking water standard and the calculated provisional drinking water standard differ by a factor of approximately 15, the $QS_{dw, hh}$ calculated from these differ similarly.

As indicated above, if the water-equivalent of the above-calculated $QS_{biota,hh}$, which is stricter than the EU drinking water standard, is accepted, the derivation of a QS for water intended for the abstraction of drinking water ($QS_{dw, hh}$) may be considered unnecessary according to the Draft Guidance Document on EQS derivation (E.C., 2010). If the water-equivalent is not accepted, the proposed $QS_{dw, hh}$ would be the value derived from the preferred regulatory standard, i.e. from the EC standard.

8 BIBLIOGRAPHY, SOURCES AND SUPPORTIVE INFORMATION

Baars A.J., Theelen R.M.C., Janssen P.J.C.M., Hesse J.M., van Apeldoom M.E., Meijerink M.C.M., Verdam L. and Zeilmaker M.J. (2001). Re-evaluation of human-toxicological maximum permissible risk levels. RIVM report 711701 025. RIVM, Bilthoven. March 2001. http://www.rivm.nl/bibliotheek/rapporten/711701025.pdf.

Berry W.O. and Brammer J.D. (1977). "Toxicity of Water-Soluble Gasoline Fractions to Fourth-Instar Larvae of the Mosquito *Aedes aegypti* L." Environmental Pollution **13**: 220-234.

Black J.A., Birge W.J., McDonnell W.E., Westerman A.G., Ramey B.A. and Bruser D.M. (1982). The aquatic toxicity of organic compounds to embryo-larval stages of fish and amphibians. Water Resources Research Institute, Lexington, NTIS PB-82-224601. 1982.

Blum D.J.W. and Speece R.E. (1991). "A database of chemical toxicity to environmental bacteria and its use in interspecies comparisons and correlations." Research Journal WPCF **63**(3): 198-207.

Bringmann G. and Kühn R. (1980). "Comparison of the toxicity thresholds of water pollutants to bacteria, algae and protozoa in the cell multiplication inhibition test." Water Research **14**(3): 231-241.

Caldwell R.S. and al. e. (1977). Effects of a Seawater-Soluble Fraction of Cook Inlet Crude Oil and Its Major Aromatic Components on Larval Stages of the Dungeness Crab, *Cancer magister* Dana. In: D.A. Wolfe (Eds.). Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms, Pergamon Press. pp. 210-220.

Chiou C.T., Porter P.E. and Schmedding D.W. (1983). "Partition equilibria of nonionic organic compounds between soil organic matter and water." Environmental Science & Technology **17**(4): 227-231.

Crump K.S. and Allen B.C. (1984). Quantitative estimates of risk of leukemia from occupational exposure to benzene. Prepared for the Occupational Safety and Health Administration by Science Research Systems, Inc., Ruston, LA. Unpublished

DeGraeve G.M., Elder R.G., Woods D.C. and Bergman H.L. (1982). "Effects of naphthalene and benzene on fathead minnows and rainbow trout." Arch. Environ. Contam. Toxicol. **11**: 487-490.

E.C. (1999). Benzene: Risk Assessment - Chapter 2. Commission of European Communities, Council Directive on Ambient Air Quality Assessment and Management, Working group on Benzene. January 1999.

E.C. (2003). Technical Guidance Document on Risk Assessment in support of Commission Directive 93/67/EEC on Risk Assessment for new notified substances, Commission Regulation (EC) N° 1488/94 on Risk Assessment for existing substances, Directive 98/8/EC of the European Parliament and of the Council concerning the placing of biocidal products on the market. Office for Official Publications of the European Communities, Luxembourg

E.C. (2004). Commission staff working document on implementation of the Community Strategy for Endocrine Disrupters - a range of substances suspected of interfering with the hormone systems of humans and wildlife (COM(1999) 706)). SEC(2004) 1372. European Commission, Brussels

E.C. (2007a). Summary Risk Assessment Report for Benzene (CAS-No.: 71-43-2, EINECS-No.: 200-753-7) (Final approved version). Institute for Health and Consumer Protection - European Chemicals Bureau. 24.10.2007.

E.C. (2007b). Commission staff working document on implementation of the "Community Strategy for Endocrine Disrupters" - a range of substances suspected of interfering with the hormone systems of humans and wildlife (COM(1999) 706), COM(2001) 262) and SEC (2004) 1372) SEC(2007) 1635. European Commission, Brussels. 30.11.2007.

E.C. (2008a). European Union Risk Assessment Report for Benzene (CAS-No.: 71-43-2, EINECS-No.: 200-753-7) (Final approved version). Institute for Health and Consumer Protection - European Chemicals Bureau. 2008.

E.C. (2008b). Regulation (EC) No 1272/2008 of the European Parliament and of the Council of 16 December 2008 on classification, labelling and packaging of substances and mixtures, amending and repealing Directives 67/548/EEC and 1999/45/EC, and amending Regulation (EC) No 1907/2006 (Text with EEA relevance). Official Journal of the European Union. **L353:** 1355.

E.C. (2010). Draft Technical Guidance Document for deriving Environmental Quality Standards (January 2010 version). Not yet published

ECHA (2008). Chapter R.8: Characterisation of dose [concentration]-response for human health. European Chemicals Agency. May 2008.

Folkins H.O. (1985). Benzene. In. Ullmann's Encyclopedia of Industrial Chemistry. 5th ed., VCH Verlagsgesellschaft mbH, Weinheim. pp. 475-505.

Freier R.K. (1976). Aqueous Solutions - Data for Inorganic and Organic Compounds. Berlin/New York, Verlag.

Galassi S., Mingazzini M., Vigano C.D. and Tosato M.L. (1988). "Approaches to Modeling Toxic Responses of Aquatic Organisms to Aromatic Hydrocarbons." Ecotoxicology and Environmental Safety **16**: 158-169.

Groshart C. and Okkerman P.C. (2000). Towards the establishment of a priority list of substances for further evaluation of their role in endocrine disruption: preparation of a candidate list of substances as a basis for priority setting. Final report (incorporating corrigenda to final report dated 21 June 2000). BKH Consulting Engineers, Delft, The Netherlands; in association with TNO Nutrition and Food Research, Zeist, The Netherlands

Herman D.C., Inniss W.E. and Mayfield C.I. (1990). "Impact of volatile aromatic hydrocarbons, alone and in combination, on growth of the freshwater alga Selenastrum capricornutum." Aquatic Toxicology **18**(2): 87-100.

Hodson P.V., Dixon D.G. and Kaiser K.L.E. (1984). "Measurement of median lethal dose as a rapid indication of contaminant toxicity to fish." Environ. Toxicol. Chem. **3**: 243-254.

Huff J.E., Haseman J.K., DeMarini D.M., Eustis S., Maronpot R.R., Peters A.C., Persing R.L., Chrisp C.E. and Jacobs A.C. (1989). "Multiple-site carcinogenicity of benzene in Fischer 344 rats and B6C3F1 mice." Environ Health Perspect **82**: 125-63.

Hutchinson T.C., Hellebust J.A., Tam D., Mackay D., Mascarenhas R.A. and Shiw W.Y. (1980). "The correlation of the toxicity to algae of hydrocarbons and halogenated hydrocarbons with their physical-chemical properties." Environ. Sci. Res. **16**: 577-586.

James A., Bonnomet V., Morin A. and Fribourg-Blanc B. (2009). Implementation of requirements on Priority substances within the Context of the Water Framework Directive. Contract N° 07010401/2008/508122/ADA/D2. Final dradt prioritisation process report on monitoring-based ranking., INERIS / IOW**:** 58.

Janssen C.R. and Persoone G. (1993). "Rapid Toxicity Screening Tests for Aquatic Biota. 1. Methodology and Experiments with Daphnia magna." Environ. Toxicol. Chem. **12**: 711-717.

Koenemann H. (1981). "Quantitative Structure-Activity Relationships in Fish Toxicity Studies." Toxicology **19**: 209-221.

Kusk K.O. (1980). "Effects of Crude Oils and Aromatic Hydrocarbons on the Photosynthesis of Three Species of Acrosiphonia Grown in Laboratory." Bot. Mar. **13**: 587-593.

Kusk K.O. (1981). "Effects of Hydrocarbons on Respiration, Photosynthesis and Growth of the Diatom Phaeodactylum tricornutum." Bot. Mar. **24**: 413-418.

Marchini S., Tosato M.L., Norberg-King T.J., Hammermeister D.E. and Hoglund M.D. (1992). "Lethal and sublethal toxicity of benzene derivatives to the fathead minnow, using a short-term test." Environmental Toxicology and Chemistry **11**(2): 187-195.

Meyerhoff R.D. (1975). "Acute Toxicity of Benzene, a Component of Crude Oil, to Juvenile Striped Bass (Morone saxatilis)." J. Fish. Res. Board. Can. **32**: 1864-1866.

Moles A. and al. e. (1979). "Sensitivity of Alaskan Freshwater and Anadromous Fishes to Prudhoe Bay Crude Oil and Benzene." Trans. Am. Fish. Soc. **108**: 408-414.

Niederlehner B.R., Cairns J. and Smith E.P. (1998). "Modeling acute and chronic toxicity of nonpolar narcotic chemicals and mixtures to *Ceriodaphnia dubia*." Ecotoxicology and Environmental Safety **39**: 136-146.

NTP (1986). National Toxicology Program. Toxicology and carcinogenesis studies of benzene (Cas N°71-43-2) in F344/N rats and B6C3F$_1$ mice (gavage studies). United states departement of Health and Human Services, serie 289 - n°86-2545

Paustenbach D.J., Bass R.D. and Price P. (1993). "Benzene toxicity and risk assessment, 1972-1992: implications for future regulation." Environmental Health Perspectives **101 Suppl 6**: 177-200.

Petersen G., Rasmussen D. and Gustavson K. (2007). Study on enhancing the Endocrine Disrupter priority list with a focus on low production volume chemicals. DHI, 53559

Pickering Q.H. and Henderson C. (1966). "Acute toxicity of some important petrochemicals to fish." J. Water Poll. Contr. Fed. **38**(1419-1429).

Potera F.T. (1975). The Effects of Benzene, Toluene and Ethylbenzene on Several Important Members of the Estuarine Ecosystem. PhD Thesis, Leigh University.

Price K.S., Waggy G.T. and Conway R.A. (1974). "Brine shrimp bioassay and seawater BOD of petrochemicals." J. Water Pollution Control Federation **46**(1): 63-77.

Rinsky R.A., Young R.J. and Smith A.B. (1981). "Leukemia in benzene workers." American Journal of Industrial Medicine **2**: 217-245.

Rinsky R.A., Smith A.B., Hornung R., Filloon T.G., Young R.J., Okun A.H. and Landrigan P.J. (1987). "Benzene and leukemia: an epidemiologic risk assessment." New England Journal of Medicine **316**(17): 1044-1050.

Rogerson A., Wan Ying S., Guo Lan H., Mackay D. and Berger J. (1983). "Determination and interpretation of hydrocarbon toxicity to ciliate protozoa." Aquatic Toxicology **3**(3): 215-228.

Russom C.L. and S.J. B. (1991). A chronic aquatic toxicity database for development of predictive toxicology models for industrial organic chemicals. US EPA, Environmental Research Laboratory-Duluth., Deliverable No. 8477, PPA: L104/G/2013 toto.

Sangster J. (1989). "Octanol-water partition coefficients of simple organic compounds." J. Phys. Chem. Ref. Data. **18**(3).

Sloof W. and al. e. (1983). "Benthic Macroinvertebrates and Water Quality Assessment: Some Toxicological Considerations." Aquatic Toxicology **4**: 73-82.

Tatem H.E.e.a. (1978). " The Toxicity of Oils and Petroleum Hydrocarbons to Estuarine Crustaceans." Estuarine and Coastal Marine Science **6**: 365-373.

TNO (2000). Determination of the effect of benzene on the growth of the freshwater green algae Selenastrum capricornutum (OECD, GL 201), V2360/01

Trucco R.G., Engelhardt F.R. and Stacey B. (1983). "Toxicity, accumulation and clearance of aromatic hydrocarbons in *Daphnia pulex*." Environ. Pollut. (Series A) **31**: 191-202.

Turnbull H., Demann J.G. and Weston R.F. (1954). "Toxicity of various refinery materials to fresh water fish." Ind. Eng. Chem. **46**(2): 324-333.

Uchrin C.G. and Mangels G. (1987). " Sorption equilibria of benzene and toluene on two New Jersey coastal plain ground water aquifier solids." Journal of Environmental Science and Health **A22**(8): 743-758.

US-EPA (2000). IRIS, Integrated Risk Information System - Benzene.

WHO (1984). Guidelines for Drinking-water Quality - First Edition, Volume 2 - Health criteria and other supporting information. WHO, Geneva

WHO (1993). Environmental Health Criteria 150: Benzene. World Health Organization, International Programme on Chemical Safety., Geneva, EHC 150 http://www.inchem.org/documents/ehc/ehc/ehc150.htm.

WHO (1996). Guidelines for Drinking-water Quality - Second Edition, Volume 2 - Health criteria and other supporting information - Chapter 14. Organic constituent. WHO, Geneva http://www.who.int/water_sanitation_health/dwq/2edvol2p2c.pdf.

WHO (2008). Guidelines for Drinking-water Quality - Third Edition Incorporating The First And Second Addenda, Volume 1 - Recommendations. WHO, Geneva http://www.who.int/water_sanitation_health/dwq/gdwq3rev/en/index.html.

Wong O. and Raabe G.K. (1995). "Cell-type-specific leukemia analyses in a combined cohort of more than 208,000 petroleum workers in the United States and the United Kingdom, 1937-1989." Regulatory Toxicology and Pharmacology **21**: 307-321.