Proposed Environmental Quality Standards for Nonylphenol in Water

R&D Note 475

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Statement of use
This Note reviews the available data on the use, fate/behaviour and aquatic toxicity of phenol. Environmental Quality Standards have been proposed which will assist NRA staff in assessing the effects of these substances on water quality.

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EXECUTIVE SUMMARY

This report, prepared for the National Rivers Authority, reviews the properties and uses of phenol, its fate, behaviour and reported concentrations in the environment and critically assesses the available data on its toxicity and bioaccumulation. The information is used to derive EQSs for the protection of fresh and saltwater life and for the abstraction of water to potable supply, Table S1.

Table S1 Proposed EQSs and guideline values for phenol (expressed as µg l^{-1})

Use	AA	MAC	Notes
Protection of freshwater life	30	300	
Protection of saltwater life	30	300	
Abstraction to potable supply	-	0.5	1,2

Notes
AA = Annual average
MAC = Maximum allowable concentration
1 Based on aesthetic considerations, see Appendix D
2 Lowest limit of detection reported as 0.034 µg l^{-1}

Phenol is widely used as a chemical intermediate and the main sources for phenol in the environment are of anthropogenic origin. Phenol may also be formed during natural decomposition of organic material.

The persistence of phenol in the aquatic environment is low with biodegradation being the main degradation process (half-lives of hours to days).

Phenol is moderately toxic to aquatic organisms and its potential to bioaccumulate in aquatic organisms is low.

The proposed EQS of 30 µg l^{-1} expressed as an annual average concentration (AA) for the protection of freshwater life against long-term exposure to phenol is derived by applying a safety factor of 100 to the lowest acute 48 hour LC50 of 3.1 mg l^{-1} reported for *Ceriodaphnia dubia*. In addition, an EQS of 300 µg l^{-1} expressed as a maximum allowable concentration (MAC) is proposed to protect against short term acute effects by applying a safety factor of 10 to the lowest observed acute toxicity value (48 hour LC50 value of 3.1 mg l^{-1} for *Ceriodaphnia dubia*).

Insufficient data are available to derive separate standards for the protection of saltwater life. However, the data available indicate that salt and freshwater species have similar sensitivities. It is therefore proposed that standards derived for the protection of freshwater life should also be applied for the protection of marine life.

The EQS for abstraction to potable supply has been based on aesthetic considerations. In order to avoid taste and odour problems should no treatment or only physical-chemical treatment be available, an EQS (expressed as an MAC) for abstraction to potable supply of 0.5 $\mu g\, l^{-1}$ has been proposed.

Current analytical techniques should be adequate to monitor these standards.

KEY WORDS

Environmental Quality Standards (EQSs), phenol, aquatic toxicity, freshwater, saltwater

1. INTRODUCTION

Phenol is listed in Annex I of Classification and Labelling Directive 67/548/EEC (CEC 1967). Manufacturing and chemical uses of phenol that result in its release to air, are prescribed processes as defined under the Environmental Protection (Prescribed Processes and Substances) Regulations 1991.

The report reviews and critically assesses the information available on the inputs to and concentrations of phenol in the environment (Section 2), the analytical methods available for the analysis of phenol (Section 3), the fate and behaviour of phenol in the environment (Section 4 and Appendix A) and its aquatic and mammalian toxicity (Section 5 and Appendices B-D).

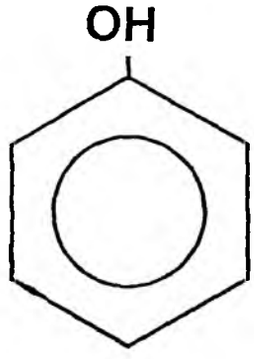
The information is used to derive Environmental Quality Standards (EQSs) for the protection of fresh- and saltwater life and for the abstraction to potable supply (Section 6).

2. PHENOL IN THE ENVIRONMENT

2.1 Physico-chemical properties

The physico-chemical characteristics of phenol outlined in Table 2.1 suggest that it is highly soluble in water and is unlikely to adsorb to suspended solids, sediments and soils to any significant extent. Volatilisation from soil surfaces should be rapid due to the high vapour pressure of phenol, although the relatively low Henrys Law Constant (H) indicates low potential for evaporation from water.

Table 2.1 Chemical and physical properties of phenol

IUPAC CHEMICAL NAME	Hydroxybenzene [2]
COMMON NAME	Phenol [2]
SYNONYMS	Carbolic acid, phenylic acid, phenyl hydroxide, phenyl hydrate monohydroxybenzene, monophenol, oxybenzene, phenyl alcohol, phenyl hydrate, PhOH, acidium carbolicum, acidium phenolicum, benzaphenol, benzene phenol, benzenol carbolic acid [1,2]
CAS NUMBER	108-95-2 [1]
MOLECULAR FORMULA	C_6H_6O [1]
MOLECULAR STRUCTURE	
MOLECULAR WEIGHT	94.11 [1]
COMPOSITION	C=76.57%, H=6.43%, O= 17.0% [1]
APPEARANCE	Colourless acicular crystals or white crystalline mass [1]
MELTING POINT (°C)	41.0-43.0 [1,2,3,4]
	40.9 (Ultrapure material) [2]
BOILING POINT (°C) (at 760 mm Hg)	181.75-182.0 [1,2,3,4]
VAPOUR PRESSURE (mm Hg)	0.357 (at 20 °C) [2]
	0.524 (at 25 °C) [4]
	2.48 (at 50 °C) [2]
	41.3 (at 100 °C) [2]
RELATIVE VAPOUR DENSITY (air =1)	3.24 [2]
VOLATILITY	Rapid from soil surfaces. Slow from water [2]
FLASH POINT (closed cup)	79.0 [1,2]

WATER SOLUBILITY (mg l^{-1})	82 000 at 15.0 °C [3] 67 000 at 16.0 °C [2]a 87 000 at 25.0 °C [4]
SOLUBILITY PRODUCT	No data
DISSOCIATION CONSTANT (pKa)	9.994 [4]
HENRY'S LAW CONSTANT (atm m^3 mol^{-1})	3.97×10^{-7} [4]
log K_{ow}	1.46 [2,4]
log K_{aw}	No data
log K_{oc}	No data
OXIDISING PROPERTIES	None. Sensitive to oxidising agents. Has antioxidant properties [2]

Notes:
a Above 68.4 °C phenol is entirely soluble in water

References:
1. Merck (1989)
2. WHO (1994)
3. Verschueren (1983)
4. Howard *et al.* (1989)

2.2 Manufacture

The most commonly used production method for phenol world-wide is by the peroxidation of cumene (Crookes *et al.* 1994). In the USA this process accounts for more than 98% of total phenol production (IARC 1989, cited in WHO 1994). In this process, cumene is first oxidised to cumene hydroperoxide by liquid phase air oxidation. The hydroperoxide is then cleaved with an acid catalyst to give phenol, acetone and by-products. The product stream is then washed and purified to yield phenol and acetone. By-products include acetophenone and α-methylstyrene. Phenol is also produced from chlorobenzene and by the oxidation of toluene. In addition, a small but steady supply of phenol is recovered as a by-product of metallurgical coke manufacture (IARC 1989, cited in WHO 1994).

World-wide production of phenol in 1981 was reported to be 3.3 million tonnes, with similar figures reported for 1986 (IARC 1989, cited in WHO 1994). The world capacity for production was estimated to be around 4.79 million tonnes in 1990, with a projected growth to around 5.5 million tonnes by 1995. World-wide consumption is thought to be around 500 000 tonnes below this figure, with 4.49 million tonnes consumed in 1990 (European Chemical News 1992, cited in Crookes *et al.* 1994).

Between 1976 and 1985 annual phenol production in the UK ranged from around 110 000-184 000 tonnes. However, this figure decreased to 52 946 tonnes in 1986 with the closure of two production plants (BP at Grangemouth and Shell at Stanlow). The remaining plant (ICI at Billingham) closed in 1992 and as a consequence phenol is no longer produced in the UK. Since 1986 there have been increases in net imports of phenol, corresponding with the closure of the UKs production plants (Crookes *et al.* 1994).

2.3 Uses

The largest single use of phenol is in the production of phenolic resins which are used as a binding material in, for example, insulation materials, chipboard and triplex, paints and casting sand foundries. Next in importance is its use as a chemical intermediate in the production of 2,2-bis-1-hydroxyphenylpropane (bisphenol A), a substance also used to manufacture phenolic resins. Other important uses are in the production of nylon-6, caprolactam, fibres, synthetics, aniline and polyphenylene oxide (WHO 1994 and Crookes *et al.* 1994).

Phenol was widely used in the 19th Century for wound treatment and as an antiseptic and local anaesthetic. The medical uses of phenol today include incorporation into disinfectants, antiseptics, lotions, salves and ointments. Another medical application is as a neurolytic agent to relieve spasm and chronic pain.

Other uses are in the manufacture of paint, varnish removers, lacquers, rubber, ink, illuminating gases, tanning dyes, perfumes, soaps and toys.

2.4 Entry into the aquatic environment

Phenol is a constituent of coal tar and is formed during the natural decomposition of organic material (e.g. during forest fires) (WHO 1994). However, most phenol in the environment arises as a result of anthropogenic emissions. Phenol is expected to enter the aquatic environment mainly via wastewater discharges and spills connected with its use in the production of phenolic resins and other commercial processes.

2.5 Recorded levels in the environment

In order to give an indication of the concentration of phenol present in the UK waters, regions of the National Rivers Authority and Scottish River Purification Boards have been approached for data and where available these are outlined below. In addition, so as to give an indication of world-wide distribution of phenol, various data on concentrations of phenol in waters and effluents are summarised below and examples given in Table 2.2.

2.5.1 Surface waters

Background concentrations of phenol in uncontaminated surface water generally range from <0.1-1.0 $\mu g\ l^{-1}$. Concentrations in industrial areas can be as high as 12-4 000 $\mu g\ l^{-1}$, while reported concentrations in surface waters arising from spills and high discharge levels range from 306-6 000 $\mu g\ l^{-1}$. Elevated levels have also been reported in surface waters as a result of urban run-off (3.0-10.0 $\mu g\ l^{-1}$) and run-off from a Hawaiian airport run-way during the rainy season (167.0 $\mu g\ l^{-1}$) (Crookes *et al.* 1994).

The River Dee incident in Welsh Region of the National Rivers Authority was a major incident involving phenol. The incident took place in January 1984 and had an impact upon drinking water supplies from six water treatment works supplying large areas of Deeside and Cheshire.

Many water companies received complaints regarding the unpleasant taste resulting from the phenol, which had been exacerbated by chlorination. Phenol concentrations has ceased to be a problem in the river by the end of the 26 January 1984, but the taste of the public supply was affected for several days after this. However, the concentration detected at one intake was still 1.5 mg l^{-1} on this date. Levels in the distribution system on the 26 and 27 January 1984 were less than 0.01 mg l^{-1}, but there were still taste effects. Following the incident and due to the particular sensitivity of the Dee catchment with respect to potable supply a system of frequent monitoring has been set up which involves twice daily monitoring at five points on the River Dee. Phenol is one of the parameters routinely monitored, the trigger level is >3 µg l^{-1}, above which water companies are monitored. Monitoring also takes place daily on two tributaries entering the Dee. As a result of this monitoring a substantial amount of data are available for phenol on the Dee (H Wilkinson, NRA Pers. Comm. 1995).

Welsh Region have a number of consented discharges for phenol including those for oil refineries, steel works, collieries and pharmaceutical and plastics manufacturers (H Wilkinson, NRA Pers. Comm. 1995).

Severn-Trent Region of the National Rivers Authority have reported to monitor for phenol in surface waters. In the majority of cases phenol was not detected. However, data have indicated phenol to be present in some surface waters in the 10-100 microgram range (maximum concentrations of 103, 119, and 221 µg l^{-1} have been reported) (Pers. Comm, A. Wallwork, NRA Severn-Trent Region, 1995). However, no additional data on the effects of these concentrations on the biota are available.

2.5.2 Ground waters

High phenol concentrations have been reported in ground waters associated with polluting industries. Concentrations of 40-460, 1 537-10 400 and 560-270 000 µg l^{-1} have been reported in ground waters in close proximity to industrial centres concerned with landfilling, timber treatment and coal/oil gasification, respectively. A concentration as high as 4.1×10^{6} µg l^{-1} has been reported in groundwater close to a waste processing facility in the US, although no other details are available (Crookes *et al.* 1994).

At a disused underground coal gasification site in the US, phenol concentrations in 1977 were reported to be 40 000 µg l^{-1}. By 1986 this level had decreased to around 1 000 µg l^{-1} by natural degradative processes. Further treatment of the groundwater by pumping through carbon adsorbers reduced the level to around <20.0 µg l^{-1} (Nordin *et al.* 1988, cited in Crookes *et al.* 1994).

In the UK, phenol has been detected in groundwater at a disused gasworks in the Welsh region which is undergoing reclamation. Levels of between 1-5 mg l^{-1} have been detected in boreholes and concentrations approaching 500 mg l^{-1} in water overlying a tar pit. In groundwater outside the site, however, concentrations had dropped to 29 µg l^{-1} (Pers. Comm. H. Wilkinson, NRA 1995).

A phenol concentration of 2.0 $\mu g\ l^{-1}$ has been reported in a Birmingham aquifer. Following complaints about taste and odour of water supplies, an investigation was conducted on three bore holes. At one of these sampling sites phenol was present at a concentration of 5.0 $\mu g\ l^{-1}$ (Leahy and Purvis 1979). The authors attributed this to the dumping of coke liquors in the area over a period of many years.

2.5.3 Sediments

Reported sediment concentrations in various rivers and estuaries from across the world range from 9.2-2 154 $\mu g\ kg^{-1}$ (dry weight), the upper value being recorded for Puget Sound in Washington (US), an area contaminated by waste disposal (mean value at this site = 337.0 $\mu g\ kg^{-1}$) (Meador *et al.* 1990, cited in Crookes *et al.* 1994).

A concentration of 10.0 $\mu g\ kg^{-1}$ (dry weight) has been reported in sediments within the discharge zone of a wastewater treatment plant in Los Angeles, USA. The corresponding effluent concentration at this site was 980.0 $\mu g\ kg^{-1}$ (Gossett *et al.* 1983).

2.5.4 Sewage sludge and sewage effluents

In a survey of 204 Michigan (USA) wastewater treatment plants (229 samples), 78% of samples were found to contain phenol concentrations ranging from 16.6-288 000 $\mu g\ kg^{-1}$ (median = 200 000 $\mu g\ kg^{-1}$) (Jacobs *et al.* 1987, cited in Crookes *et al.* 1994).

At a municipal sewage treatment plant in the US, phenol concentrations of 6.0-12.0 $\mu g\ l^{-1}$ have been reported in effluents from the primary process, while phenol was not detected in effluents from the secondary process (Pitt Jr. *et al.* 1975 cited in Crookes *et al.* 1994). However, in another survey concentrations of 89-1 400 $\mu g\ l^{-1}$ were recorded in 28% of effluents (50 sites) when the influent concentrations ranged from 1-1 400 $\mu g\ l^{-1}$ (US EPA 1982).

2.5.5 Industrial effluents

High concentrations of phenol have been measured in the effluents discharged from various industries. Effluents from coal gasification processes and chemical manufacturing plants have been reported to contain up to 200 000-6.6×10^{6} and 10-7 000 $\mu g\ l^{-1}$, respectively (levels cited in Crookes *et al.* 1994). In an untreated effluent from a dye manufacturing plant, phenol was present at levels at 110-910 $\mu g\ l^{-1}$, while phenol was undetected in the treated samples (Games and Hites 1977, cited in Crookes *et al.* 1994).

2.5.6 Biota

A survey was conducted on fish in the Columbia River (USA) following high water concentrations after the eruption of Mount St Helens in 1980. Phenol was measured above the detection limit 8.0 $\mu g\ kg^{-1}$ in 3 out of 14 samples. Concentrations of 35.0, 56.0 and 26.0 $\mu g\ kg^{-1}$ were reported in tissues of trout, sturgeon and salmon, respectively. However, the

study failed to detect significant levels of phenol in water (<0.18 $\mu g\ l^{-1}$) and sediments (<41.0 $\mu g\ kg^{-1}$). The authors attributed to the elevated levels in fish tissues to surface run-off after a fire in the area involving phenol (Campbell *et al.* 1982, cited in Crookes *et al.* 1994).

Following discharge of an effluent containing a phenol concentration of 980.0 $\mu g\ l^{-1}$ from a wastewater treatment plant in Los Angeles (USA), phenol was not detected (detection limit = 10.0 $\mu g\ kg^{-1}$) in the livers of Pacific sanddab, halibut, scorpion fish, dover sole, croaker and shrimp and other invertebrates (Gossett *et al.* 1983).

2.5.7 Drinking water

In a UK survey, phenol was detected in 5 out of 14 drinking supplies (no quantitative data available) drawn from both ground and surface water sources (Fielding *et al.* 1981).

Reported concentrations in drinking water range from 0.0008-0.357 $\mu g\ l^{-1}$ (Crookes *et al.* 1994).

2.5.8 Rain water

Although strictly an atmospheric source, it is worthy to note that phenol may enter the aquatic environment by way of contaminated rain water. Concentrations as high as 7.7 and 8.0 $\mu g\ l^{-1}$ have been reported in rain water falling on Los Angeles (USA) and Switzerland, respectively (Crookes *et al.* 1994).

Lueneberger *et al.* (1985) measured concentrations of phenol in seven rainfall events in Portland, USA. The highest concentration recorded was one of 1.2 $\mu g\ l^{-1}$, with a mean of >0.28 $\mu g\ l^{-1}$. The phenol air concentrations at the time of the events ranged from 0.22-0.43 $\mu g\ m^{-3}$.

Table 2.2 Reported environmental concentrations for phenol

Sample type	Sample site and details	Date	Lowest reported concentration (µg l^{-1})	Highest reported concentration (µg l^{-1})	Mean concentration (µg l^{-1} unless stated)	Ref
SURFACE						
Surface	Hayashida River, Japan. Highly polluted with leather industry effluents	-	-	-	306.0[1] 895.0[1]	1
Surface	Scheldt estuary, Netherlands	1972-1973	9.0	11.0		1
Surface	Scheldt estuary, Netherlands	1985-1988	1.0	2.0		1
Surface	32 North Sea locations in the UK where phenol is imported	-	-	<0.1[3]	<0.1[3]	1
Surface	River Dee, UK. Freshwater inputs to the Mersey estuary	-	<0.1	52.0	-	1
Surface	River Dee, UK.	-	-		0.601	1
Surface	Urban run-off to 51 catchments in USA. 86 samples	-	3.0[4]	10.0[4]	-	1
Surface	Arising from storm run-off during rainy season at Honolulu Airport, Hawaii.	-	-	-	167.0[5]	1
GROUND						
Ground	Near wood preserving facility in USA	-	-	10 400.0	-	1
Ground	Landfill leachate receiving mainly industrial waste, Poland	-	40.0	420.0	-	1
Ground	Near an old charcoal plant, USA	-	-	-	220.0	1
Ground	Near disused coal/oil gasification plant, USA	-	-	560.0	-	1
Ground	Near underground coal gasification plant, USA	-	-	10 200.0	-	1
Ground	At disused creosote factory, USA	-	-	18.4	-	1
Ground	Near waste processing facility, USA	-	-	4.1×10^{6}	-	1

Sample type	Sample site and details	Date	Lowest reported concentration (µg l^{-1})	Highest reported concentration (µg l^{-1})	Mean concentration (µg l^{-1} unless stated)	Ref
Ground	Aquifer, Birmingham, UK	-	-	2.0	-	
Ground	Bore hole in UK after complaints of taste and odour	-	-	-	5.0	1
Ground	37 municipal landfills in North Carolina, USA	-	-	>1.0[6]	-	1
Ground	Several coal gasification sites in USA prior to gasification process	-	15.0	20.0	-	1
Ground	Several coal gasification sites in USA after gasification process	-	50 000.0	270 000.0	-	1
Ground	Disused coal gasification site	1977	-	-	40 000.0	1
	After 9 years degradation by natural processes	1986	-	-	1 000.0	
	After further remediation treatment	1986	-	-	<20.0	
SEDIMENT						
Sediment	Commencement Bay, Washington, USA	-	-	-	66.0-399.0 µg kg^{-1} (dry weight)	1
Sediment	Puget sound, Washington, USA. Areas contaminated by waste disposal	-	-	2 154.0 µg kg^{-1} (dry weight)	337.0 µg kg^{-1} (dry weight)	1
Sediment	Los Angeles, USA. Discharge zone of wastewater treatment plant	-	-	-	10.0 µg kg^{-1} (dry weight)[7]	1
Sediment	Bottom sediments, Japan	1977	30.0 µg kg^{-1} (dry weight)[10]	40.0 µg kg^{-1} (dry weight)[10]	-	1
EFFLUENTS						
Sewage sludge	204 Michigan wastewater treatment plants, USA (229 samples)	-	16.6 µg l^{-1} (dry weight)[8]	288 000.0 µg kg^{-1} (dry weight)[8]	2 000.0 µg kg^{-1} (dry weight)[5]	1
Sewage effluent	50 sewage treatment plants, USA	-	89.0[9]	1 400.0[9]	-	1
Sewage effluent	Primary municipal treatment plant	-	6.0	12.0	-	1
	Secondary treatment	-	nd	nd	nd	

Sample type	Sample site and details	Date	Lowest reported concentration (µg l^{-1})	Highest reported concentration (µg l^{-1})	Mean concentration (µg l^{-1} unless stated)	Ref
Industrial effluent	Wastewater treatment plant, USA	-	-	-	980.0	1
Industrial effluent	Coal carbonisation process effluent	-	825 000.0	2.3×10^{6}	-	1
	- discharged to river		-	-	880.0	
Industrial effluent	Coal gasification process, USA	-	200 000.0	6.6×10^{6}	-	1
Industrial effluent	Coal gasification process, USA (untreated)	-	-	-	1.25×10^{6}	1
	- treated effluent	-	-	-		
Industrial effluent	Dye manufacturing plant (untreated)	-	110.0	910.0	-	1
	- treated effluent	-	nd	nd	nd	1
Industrial effluent	Effluent petroleum refineries, USA	-	880.0	3.8×10^{-6}	-	1
Industrial effluent	Oil Shale retorting wastewaters	-	-	-	10 000.0	1
Biota	Columbia River, USA. Trout[12]	1980	-	-	35.0 µg kg^{-1}	1
	Sturgeon[12]				56.0 µg kg^{-1}	
	Salmon[12]				26.0 µg kg^{-1}	
Biota	Los Angeles, USA. Discharge zone of wastewater treatment plant. Liver samples of Pacific sanddab, halibut, scorpion fish, dover sole, croaker and shrimp and other invertebrates					1

Notes:

nd	No detected
-	Data not seen or not reported
1	Results of two different analytical methods. Concentrations for one sample only
2	Only one value reported
3	At all locations
4	4% of samples taken
5	Median level

6 In 5% samples phenol detected above detection limit (1.0 $\mu g\ l^{-1}$)
7 Concentration in the plant effluent was 980 $\mu g\ l^{-1}$
8 78% of samples taken
9 28% of effluent samples. Influent concentrations = 1-1400 $\mu g\ l^{-1}$
10 3 out of 9 samples. Detection limits for the study ranged from 10-100 $\mu g\ kg^{-1}$ depending on method used. Not detected in the overlying surface waters
11 10-100 $\mu g\ l^{-1}$ detected in receiving waters
12 3 out of 8 samples above the detection limit of 8.0 $\mu g\ kg^{-1}$. Significant levels not detected in water or sediments. High levels in fish thought to be the result of surface water run-off after a fire in the area involving phenol
13 Detection limit = 10.0 $\mu g\ kg^{-1}$

References:

1. Cited in Crookes *et al.* (1994)

3. ANALYSIS

3.1 Analytical requirements for EQS monitoring

The adequate monitoring of EQSs requires a suitably accurate analytical method.

The accepted approach for the derivation of the accuracy requirements of an analytical system (when monitoring to a particular water quality standard) is described in WRc Report NS30 (Cheeseman *et al.* 1989).

For an EQS of X units, the error on a single analytical result should not be larger than X/10 concentration units or 20% of the concentration in the sample, whichever is the greater. Following the convention of dividing the tolerable error equally between random and systematic sources, this implies:

- a maximum tolerable standard deviation of X/40 concentration units or 5% of the concentration in the sample, whichever is the greater; and
- a maximum tolerable bias of X/20 concentration units or 10% of the concentration in the sample, whichever is the greater.

It is recommended that the target limit of detection should be set at X/10 concentration units.

For example, for a proposed EQS of 1 mg l^{-1}:

- the limit of detection should be 0.1 mg l^{-1} or less;
- the total error should not exceed 0.1 mg l^{-1} or 20% of the determinand concentration (whichever is the greater);
- the systematic error or bias should not exceed 0.05 mg l^{-1} or 10% of the determinand concentration (whichever is the greater); and
- the total standard deviation of individual results should not exceed 0.025 mg l^{-1} or 5% of the determinand concentration (whichever is the greater).

3.2 Analytical Techniques

A number of methods for the analysis of phenol in river and drinking waters have been produced as "Blue Books" by the Standing Committee of Analysts (SCA) (HMSO 1988). These are outlined below:

SCA Method A)

A sample of 42 ml water is taken and phenol derivatised in-situ to the corresponding pentafluorobenzoyl ester. The derivative is then extracted into hexane and determined using electron-capture gas chromatography (GC-ECD) with the following conditions.

column	10M fused silica capillary - BP5 (SE54 equivalent) 0.33 mm id, 0.5 µ coating thickness
temperature	Inj - 230 °C, Det - 250 °C,
	Col - 130 °C for 1 min then 3 °C min^{-1} to 145 °C then 15 °C min^{-1} to 165 °C then 25 °C min^{-1}
	to 250 °C. Hold for 3 mins.
gases	He carrier (0.40 Bar)
	N_2 make up (30 ml min^{-1} total) to detector
injection vol	1.5 µl (0.15 min residence)

The limit of detection is 0.038 µg l^{-1} and the range of application 0-1 µg l^{-1}. The average recovery for a 0.2 µg l^{-1} solution is 88.5% with a 7.4% RSD. (number of samples (n) = 7).

SCA Method B)

A water sample of 100 ml is distilled, brominated and the resulting 2,4,6-tribromophenol extracted into n-hexane and analysed by GC-ECD under the following conditions.

column	glass 1.5 m x 4mm id 4.5% Carbowax 20M on 80 - 100 Anakrom Q
temperature	Det - 300 °C, Col - 170 °C, Inj - 250 °C
	carrier gas N_2 ; 60 ml min^{-1} (oxygen free)
Injection vol	5 µl

The limit of detection is 0.25 µg l^{-1} with a range of application up to at least 10 µg l^{-1}. The standard deviation for the method was estimated as 0.1 µg l^{-1} at concentrations of 0.6-5 µg l^{-1} (no value given for n).

SCA Method C)

A sample of 100 µl is injected directly via a sample loop onto a 15 cm 5 µ PRP - 1 divinyl-benzene/polystyrene copolymer column followed by reverse phase high performance liquid chromatography (HPLC) using coulometrically efficient electrochemical detection with the following settings.

Detector 1 potential = 250 mV

Detector 2 potential = 650 mV

Guard cell potential = 750 mV

Gain (detector 2) = 150

Response time (det 2) = 10 seconds

The mobile phase used is 1:1 acetonitrile : 10 mM dibasic sodium phosphate solution at a flow rate of 0.5 ml min^{-1}. The limit of detection is 0.034 µg l^{-1} and the range of application up to at least 100 µg l^{-1}. The mean recoveries were 96.6% at 0.5 µg l^{-1} with a RSD of 3.9% (n = 7), and 100% at 1 µg l^{-1} with a RSD of 3.4% (n = 7).

SCA Method D)

Phenol is extracted from water using either solid phase extraction (SPE) on a C18 column (a1) or automated sample preconcentration on a reverse phase precolumn (a2). The extract was then analysed for phenol by either isocratic HPLC using orthophosphoric acid buffer : methanol : ethanol (b1), gradient HPLC using methanol/phosphate buffer mobile phase with UV detection (b2) or HPLC with gradient elution using 0.1% aqueous acetic acid and methanol (b3). The combinations used were a1 + b1, a2 + b2 and a1or a2 + b3.

The HPLC operating conditions are:

b1)	buffer	45:40:15 0.01M H3PO4 : methanol : ethanol
	flowrate	0.5 ml min^{-1}
	column	2 x 100 mm Chromosphere C18, 3 mm id, 5 µ particle size
	column temp	45 °C
	detector	UV 214 nm
b2)	elution gradient	70 : 30 5 mM phosphate buffer : methanol for 2.5 mins change to 25 : 75 linearly over 22 mins change to 0 : 100 linearly over 2 mins and maintain for 5 mins.
	column	Ultrasphere IP C18 250 x 4.6 mm id, particle size 5 µ
	flowrate	1.5 ml min^{-1}
	detector	UV 280 nm

b3)	elution gradient	90 : 10 0.1% acetic acid : methanol for 2 mins change to 60 : 40 linearly over 3 mins change to 0 : 100 linearly over 30 mins
	flowrate	1 ml min^{-1}
	column temp	30 °C
	detector	UV 214 nm and 254 nm
	column	C18

The limit of detection for these four SPE methods is 2.35 µg l^{-1} and the methods are tested up to 10 µg l^{-1} - it was stated that the recovery may be substantially less than 100% at higher concentrations. The methods are mainly intended for investigations and confirmation. Using a C8 precolumn and a concentration of 10 µg l^{-1} phenol in the water sample the recovery was 35.7% with a RSD of 4.2% (n = 3).

A method has also been produced by the US EPA (EPA method 604) for the analysis of phenol in waste water, drinking water and surface waters. In the EPA method a sample of one litre was adjusted to pH 2 with H_2SO_4, extracted sequentially with two 60 ml aliquots of dichloromethane, dried with sodium sulphate and, after adding 2-propanol, the solvent evaporated to a final volume of 1 ml. The extract was then analysed by GC with flame ionisation detection using the following conditions:

column	Supelcoport 80/100 mesh, 1% SP-1240DA
carrier gas	Nitrogen at 30 ml min^{-1}
column temp	initially 80 °C then 8 °C min^{-1} to 150 °C and hold

The range of application was 12 - 450 µg l^{-1} and the limit of detection 0.14 µg l^{-1}.

Many other methods for analysis of phenol are reported in the scientific literature and two of these are summarised in Table 3.1.

Table 3.1 Other Analytical Methods for phenol

Compounds Determined	phenol, methyl phenols, chlorophenols, nitrophenols and nitroquinones	phenolics
Matrices	drinking, ground and rain waters	unpolluted or slightly polluted natural waters
Concentration Range	up to 0.25 µg l^{-1}	1 - 30 µg l^{-1}
Extraction	concentrated using continuous liquid-liquid extraction with light phase rotary perforator, derivatized with diazomethane or trimethylsulfonium hydroxide	phenolics are steam distilled from the sample in an automated system under acid conditions (see notes)
Clean-up	none required	none required
Analysis	GC-MS using DB 5 30 m x 2.5 mm id column and temperature program, He carrier gas at 2 ml min^{-1}	absorbance of the chloroform extract is measured at 480 nm
Limit of Detection	50 ng l^{-1}	1 µg l^{-1}
Accuracy	41.5% recovery at 100 and 250 ng l^{-1} using diazomethane (n =5)	60 - 120% recovery at 2.6 µg l^{-1} (n = 10)
Precision	3.9% SD at 100 ng l^{-1} 5.1% SD at 250 ng l^{-1}	5.8% RSD at 2.6 µg l^{-1}
Notes		distillate reacted with 4 -aminoantipyrene at pH 10 with potassium persulphate to form a coloured antipyrine dye which is extracted into chloroform
Reference	Geiβler and Schöler (1994)	Carron and Afghan (1989)

4. FATE AND BEHAVIOUR IN THE ENVIRONMENT

This section summarises the main findings of the assessment of the fate and behaviour of phenol in the environment, in particular, the aquatic environment. A detailed assessment is outlined in Appendix A.

The main degradation process of phenol in the aquatic environment is biodegradation which is rapid (hours to days) and occurs under both aerobic and anaerobic conditions. Phenol is not expected to evaporate or hydrolyse in water. It is unlikely to bioconcentrate in aquatic organisms to any significant extent (see Sections 5.3 and Appendices A-C).

Direct photolysis could occur, although under most circumstances this process appears to be of minor importance compared with biodegradation. Under abiotic conditions phenols react rapidly in sunlit natural waters exhibiting typical half-lives of the order of days (Hwang *et al.* 1986).

In water and soils, phenol exists in a partially dissociated state. Therefore, its transport and reactivity will be dependent on the pH. Despite its high solubility and poor adsorption to soil particles, however, data for groundwater indicates that contamination by phenol is infrequent which is probably due to its rapid degradation by microbial populations. However, because of its moderately high volatility (27 Pa at 20 °C), volatilisation of phenol from the soil surface may also occur. An estimated half life for phenol volatilisation has been quoted as 1.8 days at a depth of 1 cm (Jury *et al.* 1984).

The concentration of phenol in influent to sewage treatment works varies greatly depending on the source of the sewage. The rapid anaerobic biodegradation and mineralisation of phenol by denitrifying bacteria (see Appendix A), will result in the removal of phenol during sewage treatment processes. As a result concentrations in sewage effluent (1 - 89 $\mu g\ l^{-1}$) have been recorded as being considerably lower than the influent (US EPA 1982, Staples *et al.* 1985). Concentrations of phenol in sewage sludge also show wide variation depending on the influent concentration and efficiency and type of treatment works. Any residual phenol in sewage sludge applied to land however, would be subject to rapid biodegradation or volatilisation, unless applied during a period of high rainfall which could result in a certain fraction of the phenol being solubilised.

5. SUMMARY OF AQUATIC TOXICITY AND BIOACCUMULATION IN AQUATIC ORGANISMS

This Section summarises the most reliable data on the aquatic toxicity and bioaccumulation of phenol, Appendices B and C discuss the data more fully.

5.1 Toxicity to freshwater organisms

The symptoms displayed by fish exposed to phenol include increased respiration, increased mucous secretion, muscular seizures, depressed activity and loss of equilibrium, indicating a narcotic effect on the central or peripheral nervous system. These symptoms are thought to be brought about by interference with enzyme function. ATP production and oxidative phosphorylation to produce effects consistent with nervous paralysis.

Fish appear to be the most sensitive aquatic organisms to phenol exposure, with acute and chronic LC50s ranging from approximately 3.0-49.9 and 0.12-2.67 mg l^{-1}, indicating moderate toxicity to this group of organisms, although the reliability of the lowest chronic LC50s has been questioned (see Appendix B). However, a 38 day NOECs for growth and hatch of 1.83 and 3.57 mg l^{-1} which are considered reliable have been reported for *Pimephales promelas* (Holcombe *et al.* 1982). Sub-lethal effects include inhibition of various parameters of fecundity following 30 days exposure to 12.0 mg l^{-1} and avoidance behaviour after 48 hours exposure to 5.3-8.1 mg l^{-1}. These effects are important since effects on fecundity in the field could lead to recruitment failure in fish populations, while avoidance may force fish into unsuitable habitats although this may only be temporary.

Similar in sensitivity are the crustaceans. The water flea genera, *Daphnia* and *Ceriodaphnia* appear be to the most sensitive to phenol exposure, with acute effect concentrations ranging from 3.1 mg l^{-1} and higher. Chronic NOECs of 0.16 and 0.5 mg l^{-1} based on growth and mortality, respectively, have been reported for *Daphnia magna* and the data reported by Cowgill and Milazzo (1991), 48 hour LC50 of 13 mg l^{-1} and an 11 day LC50 (mortality of 4 mg l^{-1} indicate potentially low acute to chronic ratio for this substance.

Acute toxicity data for other crustacean species range from 21.0-180.0 mg l^{-1}.

Reliable toxicity data for insects, rotifers, molluscs, platyhelminthes and annelids, and algae and macrophytes range from 15.5-260.0, 59.0-780.0, >51.0-264.5, 200.0-870.0 and 61.1-370.0 mg l^{-1}, respectively. This order of relative species sensitivity broadly agrees with the findings of other authors (Alekseyev and Antipin 1976 and Walker 1988, cited in Green *et al.* 1985 and Crookes *et al.* 1994, respectively). The lowest reliable toxicity data reported in the literature have been summarised in Table 5.1.

Table 5.1 Lowest reliable freshwater toxicity data reported for phenol

Species	Life stage	Test type	Analysis	Exposure time	Concn (mg l^{-1})	Effect	Ref
ACUTE							
ALGAE							
Selenastrum capricornutum (Green unicell)	-	S[1]	nominal	4 d	61.1	EC50 (Growth)	1
MOLLUSCS							
Physa fontinalis (Snail)	-	F	measured	48 h	80.0-120.0	LC50	3
ROTIFERS							
Brachionus calyciflorus (Rotifer)	Neonates	S[2]	nominal	2 d	59.0	EC50 (population increase)	4
	Neonates	S[2]	nominal	2 d	25.0	NOEC (population increase)	4
ARTHROPODS (CRUSTACEANS)							
Daphnia magna (Water flea)	<24 h	S	measured	48 h	4.2	EC50[3]	5
Daphnia magna (Water flea)	Adult	SS	nominal	48 h	13.0	LC50	6

Species	Life stage	Test type	Analysis	Exposure time	Concn (mg l^{-1})	Effect	Ref
Ceriodaphnia dubia (Water flea)	Neonates	SS	measured	48 h	3.1	LC50	8
			measured	4 d	5.3	EC50 (no of young per female)	8
ARTHROPODS (INSECTS)							
Baetis rhodani (Mayfly)	Larvae	F	measured	96 h	15.5	LC50	3
FISH (NON-SALMONID)							
Pimephales promelas (Fathead minnow)	30-35 d	F	measured	96 h	28.5	LC50	9
		S	measured	48 h	8.3		9
Pimephales promelas (Fathead minnow)	0.9-13.1	F[4]	measured	96 h	25.3	LC50	10
FISH (SALMONID)							
Oncorhynchus mykiss (Rainbow trout)	5.2-11.3cm	SS	measured	48 h	5.4	LC50	12
					8.0	LC50	12
					9.8	LC50	12
AMPHIBIANS							
Xenopus laevis (African clawed toad)	Tadpole	F	measured	48 h	>51.1	LC50[5]	13

Species	Life stage	Test type	Analysis	Exposure time	Concn (mg l^{-1})	Effect	Ref
CHRONIC							
Scenedesmus quadricauda *(Green algae)*	-	S	nominal	7 d	8.0	Threshold of toxicity (growth)	2
Daphnia magna (Water Flea)	Adult	SS	nominal	11 day	4.0	LC50	6
	Adult	SS	nominal	11 day	0.5	NOEC (mortality)	6
Daphnia magna (Water flea)	-	SS	nominal	16 day	0.16	NOEC (growth)	7
Pimephales promelas (Fathead minnow)	eggs to 38 day post-hatch	F	measured	38 day	3.57	NOEC (Hatch)	11
	eggs to 38 day post-hatch	F	measured	38 day	1.83	NOEC (Growth)	11

Notes:

F	Flow-through
S	Static
SS	Semi-static
h	Hours
d	Days
-	Data not seen or not reported

1	Test conducted in shake flask culture.
2	Test conducted in test tubes.
3	Immobilisation referred to by authors as 'mortality'
4	Rainbow trout and fathead minnow tested simultaneously in screened compartments
5	Tested in a multi-species bioassay along with 6 other organisms representing Platyhelminthes, Molluscs, Crustaceans and fish.

References:

1. St Laurent *et al.* (1992) cited in Crookes *et al.* (1994)
2. Bringmann and Kuhn (1980)
3. Green *et al.* (1985)
4. Snell and Moffat (1992)
5. Lewis (1983)
6. Cowgill and Milazzo (1991)
7. De Neer *et al.* (1988)
8. Oris *et al.* (1991)
9. Phipps *et al.* (1981)
10. Sulaiman (1993)
11. Holcombe *et al.* (1982)
12. Brown *et al.* (1967)
13. Holcombe *et al.* (1987)

It is apparent that temperature can influence the toxicity of phenol, although evidence concerning its effects appear to be conflicting and is difficult to assess with respect to the derivation of standards.

5.2 Toxicity to saltwater organisms

The toxicity of phenol to saltwater organisms appears to be similar to that for freshwater organisms, with the majority of short-term effect concentrations ranging from 10.4-185.0, 52.6-58.3, 7.4-171.74 and 10.0-19.99 mg l^{-1} for algae, molluscs, crustaceans and fish, respectively. Sub-lethal effects in fish can occur at concentrations as low as 1.0-5.0 mg l^{-1}. In addition, saltwater fish display a similar concentration dependent pattern of characteristic neurotoxic symptoms as freshwater species. The lowest reliable toxicity data reported in the literature have been summarised in Table 5.2.

5.3 Bioaccumulation in fresh and saltwater organisms

Phenol is not expected to accumulate to particularly high levels in freshwater organisms. The majority of reliable Bioconcentration Factors (BCFs) range from 3.5-16.0, although dry weight values (which can be expected to be an order of magnitude higher than wet weight values) as high as 277-1375 have been reported for the water flea, *D. magna*. The low experimental BCFs support a calculated BCF of 5.7 based on the octanol-water partition coefficient of phenol (log Kow 1.46). Uptake and attainment of steady state equilibrium appears to be rapid (generally within hours). Depuration also appears to be rapid on transferring exposed organisms to clean water, with some authors reporting evidence of biotransformation.

Similar findings have been reported for saltwater organisms. A BCF of just 8.75 has been found for the mud crab, *Panopeus herbstii,* following 96 hours exposure to 2.0 mg l^{-1}, with rapid but incomplete depuration (48%) recorded after 96 hours in clean water.

Table 5.2 Lowest reliable saltwater toxicity data reported for phenol

Species	Life stage	Test type	Analysis	Exposure time	Concn (mg l^{-1})	Effect	Ref
ALGAE							
Skeletonema costatum (Diatom)	-	-	nominal	5 d	49.6	EC50 (Total cell count)	1
					49.8	EC50 (Total cell volume)	
Champia parvula (Red alga)	Mature female	S[2]	nominal	14 d	21.6	LOEC (Growth - dry weight)	2
	Tetrasporo- phyte				10.4	LOEC (growth - dry weight)	2
MOLLUSCS							
Mercenaria mercenaria (Clam)	Eggs	SS	nominal	48 h	52.6	EC50 (Normal development)	6
	2 d			12 d	55.0	LC50	
ARTHROPODS - CRUSTACEANS							
Tisbe battagliai (Copepod)	Copepodid	S	measured	24 h	171.74	LC50	3
Palaemonetes pugio (Grass shrimp)	-	-	nominal	24 h	53.0	LC50	4
				48 h	11.0	LC50	4
				96 h	7.4	LC50	4

Species	Life stage	Test type	Analysis	Exposure time	Concn (mg l^{-1})	Effect	Ref
FISH							
Solea solea (Sole)	45±2.5 g	SS	measured	96 h	14.23	LC50	3
Mugil auratus (Grey mullet)	125 g	F[2]	measured	8 d	0.5	Normal behaviour. No changes in gross pathology or blood chemistry	5
				8 d	7.5	Excited, sensitive to light, later depressed activity. Severe changes in blood chemistry. 10% mortality	6
				5 h	10.0	Loss of equilibrium. Haemorrhaging. Severe changes in blood chemistry. 25% mortality	5
				1 h	25.0	Convulsions and suffocation. Severe changes in blood chemistry. 50% mortality	6

Notes:

SS Semi-static
F Flow through
h Hours
d Days
- Data not seen or not reported

1 Test solution renewed on days 7 and 11
2 Despite constant dosing the measured concentration dropped to 50% of the initial value. This was apparently due to proliferation of phenolic bacteria. The dosing system was therefore constantly adjusted to compensate for this

References

1. Cowgill *et al.* (1989)
2. Thursby *et al.* (1985)
3. Smith *et al.* (1994)
4. Tatem *et al.* (1978)
5. Krajnovic-Ozretic and Ozretic (1988)
6. Davis and Hidu (1969), cited in Crookes *et al.* (1994)

6. DERIVATION OF EQSs

6.1 Standards in other countries

No standards for phenol for the protection of aquatic organisms in other countries have been found.

6.2 Protection of freshwater life

Data on the aquatic toxicity and bioaccumulation of phenol to freshwater organisms are outlined in Appendix B and summarised in Section 5. The data indicate that phenol is of low to moderate toxicity to freshwater aquatic organisms with fish and crustaceans being the most sensitive organisms (acute LC50 values in the low mg l^{-1}).

Few chronic data are available for fish and crustaceans. The reliability of data for more sensitive fish species has been questioned and these data are therefore, not used for the derivation of EQSs (see Appendix B).

Crustacea appear to be as sensitive as fish to phenol. Cowgill and Milazzo (1991) report a 48 hour LC50 of 13 mg l^{-1}. Oris *et al.* (1991) report a 48 hour LC50 of 3.1 mg l^{-1} for *Ceriodaphnia dubia*, and Deneer *et al.* (1988) examined the effect of phenol on the growth of *D. magna* and reported EC10 and NOEC values of 0.46 and 0.16 mg l^{-1} respectively. Additionally, the authors reported a 16 day EC50 (reproduction) for *D. magna* of 10 mg l^{-1}.

An EQS (expressed as an annual average (AA) concentration) of 130 µg l^{-1} could be derived from the 48 hr LC50 for *D. magna* reported by Cowgill and Milazzo (1991) by applying a safety factor of 100 to the data. Alternatively, applying a safety factor of 100 to the lowest acute 48 hour LC50 of 3.1 mg l^{-1} reported for *Ceriodaphnia dubia* (Oris *et al.* 1991) would result in an EQS of 30 µg l^{-1}. Because of the absence of reliable chronic studies for indigenous fish species, and other freshwater species which may be more sensitive, the lower EQS of 30 µg l^{-1} (expressed as an AA concentration) is proposed for the protection of freshwater life against long term exposure of phenol. This should be reviewed when further chronic data for crustaceans or fish become available.

An EQS, expressed as a maximum allowable concentration (MAC), of 300 µg l^{-1} is also proposed to protect against short term episodic events. This is derived by applying a safety factor of 10 to the lowest acute toxicity value (a 48 hour LC50 of 3.1 mg l^{-1} for *Ceriodaphnia dubia*).

6.3 Protection of saltwater life

As outlined in Section 5.2 and discussed in more depth in Appendix C, the acute toxicity of phenol to saltwater organisms appears to be similar to that reported for freshwater organisms. In addition, saltwater fish display a similar concentration dependent pattern of characteristic

neurotoxic symptoms as freshwater species. While there are too few data to assess the acute/chronic/NOEC ratios for saltwater species, the available data suggest that a standard in the same range as that proposed for freshwaters will be sufficient to protect saltwater organisms. Therefore Environmental Quality Standards of 30 $\mu g\ l^{-1}$, expressed as an annual average and 300 $\mu g\ l^{-1}$ expressed as a maximum allowable concentration are proposed.

6.4 Abstraction of water for potable supply

Data on the mammalian toxicity of phenol is outlined in Appendix D. The US Health Advisory values for phenol for one day, ten day and longer term are set at 6 $mg\ l^{-1}$ based on a 10 kg child drinking one litre of water a day. The RfD (reference dose) and lifetime levels set for a 70 kg adult drinking two litres of water a day are 0.6 $mg\ kg^{-1}\ day^{-1}$ and 4 $mg\ l^{-1}$ respectively. These values are based on the health effects of chronic exposure as reported in the NTP teratology study and NCI carcinogenicity study. However, the aesthetic effects of phenol contamination of drinking water will become apparent two orders of magnitude below these values. The current standard for phenol in the Water Supply (Water Quality) Regulations (HMSO 1989) is 0.5 $\mu g\ l^{-1}$ which is based on aesthetic considerations. It is therefore proposed, in order to avoid taste and odour problems should no treatment or only physical-chemical treatment be available, that the EQS (expressed as an MAC) for abstraction to potable supply be 0.5 $\mu g\ l^{-1}$.

7. CONCLUSIONS

1. Phenol is highly soluble in water and is unlikely to adsorb to suspended solids, sediments and soils to any significant extent. Volatilisation from soil surfaces should be rapid due to the high vapour pressure of phenol, although the relatively low Henrys Law Constant (H) indicates low potential for evaporation from water.

2. If phenol is released to water, its primary removal route will be by biodegradation which will be rapid (hours to days) and occurs under both aerobic and anaerobic conditions. Phenol is not expected to evaporate or hydrolyse in water. It is unlikely to bioconcentrate in aquatic organisms to any significant extent.

3. In water and soils, phenol exists in a partially dissociated state. Therefore, its transport and reactivity will be dependent on the pH. However, despite its high solubility and poor adsorption to soil particles, data for groundwater indicates that contamination by phenol is infrequent probably due to its rapid degradation by microbial populations and because of its moderately high volatility (27 Pa at 20 °C), volatilisation of phenol from the soil surface may also occur.

4. Fish appear to be the most sensitive aquatic organisms to phenol exposure, with acute and chronic LC50s ranging from approximately 3.0-49.9 and 0.12-2.67 mg l^{-1}, indicating moderate toxicity to this group of organisms, although the reliability of the lowest chronic LC50s has been questioned. Crustaceans are similar in sensitivity with the water flea *Daphnia* and *Ceriodaphnia* the most sensitive tested.

5. The toxicity of phenol to saltwater organisms appears to be similar as for freshwater organisms.

6. Phenol is not expected to accumulate to particularly high levels in the majority of freshwater organisms. The majority of reliable Bioconcentration factors (BCFs) range from 3.5-16.0. Depuration also appears to be rapid on transferring exposed organisms to clean water.

7. An EQS expressed as an annual average (AA) concentration of 30 µg l^{-1} and a Maximum Allowable Concentration (MAC) of 300 µg l^{-1} are proposed for the protection of freshwater and saltwater life.

8. An EQS of 0.5 µg l^{-1} based on aesthetic considerations is proposed for abstraction of potable water.

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APPENDIX A FATE AND BEHAVIOUR IN THE ENVIRONMENT

Numerous studies have been undertaken to investigate the environmental fate and behaviour of phenol, and in particular it's biodegradation in water. This section and Table A1 summarise the most pertinent studies that have been conducted.

A1. FATE AND BEHAVIOUR IN SOIL

From the available literature it is apparent that the major process of phenol decomposition in soil is through biodegradation.

A1.1 Abiotic processes

Due to its chemical properties under normal environmental conditions phenol would not be expected to hydrolyse in soil (Howard 1989). As the major abiotic route for the degradation of phenol in water is photolysis (Section A2.1), photolysis may occur at the very surface of soils, although it would still be considered insignificant with respect to biological degradation.

A1.2 Biodegradation

Biodegradation is by far the most important removal process for phenol in soils.

A1.2.1 Aerobic Biodegradation

Aerobic biodegradation of phenol has been shown to be both rapid and extensive in soils (Crookes *et al.* 1994). *Pseudomonas putida*, for example, isolated from a soil enriched culture has been shown to be capable of degrading phenol up to a concentration of 1 g l^{-1}. Above this level inhibition of cell growth was observed (Hinteregger *et al.* 1992). Investigations using soil slurries have shown that phenol mineralisation occurred over a period of 8 to 32 hours, although the maximum rate of mineralisation decreased with increasing depth of soil (Baker and Mayfield 1980). Other authors have measured phenol mineralisation down to a soil depth of 20 m with a half life of 13.4 hours (Federle 1988).

A1.2.2 Anaerobic Biodegradation

Again anaerobic biodegradation of phenol in soils has been shown to be widespread (Crookes *et al.* 1994). Many experiments have utilised bacteria isolated from sewage treatment work's anaerobic sludge (see Section A3). Carboxylation of phenol to 4-hydroxybenzoate is thought to be the initial reaction in the degradation of phenol by denitrifying *Pseudomonads* (Tschech and Fuchs 1987).

Concentrations of phenol up to 189 mg l^{-1} have been shown to be degraded by bacteria isolated from organic-rich mud collected from the North Sea (Bak and Widdel 1986). No significant degradation was measured, however, for phenol spiked clay loam soil with no added nutrients (Baker and Mayfield 1980).

A1.3 Sorption

The high water solubility of phenol means that its affinity for sorption to soil is low. As a result it does not persist in soil, it is either washed out, or rapidly biodegraded.

In experiments where very high concentrations of soil have been used (50 g l^{-1}), the percentage of iron oxides present in the soil has had a bearing on phenol adsorption (Artiola-Fortuny and Fuller 1982). Unlike clays, iron oxides show a strong tendency to react with anions as well as cations, leading to soils containing iron oxides to have a greater affinity for phenol. Another study (Phillips 1964) using a sandy loam (4.15% organic matter), a silty loam (3.28% organic matter) and a peaty loam (30.6% organic matter), produced soil organic carbon/water partition coefficient (Koc) values of 137, 131 and 82 respectively. The low values reflect the water soluble nature of phenol. Other authors have derived Koc values for phenol of 30 (2.51% organic matter, Briggs 1981), 72 (Bahnick and Doucette 1988), and 405 (0.84% organic carbon, Dao and Lavy 1987). A batch equilibrium experiment using phenol adsorbed to a clay loam (5.1% organic matter) produced a Koc value of 16.1 (assuming % C = % organic matter/1.9) (Boyd 1982). The relatively low affinity of phenol for adsorption to soil organics, means that the rate of biodegradation is unlikely to be increased through adsorption to particulates.

A1.4 Volatility

Phenol has a moderately high vapour pressure (27 Pa at 20 °C) and so volatilisation may occur from spills onto land (Howard 1989). An estimated half life for the volatilisation of phenol from soil has been quoted as 1.8 days at a depth of 1 cm and 20.9 days at a depth of 10 cm (Jury *et al.* 1984).

A2. FATE AND BEHAVIOUR IN WATER

Because of the high solubility of phenol in water, there have been many studies investigating the fate and behaviour of phenol in aquatic systems. The fact that phenol is relatively stable chemically, but undergoes rapid biodegradation, has meant that research has focused on the biological aspects of phenol degradation.

A2.1 Abiotic processes

It is not thought that phenol will hydrolyse significantly under typical environmental conditions (Howard *et al.* 1989). The major abiotic process associated with phenol is photodecomposition, via reactions with photochemically produced hydroxyl and peroxyl radicals. Typical half lives for these radicals are 100 and 19.2 hours respectively (Mill *et al.* 1982, cited in Howard *et al.* 1989). It has been reported that p-benzoquinone and hydroxyquinone are the major decomposition products (Lipcznska and Kochany 1992), following irradiation with xenon lamps. There was little effect of pH from 3.0 to 8.0, but yields increase markedly between a pH of 9.0 and 10.0 due to the enhanced absorption of light by the phenolate ion in the 250-350 nm wavelength range (pKa of phenol = 10).

Half lives for phenol photodecomposition in estuarine waters have been quoted as 48 hours (Hwang *et al.* 1986), with half lives for mineralisation to carbon dioxide varying from 16 days in summer to around 150 days in winter (Hwang *et al.* 1987). Enhanced photo-mineralisation has been observed for phenol bound to humic acids (which acts as photosensitising agents) (Amador and Alexander 1991), for phenol in water containing blue-green and green algae (Zepp and Schlotzhauer 1983) and also through catalysis by manganese oxides (Stone 1987). Lumichrome, the product of the photolysis of riboflavin, has also been shown to sensitise the photodecomposition of phenol, albeit at enhanced concentrations than those expected in the environment (Tatsumi *et al.* 1992).

In water treatment studies, addition of hydrogen peroxide (1%) to water containing phenol (60 mg l^{-1}), has been shown to result in a 78% reduction in phenol in 60 minutes (Yue and Legrini 1992), compared with only 10% in 60 minutes for 0.05% H_2O_2. Ultra-violet light facilitates the breakdown of the peroxide to hydroxyl radicals which then promote the oxidation of phenol. Complete photodegradation of phenol in water passed over Titanium dioxide (TiO_2) coated glass (Al-Ekabi and Serpone 1988), has been shown to occur in 6 hours (half life of 18-100 minutes), with degradation exhibiting first order kinetics.

A2.2 Biodegradation

The bulk of research on the fate and behaviour of phenol in water has concentrated on biodegradation. A selection of relevant studies are discussed below.

A2.2.1 Aerobic degradation

Bacteria, fungi, yeasts and other micro-organisms have been shown to utilise phenol as a sole source of carbon and energy (Rast *et al.* 1980, Kotturi *et al.* 1991, Hofrichter *et al.* 1993). Two pathways have been identified in the aerobic degradation of phenol, both of which involve hydroxylation of phenol to catechol as the first step. Catechol then undergoes either ortho-fission to give initially cis, cis muconic acid and then B-ketoadipate and finally acetyl-CoA and succinate, or meta-fission to give initially 2-hydroxy muconic semialdehyde and finally acetaldehyde and pyruvate. The final step in both cases is the formation of carbon dioxide and water (Crookes *et al.* 1994).

Rates of mineralisation are often proportional to the initial phenol concentration (e.g. ca 0.05 ng ml^{-1} hr^{-1} for a concentration of 1 ng ml^{-1} and 300 ng ml^{-1} hr^{-1} for a concentration of 1 mg ml^{-1}, Rubin *et al.* 1982); whether adsorbed to sediment or dissolved (Shrimp and Young 1988). Aerobic decomposition displays first order kinetics with measured rate constants being measured between 0.0001 to 0.01 h^{-1} (Swindoll *et al.* 1988), which can result in biodegradation rates of up to 2 mg l^{-1} day^{-1} (CEPS 1985, cited in Mallet *et al.* 1992).

Certain yeasts have been shown to grow in solutions of phenol concentration up to 1000 mg l^{-1} (Katayama-Hirayama *et al.* 1991 and 1992). Degradation of phenol in solution generally only takes a matter of hours, for instance, Pseudomonas degraded 90% of added phenol in 10 hours (D'Aquino *et al.* 1988) and Hutchinson and Robinson (1988) using the same species found that

both phenol and p-cresol (total concentration 135 mg l^{-1}) were removed from solution within 14 hours. Higher organisms have also been shown to rapidly mineralise phenol in aquatic systems. The marsh plant *Schoenoplectus* for example, has been shown to mineralise up to 200 mg l^{-1} of phenol in 60 hours (Gassner and Neugebohrn 1994).

A2.2.2 Anaerobic degradation

Similar to aerobic biodegradation numerous organisms have been shown to rapidly degrade and mineralise phenol under a variety of anaerobic conditions (Crookes *et al.* 1994), meaning phenol will not persist in anaerobic water/sediment. Phenol at concentrations of up to 1.4 g l^{-1} have been mineralised to methane and carbon dioxide within 350 hours, using batch methanogenic cultures that use nitrate as electron acceptors (Wang *et al.* 1988 and 1989). Although phenol at such high concentrations did result in a significant inhibition of microbial activity without actually stopping it. Denitrifying *Pseudomonads* are thought to carboxylate phenol to 4-hydroxybenzoate as the first step in the degradation pathway (Tschech and Fuchs 1987).

A2.3 Sorption

Due to the biodegradable nature of phenol in sediment/water systems, it is not always obvious whether the removal of phenol from solution during soil absorption studies is due to adsorption or biodegradation. As a result any studies not performed under sterile conditions must take such possibilities into consideration. Adsorption is thought, however, to be rapid (equilibrium attained within 10 minutes, Dao and Lavy 1987). The solution pH is considered to be the major limiting parameter in phenol adsorption. It has been shown that sorption will only occur when pH<pKa (Laquer and Manahan 1987), demonstrating the lack of affinity of sediment for the dissociated species. The percentage of iron oxides in a sediment is also likely to effect its adsorption capacity (Artiola-Fortuny and Fuller 1982), due to the ability of iron oxides to react with anions as well as cations, leading to soils containing iron oxides to have a greater affinity for phenol.

Although adsorption of phenol onto sediments is rapid, it's low log Kow value (1.46) means that adsorption to sediments is unlikely to significantly affect the rate of degradation by biota.

A2.4 Volatility

Phenol has a low Henry's Law constant (0.0402 Pa m^3 $mole^{-1}$) and would not be expected to volatilise to any significant extent from water unless highly aerated.

A3 BEHAVIOUR IN SEWAGE TREATMENT PROCESSES

The presence of phenol in wastewater is widespread (78% of sludges in a USA survey contained phenol, Jacobs *et al.* 1987). Influent concentrations have been shown to vary between 1 and 1400 µg l^{-1} in a survey of 50 US sewage treatment plants (USEPA 1982). Laboratory-scale experiments using anaerobic sewage sludge as an inoculum, acclimated to phenol for 6 months (4-6 mg l^{-1}), showed that up to 70% of the added phenol or 95% if glucose was also added were degraded (Hendricksen *et al.* 1991). The rapid mineralisation of

phenol by denitrifying bacteria in sludge digesters (Birch *et al.* 1989, O'Conner and Young 1989), means that concentrations in effluents are much lower (1-89 $\mu g\ l^{-1}$) than in influents (USEPA 1982, Staples *et al.* 1985).

Concentrations of phenol in sewage sludge vary widely (e.g. 0.0166 and 288 $mg\ kg^{-1}$, Jacobs *et al.* 1987) depending on the concentration in the influent sewage and also the efficiency of the treatment works. Any residual phenol present in the sewage sludge when applied to land would be rapidly biodegraded and so persistence would not be considered a potential problem. Due to its low adsorption, in soils some leaching could occur if rain falls very soon after application. In general for a typical secondary treatment works, the effluent concentrations are likely to be relatively low (e.g. 6 to 12 $\mu g\ l^{-1}$, Pitt Jr. *et al.* 1975, cited in Crookes *et al.* 1994) and are therefore unlikely to have an adverse effect on receiving water.

Table A1 Summary of fate and behaviour of phenol in soil

	Chemical	Laboratory, field or modelled data	Media	pH	Temp (°C)	Initial concn (mg l^{-1})	Final concn (mg l^{-1})	Half-life (Hours)	Degradation products	Comments	Ref
Abiotic processes											
Hydrolysis	Phenol	L	S					No significant hydrolysis			1
Biodegradation											
Subsurface microbes	^{14}C Phenol	L	S		17			rate const = 0.058 hr^{-1}	Carbon dioxide and water		2
Methanogenic consortia	Phenol	L	S		28	60	0	Disappearance in 20 days		Microbes in black anoxic Saale mud previously acclimated to cresol	3
Microbial degradation	^{14}C Phenol	L	Slurry soil		24	0.005-10 mg kg^{-1}			Carbon dioxide and water	Mineralisation decreased with depth	4
Microbial degradation	^{14}C Phenol	L	S		24	750 ng in 15 g soil		Rate varied between 0.31 and 3.3 day^{-1} Half life of 13.4 hrs	Carbon dioxide and water	Soils from two sites from USA, incubated for 64 days. 20-25% moisture content,	5
Microbial degradation	Phenol	L	S		20	700 mg kg^{-1}		24-130		Alluvial soil from Colorado river, some volatilisation also occurred	6

	Chemical	Laboratory, field or modelled data	Media	pH	Temp (°C)	Initial concn (mg l^{-1})	Final concn (mg l^{-1})	Half-life (Hours)	Degradation products	Comments	Ref
Microbial degradation	Phenol	L	Clay		23	100 mg kg^{-1}		complete removal in 5 days		70% disappearance in 1 day.	7
by Marsh plants	Phenol	L	W			50-200	0	ca. 30 hours for 200 ppm	CO_2 + water	Marsh plants, e.g. Schoenoplectus	8
Sorption	Phenol	L	S/W		20					Three different organic carbon contents gave Koc values between 82-137	9
	Phenol	L	Silt /loam			20				Koc of 30 for organic matter content of 2.5% after 2 hours shaking	10
	Phenol	L	Clay /loam	5.7	21	5-50				Organic content 5.1%, gave Koc value of 16.1	11

	Chemical	Laboratory, field or modelled data	Media	pH	Temp (°C)	Initial concn (mg l^{-1})	Final concn (mg l^{-1})	Half-life (Hours)	Degradation products	Comments	Ref
Volatility											
	Phenol	L	S					40 hours		At 1cm (half life 20.9 days at 10cm)	12
	Phenol	L								Henry's law constant of 0.0402 Pa m^3 $mole^{-1}$, vapour pressure of 27 Pa at 20°C	1

Notes:

L	Results from a laboratory study
F	Results from a field study
M	Results generated by a model
W	Water
A	Air
S	Soil
Sed	Sediments
ND	No data
N	None

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1. Howard (1989)
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3. Kaminski *et al.* (1990)
4. Dobbins *et al.* (1987)
5. Federle (1998)
6. Namkoog *et al.* (1989)
7. Baker and Mayfield (1980)
8. Gassner and Neugebohrn (1994)
9. Phillips (1964)
10. Briggs (1981)
11. Boyd (1982)
12. Jury *et al.* (1984)

Table A 2 Summary of fate and behaviour of phenol in water

	Chemical	Laboratory field or modelled data	Media	pH	Temp (°C)	Initial concn (mg l^{-1})	Final concn (mg l^{-1})	Half-life (Hours)	Degradation products	Comments	Ref
Abiotic processes											
Photolysis	Phenol	L	W	3.5	40-45	10-60	ca. 10	15mins for 1% peroxide addition		Photolysis of phenol in the presence of hydrogen peroxide, enhanced through production of hydroxyl radicals	1
Photolysis	Phenol	L	Water + TiO_2 on glass		25-35			ca. 1	Carbon dioxide and water	First order kinetics for degradation.	2
Photolysis	Phenol	L	W	3-11					Hydroxy-quinone and benzoquinone	Little difference in degradation rate between pH 3 ad 8 but increased above pH10 due to formation of phenoate anion which absorbs UV more strongly	3
Photolysis	Phenol	L	W		20	1.9		176		Sample containing 4.1 mg l^{-1} fulvic acid, irradiated in sun for 4 hours at noon in Zurich	4

	Chemical	Laboratory field or modelled data	Media	pH	Temp (°C)	Initial concn (mg l^{-1})	Final concn (mg l^{-1})	Half-life (Hours)	Degradation products	Comments	Ref
Photolysis	Phenol	L	W	8				3.3		Enhanced photooxidation by addition of lumichrome at wavelength of over 320nm. Concentrations much higher than found in environment though	5
Photolysis	Phenol	L	Estuarine water					48			6
Biodegradation											
Microbial	^{14}C-Phenol	L	settled bottom sediments		20	0.008 - 2.051		Rate constant of 0.0025hr^{-1}	Carbon dioxide + water	Adsorbed phenol degradation was a function of initial concentration and was bio-available	7
Degradation by algae	Phenol	L	W	7		700-1000	0			Degradation by Scenedesmus, Spirulina and Chliorella	8
Microbial degradation	^{14}C Phenol	L	W	6.6-8.9	25	0.0003-2			Carbon dioxide and water	Degradation of phenol shown to be multiphasic	6

	Chemical	Laboratory field or modelled data	Media	pH	Temp (°C)	Initial concn (mg l^{-1})	Final concn (mg l^{-1})	Half-life (Hours)	Degradation products	Comments	Ref
Microbial degradation	Phenol	L	Ground water	8.2	21	0-3.2		20	Carbon dioxide and water		9
	Phenol	L	River water	7.8	21	0-3.2		11	Carbon dioxide and water		10
	Phenol	L	Harbour water	7.6	21	0-3.2		3	Carbon dioxide and water		10
Microbial (oligotrophs and eutrophs)	^{14}C phenol	L	Lake Water		29	$1x10^{-6}$ - 1.0				Degradation rate proportional to initial concentration	10
Pseudomonas	^{14}C Phenol	L	W		21	100		Complete removal in 3 days	Carbon dioxide and water		11
Mixed culture	Phenol	L	River water		15	19		Degradation after 20 hour lag of $3x10^{-12}$ mole.hr^{-1}		At 23^{0}C using same water 23 hr lag and rate of $9x10^{-13}$ mole.hr^{-1}	12
Rhodorula	Phenol	L	W		30	Up to 1000		Complete removal of 50 mg l^{-1} after 72 hours	Catechol, muconic acid, muconolactone, enol-lactone etc		13

	Chemical	Laboratory field or modelled data	Media	pH	Temp (°C)	Initial concn (mg l^{-1})	Final concn (mg l^{-1})	Half-life (Hours)	Degradation products	Comments	Ref
Microbial degradation	^{14}C Phenol	L	Seawater	7.3	15.5	0.0005-5			Carbon dioxide and water	Degradation in seawater and sediment. Stated experiments should be performed at environmental levels	14

Notes:

L	Results from a laboratory study
F	Results from a field study
M	Results generated by a model
W	Water
A	Air
S	Soil
Sed	Sediments
ND	No data
N	None

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9. Vaishnav and Babeu(1987)
10. Rubin and Alexander (1983)
11. Murakami and Alexander (1989)
12. Banjeree *et al.* (1984)
13. Katayama *et al.* (1992)
14. Ursin (1985)

Table A3 Summary of fate and behaviour of phenol in sewage treatment processes

	Chemical	Laboratory field or modelled data	Media	pH	Temp (°C)	Initial concn (mg l^{-1})	Final concn (mg l^{-1})	Half-life (Hours)	Degradation products	Comments	Ref
Biodegradation											
Anaerobic degradation	Phenol	L	W			0.3		Complete disappearance after 2 weeks	Carbon dioxide and methane	2.5 week acclimation period prior to degradation of phenol. Introduction of a second lot of phenol resulted in no lag and removal in 10 days.	1
Anaerobic degradation	Phenol	L	W		35	50-200		47-80% mineralised in 28-62 days			2
Anaerobic degradation	Phenol	L	W		37	20-200		Complete removal in 20-25 days	Carbon dioxide and methane	No lag measured	3

	Chemical	Laboratory field or modelled data	Media	pH	Temp (°C)	Initial concn (mg l^{-1})	Final concn (mg l^{-1})	Half-life (Hours)	Degradation products	Comments	Ref
Anaerobic degradation		L	W		35	Up to 1400		Complete removal in 350 hours	Carbon dioxide and methane	Concentrations greater than 600 mg l^{-1} inhibited degradation, but did not stop it.	4

Notes:

L	Results from a laboratory study
F	Results from a field study
M	Results generated by a model
W	Water
A	Air
S	Soil
Sed	Sediments
ND	No data
N	None

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APPENDIX B FRESHWATER TOXICITY AND BIOACCUMULATION

Toxicity and bioaccumulation data are shown in Tables B1 and B2, respectively. The most pertinent studies are discussed below.

B1. FRESHWATER TOXICITY

There is a vast amount of data in the literature concerning the toxicity of phenol to freshwater organisms. In addition, several reviews exist that summarise available data (e.g. WHO 1994, Crookes *et al.* 1994, Mallet *et al.* 1992 and Walker 1988). For this reason, only the lowest and more reliable toxicity data have been included in Table B1 and discussed in the text. Attention has also been paid to reliable studies not included in the aforementioned reviews. However, where data are scarce for a particular group of organisms, all available information has been included, regardless of reliability.

B1.1 Algae

Reported EC50s for algae range from 61.1-370 mg l^{-1}, for a wide range of exposure periods (4 hours to 14 days), indicating that phenol is of low acute and chronic toxicity to this group of organisms. A 7 day toxicity threshold (growth) of 8.0 mg l^{-1} has been reported for *Scenedesmus quadricauda* (Bringmann and Kuhn 1980).

B1.2 Macrophytes

Phenol also appears to be of low toxicity to aquatic macrophytes. Wang (1986) reported an EC50 based on growth for the duckweed, *Lemna minor*, of above 12.0 mg l^{-1}. This is supported by 7 day EC50s of 229-242 and 157-183 mg l^{-1} based on plant and frond number, and dry weight, reported for *L. gibba and L. minor,* respectively. The corresponding NOEC for *L. gibba* was found to be 14.0 mg l^{-1} (Cowgill *et al.* 1991).

B1.3 Molluscs

LC50s ranging from >51.0-264.5 mg l^{-1} have been reported for a variety of freshwater snails following 12-96 hours exposure, indicating low acute toxicity. The lowest reliable value is a measured LC50 of 80.0 mg l^{-1} reported for *Physa fontinalis*, following 48 hours under flow-through conditions (Green *et al.* 1985).

B1.4 Platyhelminthes

Phenol appears to be of low acute toxicity to flatworms with a 48 hour LC50 of 200.0 mg l^{-1} reported for *Polycelis tenuis* (Green *et al.* 1985).

B1.5 Annelids

Phenol appears to be of low acute toxicity to aquatic worms. LC50s of 870.0 and 780.0 mg l^{-1} have been reported for the Oligochaete, *Limnodrilus hoffmeiseri*, following 48 and 96 hours exposure, respectively (Green *et al.* 1985).

B1.6 Rotifers

A 24 hour LC50 of >150.0 mg l^{-1} has been reported by Snell *et al.* (1991) for the rotifer, *Brachionus calyciflorus*, indicating low acute toxicity. This is supported by a 24 hour LC50 of 780.0 mg l^{-1} reported for the same organism by Snell and Moffat (1992).

In an attempt to assess the effects of phenol on the intrinsic rate of population increase of *B. calyciflorus*, Snell and Moffat (1992) exposed neonates in laboratory bioassays. The test can be considered as a chronic exposure test as it measured reproduction over 2 generations. A 2 day EC50 (population increase) of 59.0 mg l^{-1} was reported, with corresponding NOEC and LOEC values of 25.0 and 43.0 mg l^{-1}, respectively, suggesting low chronic toxicity to rotifers. The authors attributed the effects on reproductive schedule and consequently population increase, to a delay in the time for the first brood to be produced and an increased interval period between births.

B1.7 Crustaceans

The water flea species, *Daphnia magna* and *Ceriodaphnia dubia* appear to be more sensitive to phenol exposure than other crustaceans and insects. Short and long-term EC50s based on immobilisation range from 4.2-37.17 mg l^{-1} for *D. magna*, the lowest value being a measured concentration obtained in a 48 hour static bioassay. The author (Lewis 1983) referred to immobilisation as 'mortality' in this study, although McCahon *et al.* (1990) have shown that immobilised crustaceans can recover on transferring to clean water. Therefore, the EC50 of 4.2 mg l^{-1} reported by Lewis could over estimate the toxicity of phenol. Corresponding LC50s for this species range from 4.0-13.0 mg l^{-1}, suggesting a small ratio between the concentrations which cause lethal and sub-lethal effects.

NOECs of 0.5 and 0.16 based on mortality, growth and reproduction, respectively, have been reported for *D. magna*, following respective exposure periods of 11 and 16 days respectively.

The non-indigenous cladoceran, *C. dubia* appears to be similar in sensitivity to *D. magna*, with short and long-term EC50s based on various parameters of fecundity ranging from 5.3-10.0 mg l^{-1} (corresponding *Daphnia* values range from 6.0-7.0 mg l^{-1}), indicating low chronic toxicity. LC50s for *C. dubia* range from 3.1-20.0 mg l^{-1}, following 48 hours exposure. These data indicate that phenol is of moderate acute toxicity to *Daphnia* spp. and *Ceriodaphnia* spp.

Phenol is of moderate to low acute toxicity to other crustacean species, with 96 hour LC50s ranging from 21.0-180.0 mg l^{-1}, the most reliable of which is a measured concentration of 69.0 mg l^{-1} reported for the freshwater shrimp, *Gammarus pulex*, following exposure under flow-through conditions (Green *et al.* 1985).

McCahon *et al.* (1990) investigated the effects of phenol to the water hog louse, *Assellus aquaticus,* and studied the relationship between response and measured body burdens of phenol including a recovery phase in which animals were transferred to clean water. Analysis of body phenol concentrations (see Section B2 for data) showed differences between immobilised and recovered animals, with values for the latter being significantly lower. The authors suggested that phenol must reach a certain concentration in the body before a sub-lethal response (immobilisation) is observed and that this level is independent of test temperature and aqueous phenol concentration. Similarly, when immobilised animals are transferred to clean water, recovery only occurs when sufficient phenol has been eliminated to reduce the body concentration to a level below a critical body burden. However, it is unclear how the three processes of absorption, detoxification and excretion differ under different temperature-toxicant regimes.

The study conducted by McCahon *et al.* (1990) clearly shows that temperature affects the toxicity of phenol to *A. aquaticus*, with increasing temperature decreasing the toxic response. A very similar response was reported by Green *et al.* (1985) for the same organism. However, many authors have found that the converse is true. This is clearly shown in a study conducted by Cowgill *et al.* (1985) in which 48 hour LC50s of 12.1 and 4.3 mg l^{-1} were reported for the water flea, *C. dubia*, exposed at temperatures of 20.0 and 24.0 °C, respectively.

B1.8 Insects

Phenol is of moderate to low acute toxicity to insect larvae, with the majority of LC50s ranging from 15.5 to 260.0 mg l^{-1}, the lower value being a measured concentration reported for the mayfly, *Baetis rhodani*, following exposure under flow-through conditions (Green *et al.* 1985). LC50s as low as <1.5 and 6.5 mg l^{-1} have been reported for *Baetis* spp and caddis fly (*Limnephilus stigma*) in old studies (Alekseev 1973 and UK Min. of Tech. 1968, cited in Mallet *et al.* 1992), although information on the methodology and reliability of these studies is unavailable.

B1.9 Fish

B1.9.1 Mode of action and symptoms

Phenol is thought to act as a non-specific metabolic inhibitor, interfering with enzyme function, ATP production and oxidative phosphorylation (Gupta 1987) to produce symptoms consistent with nervous paralysis. The symptoms of exposure to phenol have been reported by many workers. Typically, fish exhibit increased respiration and become more sensitive to external stimuli. Colour changes, increased mucous secretion, depressed activity and loss of equilibrium interrupted by occasional convulsions can all occur prior to death of exposed fish (Mallet *et al.* 1992).

Bradbury *et al.* (1989) investigated the narcotic action of phenol on adult rainbow trout (*Oncorhynchus mykiss*) by exposing four fish in a flow-through system to a measured concentration of 8.82 mg l^{-1}. After a period (7.8 hours) of increasing frequencies of seizures and coughing the fish became mostly inactive and unresponsive, with occasional tremors and coughs. There was a corresponding decrease in arterial oxygen, carbon dioxide and pH levels

with the increased muscular activity. The mean survival time of the fish was 13 hours. On transferring affected fish to clean water, the sub-lethal effects were found to be reversible, as noted with exposed invertebrates.

In general the changes in physiological parameters were consistent with increased muscular activity and oxygen demand associated with seizures (Bradbury *et al.* 1989). A continued depression of respiratory activity with increasing exposure is consistent with a general narcotic effect. The associated changes in blood chemistry parameters suggest a rapid shift towards anaerobic metabolism which is consistent with the increased muscular activity. The reversible nature of these symptoms also fulfils an important criterion for defining the observed responses as a narcosis effect (Bradbury *et al.* 1989).

B1.9.2 Lethal effects

The available data indicate that phenol is of moderate acute and chronic toxicity to fish. Non-salmonid fish appear to be less sensitive to the toxic effects of phenol than salmonids. The majority of acute LC50s for non-salmonids and salmonids range from 9.4-49.9 and 3.0-10.5 mg l^{-1}, respectively, although a 96 hour LC50 as low as 0.46 mg l^{-1} has been recorded for the guppy, *Poecilia reticulata* (Saarikoski and Viluksela 1982). However, this is a nominal concentration calculated from the authors data and so the reliability of the value is questionable. Chronic data indicate a similar pattern. Milleman *et al.* (1984) reported measured LC50s of 2.67 and 0.12 mg l^{-1} for embryo/larval stages of largemouth bass (*Micropterus salmoides*) and rainbow trout (*Oncorhynchus mykiss*) following exposure from egg fertilisation to 4 days post-hatch (total exposure periods of 7 and 27 days, respectively) in a flow-through system. However again the reliability of this study has been questioned since studies by the co-authors have indicated a much higher sensitivity than other researchers (Jones *et al.* 1992).

Holdway *et al.* (1991) investigated the effects of a 2 hour pulse exposure of phenol on 8-day old larvae of the American flagfish, *Jordanella floridae*. The larvae were exposed to nominal concentrations that were renewed every 15 minutes for a period of 2 hours, and were then transferred to clean water. Mortality was observed over the subsequent 94 hours. From these observations LC20 and LC50 values of 0.7 and 5.1 mg l^{-1} were calculated. These values are lower than most other acute data reported for non-salmonid fish, and although they are for a species which is not indigenous to the UK, they provide an interesting insight to toxic effects caused by pulsed exposures such as might arise from an episodic pollution incident.

As with invertebrates, it is apparent that temperature may affect the toxicity of phenol to fish. Brown *et al.* (1967) reported that the resistance of rainbow trout (*O. mykiss*) to low phenol concentrations increased with an increase in temperature. The authors calculated 48 hour LC50s of 5.0, 5.8 and 9.8 mg l^{-1} (measured concentrations) following exposure in a semi-static bioassay. The authors attributed the increased toxicity with decreased temperature to an impairment in detoxification mechanisms (conjugation). This effect gives cause for concern since fish populations are likely to be most susceptible to phenol toxicity at lower temperatures, when conditions are normally considered most favourable (Brown 1967). Since temperature can influence both uptake and detoxification of phenol, it is conceivable that toxicity may be enhanced as well as diminished, by increasing temperature, depending on which parameter is influenced most.

B1.9.3 Sub-lethal effects

The majority of available data relate to lethal effects of phenol on fish. However, some reliably reported toxicity studies have shown sub-lethal effects at concentrations ranging from 1.9-12.0 mg l^{-1}, although these levels are easily within the range of concentrations found to be lethal in other acute studies. The lowest reliable sub-lethal NOECs are measured concentrations of 0.75 and 1.83 mg l^{-1} based on growth for fathead minnow (*Pimephales promelas*) following exposure under flow-through conditions for 30 and 38 days, respectively (DeGraeve 1980 and Holcombe *et al.* 1982). Corresponding NOECs of 83.2 and 3.57 mg l^{-1} based on hatching were also reported in these studies. However a measured NOEC based on hatching as low as 0.009 mg l^{-1} has been reported in a flow-through test for rainbow trout (*O. mykiss*) (Black *et al.* 1983), although this was the highest concentration tested and so the true NOEC could have been considerably higher than this.

Kumar and Mukherjee (1988) investigated the effects of phenol on the reproductive physiology and biochemistry of common carp (*Cyprinus carpio*) by exposing sexually maturing females to a supposedly sub-lethal concentration of 12.0 mg l^{-1} (although this concentration is well within the lethal range reported in Section B1.2) in a semi-static system over a 30 day period. Gonadal maturation was found to be depressed, indicating a probable liver dysfunction, since this is the organ responsible for synthesis of egg-yolk protein and biotransformation of steroid hormones and xenobiotics. Ovarian cholesterol levels increased gradually up to day 14 after which levels remained at a steady state for the duration of the experiment. The observed accumulation of cholesterol, the precursor of steroid hormones, indicates interference with vitellogenesis (Kumar and Mukherjee 1988). These effects are important since adversely affected fecundity in the field could lead to recruitment failure in fish populations.

In an effort to assess the avoidance behaviour of rainbow trout (*O. mykiss*) to phenol, DeGraeve (1982) exposed three fingerlings to each measured concentration in a specially modified flow-through apparatus. Behaviour was monitored with an 8-mm movie camera mounted above the surface of the water. Following 48 hours exposure to concentrations of 6.8-8.1 mg l^{-1}, the fish showed 70-93% avoidance of the toxicant containing flow. Avoidance behaviour during exposure to 5.3-6.5 mg l^{-1} was less pronounced but was still significant, whereas the fish were apparently attracted to concentrations of 1.9-2.3 mg l^{-1}. During exposure to the phenol, especially at the higher concentrations, the fish showed obvious signs of sub-lethal toxicity (e.g. coughing, rapid respiration, erratic swimming, muscular spasms and loss of equilibrium). These responses were more apparent on initial exposure. Avoidance behaviour is important, since avoidance of contaminated areas in the field may force fish into unsuitable habitats.

B1.10 Amphibians

A 48 hour LC50 of >51.1 mg l^{-1} has been reported for tadpoles of the African clawed toad, *Xenopus laevis* (Holcombe *et al.* 1987), indicating moderate to low acute toxicity to amphibians. However, Birge *et al.* (1980) have reported much lower toxicity data for a range of amphibians, the lowest of which being an LC50 of 0.04 mg l^{-1} recorded for the leopard frog, *Rana pipiens*, at 4 days post-hatch, following exposure of eggs and larvae in a flow-through regime for a total of 9 days. However, there has been some concern over the reliability of these studies, for example the wide spread of exposure concentrations has been criticised (*Rana*

pipiens was exposed to concentrations of 0.0047, 0.0073, 0.074, 1.09 and 11.5 mg l^{-1}). In addition, confirmative studies using toluene have shown that Black *et al.*'s results indicating much higher sensitivity than those found by other researchers (Jones *et al.* 1992).

B2. BIOACCUMULATION

Phenol does not appear to be accumulated to particularly high levels in the majority of freshwater organisms. The majority of reliable BCFs range from 3.5-16.0, although values as high as 277-1375 have been reported for the water flea, *D. magna* (see Table B2). Uptake and attainment of steady state equilibrium appears to be rapid (generally within hours). Depuration also appears to be rapid on transferring exposed organisms to clean water, with some authors reporting evidence of biotransformation.

B2.1 Algae

Hardy *et al.* (1985) assessed the bioaccumulation potential of algae by exposing *Scenedesmus quadricauda* to measured concentrations of 0.36 and 2.0 mg l^{-1}, over a 24 hour period. Uptake over the first few hours of exposure was observed to be rapid but then slowed, with equilibrium being reached by the end of the 24 hour period. A BCF (dry-weight) of 16 was calculated, based on kinetic data. However, significant quantities of phenol were biotransformed by the algae at the highest exposure concentration (6.1% of body-burden per hour, 78% total), leading to a 'biotransformation-corrected' BCF (dry-weight) of only 3.5. Following transfer to clean water, rapid elimination was observed within a few hours which slowed over the subsequent 22 hours. Despite these losses, high concentrations of phenol remained in the algae even after 24 hours depuration. The authors attributed the initial rapid uptake and elimination rates to adsorption to surfaces of the algal cells rather than true uptake. However, both adsorbed and absorbed phenol may be available for transfer up aquatic food chains.

Freitag *et al.* (1985) reported a much higher BCF of 200 for the algal species, *Chlorella fusca* following exposure over 1 day. However, it is difficult to assess the reliability of this study as exposure concentration is not reported and information concerning method of analysis, uptake, elimination and biotransformation is not given.

B2.2 Molluscs

A BCF of 0.5 has been estimated for the snail species, *Physa fonitinalis* and *Radix perega*, following 6 days exposure to measured concentrations of 100-300 mg l^{-1} (Erben 1982, cited in Crookes *et al.* 1994). However, this value should be treated with caution as the test concentration decreased by 9.2-94.4% over the exposure duration. The authors attributed this to biodegradation of phenol as evidenced by an increase in bacterial activity. Furthermore, a significant percentage of the test organisms died during the study.

Table B1 Toxicity of phenol to freshwater organisms

Species	Life stage	Test type	Analysis	Temp (°C)	Hardness (mg $CaCO_3$ l^{-1})	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
ALGAE										
Selenastrum capricornutum (Green unicell)	Exp gro phase	S[1]	-	21±1	-	-	96 h	150.0	EC50 (Cell number)	1
Selenastrum capricornutum (Green unicell)	-	S[2]	nominal	-	140.0	7.6	4 h	287.0	EC50 (Assimilation of inorganic ^{14}C)	2
Selenastrum capricornutum (Green unicell)	-	-	nominal	-	-	-	14 d	175.0	EC50 (Growth)	3
Selenastrum capricornutum (Green unicell)	-	S[3] S[1]	nominal nominal	24 24	-	-	4 d 4 d	63.1 61.1	EC50 (Growth)	4
Chlorella vulgaris (Green unicell)	Exp gro phase	S[1]	-	21±1	-	-	96 h	370.0	EC50 (Cell number)	1
Scenedesmus quadricauda	-	S	nominal	27	72.3	7.0	7 d	8.0	Threshold of toxicity (growth)	38

Species	Life stage	Test type	Analysis	Temp (°C)	Hardness (mg $CaCO_3$ l^{-1})	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
MACROPHYTES										
Lemna minor (Duckweed)	-	-	nominal	27±2	-	7.5	4 d	>12.0	EC50 (Growth of fronds)	5
Lemna minor (Duckweed)	-	-	-	-	-	-	7 d 7 d 7 d	171.0 183.0 157.0	EC50 (Number of plants) EC50 (Number of fronds) EC50 (Dry weight)	50
Lemna gibba (Duckweed)	-	-	-	-	-	-	7 d 7 d 7 d 7 d	229.0 226.0 242.0 14.0	EC50 (Number of plants) EC50 (Number of fronds) EC50 (Dry weight) NOEC (plants and fronds)	50
MOLLUSCS										
Aplexa hypnorum (Snail)	Adult	F	measured	17.2	44.7	7.4	96 h	>51.1	LC50	17
Physa fontinalis (Snail)	-	F	measured	11±1	99.5±7.7	7.5-8.1	48 h	80.0-120.0	LC50	6
Physa gyrina (Snail)	7.5 mm	S	measured	19.5-20.5	140.0	7.8	48 h	260.0	LC50	2
Lymnaea acuminata (Snail)	-	SS	nominal	20±2	190-223	7.9±0.2	96 h	129.0	LC50	39

Species	Life stage	Test type	Analysis	Temp (°C)	Hardness (mg $CaCO_3$ l^{-1})	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
Indoplanorbis exustus (Snail)	-	S	nominal	'Room'	-	-	12 h 24 h 48 h 72 h 96 h	264.5 215.0 200.1 155.5 125.75	LC50 LC50 LC50 LC50 LC50	18
PLATY-HELMINTHES										
Polycelis tenuis (Flatworm)	-	F	measured	11±1	99.5±7.7	7.5-8.1	48 h	200.0	LC50	6
ANNELIDS										
Limnodrilus hoffmeisteri (Oligochaete worm)	-	F	measured	11±1	99.5±7.7	7.5-8.1	48 h 96 h	870.0 780.0	LC50 LC50	6
ROTIFERS										
Brachionus calyciflorus (Rotifer)	Neonates	S[4]	nominal	25	-	7.5	2 d 24 h	59.0 25.0 43.0 780.0	EC50 (population increase) NOEC (population increase) LOEC (population increase) LC50	7
Brachionus calyciflorus (Rotifer)	Neonates	S[5]	nominal	25	-	-	24 h	>150.0	LC50	8

Species	Life stage	Test type	Analysis	Temp (°C)	Hardness (mg $CaCO_3$ l^{-1})	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
ARTHROPODS (CRUSTACEANS)										
Daphnia magna (Water flea)	Adult	S	nominal	20±1	-	-	24 h	37.17	EC50 (immobilisation)	9
Daphnia magna (Water flea)	6-24 h	S	nominal	20	2.4[6]	-	24 h	21.0 9.0 >100.0	EC50 (ability to swim) EC0 (ability to swim) EC100 (ability to swim)	10
							48 h	10.0 3.5 16.0	EC50 (ability to swim) EC0 (ability to swim) EC100 (ability to swim)	10
Daphnia magna (Water flea)	Neonates	S	nominal	19.8-20.9	157±4	7.7-8.3	48 h	12.9	LC50	11
Daphnia magna (Water flea)	<24 h	S	measured	21.7±0.9	146±14.6	6.8-7.8	48 h	4.2	EC50[7]	12
Daphnia magna (Water flea)	Neonates	S	nominal	20 24	-	8.0-8.6 8.2-8.5	48 h 48 h	12.9 12.8	LC50 LC50	13
Daphnia magna (Water flea)	Adult	SS	nominal	25±2	160-180	8.2±.2	48 h 264 h 264 h	13.0 4.0 0.5	LC50 LC50 NOEC (Mortality)	15
Daphnia magna (Water flea)	Adult	SS	nominal	25±2	160-180	8.2±.2	264 h 264 h 264 h	6.0 7.0 7.0	EC50 (Progeny) EC50 (Number of broods) EC50 (Mean brood size)	15

Species	Life stage	Test type	Analysis	Temp (°C)	Hardness (mg $CaCO_3$ l^{-1})	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
Daphnia magna (Water flea)	24 h	S	measured	19.5-20.5	140.0	7.8	48 h	19.8	EC50[7]	2
Daphnia magna (Water flea)	Juvenile	S	n	20±1	130.0	6.5-8.5	96 h	4.0	LC50[8]	16
Daphnia magna (Water flea)	Adult	F	measured	17.2±0.2	40.8-47.6	6.8-7.8	48 h	12.6	EC50[8] (immobilisation)	17
Daphnia magna (Water flea)	Neonates	S	-	21±1	-	7.6	24 h	9.13	EC50 (immobilisation)	20
Daphnia magna (Water flea)	24 h	SS	nominal	19±1	199	8.2	16 d	0.16	NOEC (Growth)	42
Daphnia magna (Water flea)	-	SS	nominal	19±1	199	8.2±0.3	21 d	3.2	NOEC (Growth and repro.)	43
Ceriodaphnia dubia (Water flea)	Neonates	S	nominal	20 24	-	8.1-8.7 8.2-8.6	48 h 48 h	12.1 4.3	LC50 LC50	13
Ceriodaphnia dubia (Water flea)	Adult	SS	nominal	25±2	160-180	8.2±.2	48 h 192 h 240 h	20.0 9.0 0.84	LC50 LC50 NOEC (Mortality)	15
Ceriodaphnia dubia (Water flea)	Adult	SS	nominal	25±2	160-180	8.2±.2	240 h 240 h 240 h 240 h	7.0 10.0 8.0 6.5	EC50 (Progeny) EC50 (Number of broods) EC50 (Mean brood size) NOEC	15

Species	Life stage	Test type	Analysis	Temp (°C)	Hardness (mg $CaCO_3$ l^{-1})	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
Ceriodaphnia dubia (Water flea)	Neonates	SS	measured	25	57.07±4.14	8.18	48 h 4 d	3.1 5.3	LC50 EC50 (no of young per female)	14
Asellus aquaticus (Aquatic sowbug)	8-11mm	SS	measured	10.0 20.0 10.0 20.0	82-97	7.1-8.0	197 min 677 min 43.4 min 45.6 min	98.5 96.9 150.9 150.1	EC50 (immobilisation)[9] EC50 (immobilisation)[10] EC50 (immobilisation)[11] EC50 (immobilisation)[12]	19
Asellus aquaticus (Aquatic sowbug)	-	F	measured	11±1	99.5±7.7	7.1-8.0	48 h 96 h	200.0 180.0	LC50 LC50	6
Asellus intermedius (Pillbug)	0.012 g	S	nominal	20±1	130	7.4	96 h	25.0	LC50[8]	16
Gammarus pulex (Freshwater shrimp)	-	F	measured	11±1	99.5±7.7	7.1-8.0	48 h 96 h	85.0 69.0	LC50 LC50	6
Gammarus minus (Scud)	Adult	S	measured	21-24	-	-	48 h	37.4	LC50	2
Gammarus fasciatus (Sideswimmer)	0.007 g	S	nominal	20±1	130.0	7.4	96 h	21.0[8]	LC50	16
ARTHROPODS (INSECTS)										
Baetis rhodani (Mayfly)	Larvae	F	measured	11±1	99.5±7.7	7.1-8.0	48 h 96 h	18.5 15.5	LC50 LC50	6

Species	Life stage	Test type	Analysis	Temp (°C)	Hardness (mg $CaCO_3$ l^{-1})	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
Hydropsyche angustipennis (Mayfly)	Larvae	F	measured	11±1	99.5±7.7	7.1-8.0	48 h 96 h	720.0 260.0	LC50 LC50	6
Baetis spp (Mayfly)	Larvae	SS	-	-	-	-	48 h	<1.5	LC50	49
Chironomus riparius (Midge)	Larvae	F	measured	11±1	99.5±7.7	7.1-8.0	48 h 96 h	500.0 240.0	LC50 LC50	6
Chironomus tentans (Midge)	Larvae	S	measured	23-26	-	-	48 h	105.0	LC50	2
Tanytarsus dissimilis (Midge)	Larvae	F	measured	17.2±0.2	40.8-47.6	6.8-7.8	48 h	>51.1	LC50[8]	17
Limnephilus stigma (Caddisfly)	Larvae	SS	-	-	-	-	48 h	6.5	LC50	48
FISH (NON-SALMONID)										
Lebistes reticulatus (Guppy)	170 mg	S	-	-	-	-	48 h 96 h	34.0 31.0	LC50	33
Poecilia reticulata (Guppy)	40-60 mg	SS	nominal	26±1	-	-	96 h	0.46	LC50[17]	27
Poecilia reticulata (Guppy)	2 g	S	measured	22-25	80	6.5	96 h	40.0	LC50	34

Species	Life stage	Test type	Analysis	Temp (°C)	Hardness (mg $CaCO_3$ l^{-1})	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
Poecilia reticulata (Guppy)	0.1-0.2 g	S	nominal	25	20	7.5	48 h 96 h	49.9 39.2	LC50 LC50	35
Rutilus rutilus (Roach)	63±3	F	nominal	10.3±0.4	260	7.8	6 h	9.4	LC50[18]	30
Perca fluviatilis (Perch)	-	F	-	18	-	-	24 h	15.0	LC50	50
Cyprinus carpio (Carp)	50-60 g[14]	SS	nominal	28.3±1.1	271.66±22.48	7.2±0.1	30 d	12.0	Significant inhibition on development of ovary. Significant increase in ovarian and hepatic cholesterol levels.	21
Micropterus salmoides (Largemouth bass)	Eggs and larvae	F	measured	20.2-23.2	86.8-116.3	-	7 d[13]	2.67	LC50	2
Pimephales promelas (Fathead minnow)	30-35 d	F S	measured	23-27	43-49	≅ 7.5	96 h 48 h	28.5 8.3	LC50	37
Pimephales promelas (Fathead minnow)	0.9-13.1	F[19]	measured	16.6-17.4	40.8-47.6	7.03-7.7	96 h	25.3	LC50	31
Pimephales promelas (Fathead minnow)	1-2 mo	S	measured	20±0.5	-	-	96 h	25.6	LC50	2
Pimephales promelas (Fathead minnow)	0.2-0.5 g	S	nominal	20±1	130.0	7.4	96 h	32.0	LC50[8]	16

Species	Life stage	Test type	Analysis	Temp (°C)	Hardness (mg $CaCO_3$ l^{-1})	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
Pimephales promelas (Fathead minnow)	Fry Juvenile Sub-adult	S	nominal	21-23	96-125	7.2-8.5	96 h	23.0 43.7 31.2	LC50 LC50 LC50	26
Pimephales promelas (Fathead minnow)	eggs to 30 day post-hatch	F	measured	25±1	725.3	8.0	30 d	83.2 0.75	NOEC (Hatch) NOEC (Growth)	44
Pimephales promelas (Fathead minnow)	eggs to 38 day post-hatch	F	measured	25±2	46	7.7	38 d	3.57 1.83	NOEC (Hatch) NOEC (Growth)	45
Brachydanio rerio (Zebrafish)	-	F	measured	25±0.5	350-375	8.0-8.3	48 h 96 h	31.0 29.0	LC50 LC50	40
Brachydanio rerio (Zebrafish)	-	SS	measured	24±1	64	-	96 h	25.0	LC50	41
Carassius auratus (Goldfish)	1.0-2.0 g	S	nominal	25	20	7.5	48 h 96 h	49.1 44.5	LC50 LC50	36
Catostomus commersoni (White sucker)	4.4	F	measured	17.2±0.2	40.8-47.6	6.8-7.8	96 h	10.6	LC50[8]	32
Lepomis macrochirus (Bluegill sunfish)	2.5	F	measured	17.2±0.2	40.8-47.6	6.8-7.8	96 h	17.4	LC50[8]	17
Notopterus notopterus (Freshwater teleost)	20.6±0.3g	SS	nominal	-	-	-	96 h	1.25	35.6-66.1% inhibition of brain hydrolytic enzyme activity	22

Species	Life stage	Test type	Analysis	Temp (°C)	Hardness (mg $CaCO_3$ l^{-1})	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
Notopterus notopterus (Freshwater teleost)	20.6±0.3g	SS	nominal	-	-	-	96 h	0.83	26.5-56.4% inhibition of brain hydrolytic enzyme activity	22
								0.63	25.5-54.6% inhibition of brain hydrolytic enzyme activity.	22
Jordanella floridae (American flagfish)	8 d	SS	nominal	25.1	333	7.97	2 h[15] 2 h[15]	0.7 5.1	LC20[15] LC50[15]	24
Jordanella floridae (American flagfish)	0.1-0.3 g	F	measured	25	350-375	8.0-8.3	48 h 96 h	36.3 36.3	LC50 LC50	33
Campostoma anomalum (Central stoneroller)	47.5 mm	SS	measured	23.0	45.0	7.8	48 h	17.9 12.0	LC50 No mortality. However, thermal tolerance reduced[16]	25
Danio devario	5 cm	F	nominal	-	96-102	7.5-7.9	96 h	28.7	LC50	29
Esomus danricus	5 cm	F	nominal	-	96-102	7.5-7.9	96 h	30.5	LC50	29
Chela atpar	5 cm	F	nominal	-	96-102	7.5-7.9	96 h	12.7	LC50	29
Ambassis gymnocephalus	5 cm	F	nominal	-	96-102	7.5-7.9	96 h	6.9	LC50	29
Puntius sophore	5 cm	F	nominal	-	96-102	7.5-7.9	96 h	14.1	LC50	29
Oryzias melanostigma	5 cm	F	nominal	-	96-102	7.5-7.9	96 h	9.6	LC50	29
Aplocheilus panchax	5 cm	F	nominal	-	96-102	7.5-7.9	96 h	9.5	LC50	29
Puntius ticto	5 cm	F	nominal	-	96-102	7.5-7.9	96 h	15.5	LC50	29

Species	Life stage	Test type	Analysis	Temp (°C)	Hardness (mg $CaCO_3$ l^{-1})	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
Gambusia affinis (Mosquito fish)	5 cm	F	nominal	-	96-102	7.5-7.9	96 h	15.3	LC50	29
Gambusia affinis (Mosquito fish)	1.9 g	S	measured	-	-	-	96 h	26.0	LC50	34
FISH (SALMONID)										
Oncorhynchus mykiss (Rainbow trout)	Eggs and larvae	F	measured	13.3-14.2	86.8-116.3	-	27 d^{13}	0.12	LC50	2
Oncorhynchus mykiss (Rainbow trout)	7.5-9.3cm	F	measured	12.0	707-715	8.0-8.1	48 h	6.8-8.1 5.3-6.5 1.9-2.3	70-93% avoidance of toxicant Significant avoidance of toxicant Apparent attraction to phenol	23
Oncorhynchus mykiss (Rainbow trout)	5.2-11.3cm	SS	measured	6.3 11.8 18.1	320	7.8±0.3	48 h	5.4 8.0 9.8	LC50 LC50 LC50	28
Oncorhynchus mykiss (Rainbow trout)	0.9-13.1	F^{19}	measured	16.6-17.4	40.8-47.6	7.03-7.7	96 h	10.5	LC50	31
Oncorhynchus mykiss (Rainbow trout)	13-15 cm	F	measured	12.6	-	7.8	48 h	7.5	LC50	32
Oncorhynchus mykiss (Rainbow trout)	0.2-0.6 g	F	measured	15.0	350-375	8.0-8.3	96 h	11.6	LC50	33

Species	Life stage	Test type	Analysis	Temp (°C)	Hardness (mg $CaCO_3$ l^{-1})	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
Oncorhynchus mykiss (Rainbow trout)	-	F	-	3.0-4.0 12.0-13.0	- 320 10	-	48 h 48 h 48 h 48 h	3.0 5.0 6.8 5.2	LC50 LC50 LC50 LC50	49
Oncorhynchus mykiss (Rainbow trout)	Eggs to 4 days post-hatch	F	measured	13.3-14.2	100	7.4-8.1	27 d	0.009[20]	NOEC (Hatching)	46
AMPHIBIANS										
Xenopus laevis (African clawed toad)	Tadpole	F	measured	17.2±0.2	40.8-47.6	6.8-7.8	48 h	>51.1	LC50[8]	17
Xenopus laevis (African clawed toad)	Eggs to 4 days post-hatch	F	-	21.7	99.6	7.7	6 d 6 d	12.7 7.69	LC50 at time of hatch (2 days) LC50 at 4 days post-hatch	47
Rana palustris (Pickerel frog)	Eggs to 4 days post-hatch	F	-	20.5	106.4	7.7	8 d 8 d	11.2 9.87	LC50 at time of hatch (2 days) LC50 at 4 days post-hatch	47
Rana pipiens (Leopard frog)	Eggs to 4 days post-hatch	F	-	19.0	113.8	7.6	9 d 9 d	0.05 0.04	LC50 at time of hatch (2 days) LC50 at 4 days post-hatch	47
Rana catesbeiana (Bull frog)	Eggs to 4 days post-hatch	F	-	20.0	113.1	7.5	8 d 8 d	0.6 0.23	LC50 at time of hatch (2 days) LC50 at 4 days post-hatch	47

Species	Life stage	Test type	Analysis	Temp (°C)	Hardness (mg $CaCO_3$ l^{-1})	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
Rana temporaria (European common frog)	Eggs to 4 days post-hatch	F	-	21.7	99.6	7.7	9 d	0.46 0.38	LC50 at time of hatch (2 days) LC50 at 4 days post-hatch	47
Ambystoma gracile (North-western salamander)	Eggs to 4 days post-hatch	F	-	21.7	99.6	7.7	9 d	0.46 0.38	LC50 at time of hatch (2 days) LC50 at 4 days post-hatch	47

Notes

LC50	Median lethal concentration
EC50	Median effect concentration
ft	Flow-through
s	Static
ss	Semi-static
mi	Minutes
h	Hours
d	Days
w	Weeks
mo	Months
m	measured
n	nominal
-	Data not seen or not reported

1	Test conducted in shake flask culture.
2	Test conducted in scintillation vials.
3	Test conducted using a microplate system.
4	Test conducted in test tubes.
5	Test conducted in multi-well plate.
6	As mmol l^{-1}
7	Immobilisation referred to by authors as 'mortality'
8	Tested in a multi-species bioassay along with 6 other organisms representing Platyhelminthes, Molluscs, Crustaceans and fish.
9	Corresponding 50% recovery time on placing in clean water is 17.0 min
10	Corresponding 50% recovery time on placing in clean water is 5.7 min
11	Corresponding 50% recovery time on placing in clean water is 25.8 min
12	Corresponding 50% recovery time on placing in clean water is 9.2 min
13	Test ended 4 d after mean hatch.
14	Sexually maturing females

15 Exposed for 2 hours. Mortality observed over subsequent 94 hours in toxicant-free water
16 Thermal tolerance measured by CTMax. This is the temperature at which fish lose equilibrium. CTMax for control fish acclimated at 23 °C was 35.8 °C
17 Calculated from authors data which are reported as mMol l^{-1}
18 The LC50 is considered to be the asymptotic LC50 (i.e. Continued exposure duration will not decrease the LC50 any further)
19 Rainbow trout and fathead minnow tested simultaneously in screened compartments
20 Highest concentration tested

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B2.3 Crustaceans

Considerably higher BCFs have been reported by Dauble *et al.* (1986) for the water flea, *D. magna.* In this study the authors exposed adults to measured concentrations of 634-696 $\mu g\ l^{-1}$ and measured accumulation as measured by ^{14}C uptake over 24 hours. A BCF as high as 1375 was calculated based on direct measurements of phenol concentrations (dry-weight), although when the BCF was based on the kinetics of uptake and elimination a value of 277 ± 66 was estimated. following transfer to clean water, a depuration half-life of 8 hours was calculated. In addition, it was apparent that the Daphnids were able to metabolise the phenol, although since the metabolites were not characterised, it was not possible to determine relative amounts against the parent compound.

B2.4 Fish

Following 24 hours exposure to a measured concentration of 24 $nmol\ l^{-1}$ in either river or tap water, respective BCFs of 7 and 16 were calculated for the zebra fish, *Brachydanio rerio* (Ensenbach and Nagel 1991). Time taken to reach steady-state equilibrium was 4 hours in tap water and 8 hours in river water. The higher BCF recorded in tap water represents a doubling of uptake rate and may be partially explained by increased bioavailability of phenol in tap water compared with the test compound in river water. Following transfer to clean water, complete elimination was observed within 48 hours.

A similar pattern of rapid uptake and elimination has been reported for rainbow trout (*O. mykiss)* (Swift 1978, cited in Crookes *et al.* 1994). BCFs of 1 and 2 were reported for fish exposed to phenol concentrations of <9.0 and 10.0 $mg\ l^{-1}$ for 24 hours under flow-through conditions. Uptake equilibrium was observed within 2-3 hours and elimination occurred within a similar time period on placing the exposed organisms in clean water.

Freitag *et al.* (1985) reported a BCF of 20 for the golden ide (*Leuciscus idus melanotus*) following exposure over 3 days. However, it is difficult to assess the reliability of this study as exposure concentration is not reported and information concerning method of analysis, uptake, elimination and biotransformation is not given.

Table B 2 Bioaccumulation of phenol in freshwater organisms

Species	Life stage	Test type	Analysis (m/n)	Temp (°C)	Exposure time	Concentration		BCF[2]	Ref
						Water (mg l^{-1})	Tissue[1] (mg kg^{-1})		
ALGAE									
Scenedesmus quadricauda (Green alga)	log phase (6 d)	S	m	20-22	24 h	0.36-2.0	108-2200 nmol l^{-1}	16 (dw) 3.5[3] (dw)	1
Chlorella fusca (Green alga)	-	-	-	-	1 d			200.0	2
MOLLUSCS									
Physa fontinalis and *Radix perega* (Snail)	-	-	m	18-21	6 d	100-300[4]	-	0.5	3
ARTHROPODS (CRUSTACEANS)									
Daphnia magna (Water flea)	Adult	s	m	-	24 h	0.634-0.696	-	1375.0[5] (dw) 277+/-66[6]	4
FISH (NON - SALMONID)									
Brachydanio rerio (Zebra fish)	360 mg	s	m	26±1	24 h	24 nmol l^{-1}	-	7.0[7] 16.0[8]	5
Leusiscus idus melanotus (Golden ide)	-	-	-	-	3 d	-	-	20.0	2

Species	Life stage	Test type	Analysis (m/n)	Temp (°C)	Exposure time	Concentration		BCF[2]	Ref
						Water (mg l^{-1})	Tissue[1] (mg kg^{-1})		
FISH (SALMONID)									
Oncorhynchus mykiss (Rainbow trout)	-	ft	-	-	24 h	<9.0 10.0	-	1.0 2.0	6

Notes:

LC50	Median lethal concentration
EC50	Median effect concentration
ft	Flow-through
s	Static
ss	Semi-static
mi	Minutes
h	Hours
d	Days
w	Weeks
mo	Months
m	measured
n	nominal
-	Data not seen or not reported
1	Wet weight unless indicated otherwise
2	Bioconcentration factor - concentration in tissue/concentration in water
3	BCF taking into account biotransformation of phenol by the alga
4	Concentrations decreased by 9.2-94.4% over the exposure period. Corresponding increase in bacterial numbers indicates biodegradation as the cause
5	BCF based on ^{14}C activity and dry-weight conversion
6	BCF based on kinetics of uptake and elimination
7	BCF in river water
8	BCF in tap water

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APPENDIX C SALTWATER TOXICITY AND BIOACCUMULATION

Toxicity data are shown in Table C1. The most pertinent studies for saltwater toxicity and bioaccumulation are discussed below.

C1. SALTWATER TOXICITY

There is a considerable amount of data in the literature concerning the toxicity of phenol to saltwater organisms. In addition, several reviews exist that summarise available data (e.g. WHO 1994 and Crookes *et al.* 1994). For this reason, only the lowest and more reliable toxicity data have been included in Table C1 and discussed in the text. Attention has also been paid to reliable studies not included in the aforementioned reviews. However, where data are scarce for a particular group of organisms, all available information has been included, regardless of reliability.

C1.1 Algae

The only available short-term results regarding the effects of phenol on saltwater algae relate to a study in which a 4 hour EC50 of 185.0 mg l^{-1} based on assimilation of inorganic ^{14}C was reported for the diatom, *Nitzschia palea* (Milleman *et al.* 1984), indicating low acute toxicity. The majority of available data also suggest that phenol is of low chronic toxicity to saltwater algae. Cowgill *et al.* (1989) reported a 5 day EC50 (growth - cell count) of 49.6 mg l^{-1} for the diatom, *Skeletonema costatum*, while Thursby *et al.* (1985) reported LOECs of 21.6-60.0 mg l^{-1} for a range of growth and reproductive parameters.

C1.2 Echinoderms

A study was conducted by Falk-Petersen *et al.* (1985) to assess the effects of phenol on fertilised sea urchin (*Strongylocentrotus droebachiensis*) eggs. The authors were unable to calculate EC50 values (mortality and any sub-lethal effects) based on their data, since the highest measured concentration tested, 30.0 mg l^{-1} did not cause 50% effects.

C1.3 Molluscs

Available data for molluscs are conflicting. EC50s (normal development) of 58.3 and 52.6 mg l^{-1} have been reported for oysters and clams (*Crassostrea virginica* and *Mercenaria mercenaria*, respectively, Davis and Hidu 1969, cited in Crookes *et al.* 1994), suggesting moderate acute toxicity to these species. This is supported by a 12 day LC50 of 55.0 mg l^{-1} reported by the same authors for the clam. However, Mayer (1987) and Portman and Wilson (1971, cited in Crookes *et al.* 1994) have reported a 96 hour EC50 of >2000 and a 48 hour LC50 of >500 mg l^{-1}, for the oyster (*C. virginica*) and the cockle (*Cardium edule*) respectively.

C1.4 Annelids

The only available data relate to 48 hour LC50s ranging from 100-330 mg l^{-1} reported for the polychaete worm, *Orphryotrocha diadema* (Parker 1984, cited in WHO 1994), indicating low acute toxicity to this species.

C1.5 Crustaceans

Phenol appears to be of moderate to low acute toxicity to crustaceans, with the majority of acute LC50s ranging from 7.4-56.0 mg l^{-1}. A 24 hour LC50 of 171.74 mg l^{-1} (measured concentration) has been reported for the copepod, *Tisbe battagliai* (Smith *et al.* 1994), indicating low acute toxicity to this species. At sub-lethal concentrations (0.05-5.0 mg l^{-1}) a significant avoidance response was observed for the mysid shrimp, *Neomysis mirabilis*, with 31.3%-95.8% of animals preferring the pollutant free zone of the test apparatus after an exposure period of 7-10 minutes (Luk'yanenko *et al.* 1994).

C1.6 Fish

C1.6.1 Lethal effects

Phenol appears to be of moderate acute toxicity to saltwater fish, with 96 hour LC50s ranging from 10.0-20 mg l^{-1}. The most reliable values are 96 hour LC50s of 14.23 and 19.99 mg l^{-1} (measured concentrations) reported for sole (*Solea solea*) and flounder (*Platichthys flesus*) in semi-static studies that followed OECD standard guidelines (Smith *et al.* 1994).

C1.6.2 Sub-lethal effects

To assess the sub-lethal effects of phenol on saltwater fish, Krajnovic-Ozretic and Ozretic (1988) exposed grey mullet (*Mugil auratus*) to measured concentrations of 0.5-25.0 mg l^{-1} under flow-through conditions. Despite the continuous dosing, the authors noted that the measured concentrations were dropping to around 50% of the initial values. This they attributed to proliferation of phenol degrading bacteria in the system. As a consequence, constant adjustments to the dosing regime were necessary to account for the losses. As with freshwater fish (see Section B1.9.1), exposed animals displayed a concentration dependent pattern of characteristic neurotoxic symptoms. Exposure to a concentration of 5.0 mg l^{-1} over an 8 day period initially caused rapid, excited swimming, but within 1 day the fish recovered. The first signs of lasting intoxication (excitation followed by depressed activity) occurred after 8 days exposure to 7.5 mg l^{-1}. These effects were accompanied by haemorrhaging of opercula and swelling of gall bladder. Exposure to 10.0 mg l^{-1} for 5 hours led to loss of equilibrium, inflamed gills, haemorrhaging in circulatory system and 25% mortality. At a concentration of 25.0 mg l^{-1} for just 1 hour convulsions and suffocation were observed along with 50% mortality.

Certain changes in blood chemistry accompanied the above physiological/behavioural effects. In the fish exposed to concentrations above 7.5 mg l^{-1}, haemoglobin and hematocrit levels were reduced. It was assumed that this was due to loss of erythroctes and haemorrhaging in the various organs. Glucose levels were unaffected, but there were significant decreases in lactate, protein, lipids, triglycerides and cholesterol levels. Concurrent with the decreased lipid and

protein levels, enzyme activity in the plasma was increased. The authors attributed the increased enzyme activity either to leakage of enzymes from damaged tissue or as a result of the changed metabolic activity as a consequence of the stress caused by phenol intoxication (Krajnovic-Ozretic and Ozretic 1988).

Other reported sub-lethal effects include inhibition of body pigmentation in 6-day post-hatch larvae of cod (*Gadus mohua*) following 96 hours exposure to measured concentrations of 10.0-30.0 mg l^{-1} (Falk-Petersen 1985), and avoidance of juvenile fish exposed to concentrations of 1.0-20.0 mg l^{-1} for 7-10 minutes (Luk'yanenko *et al.* 1988). The lack of body pigmentation may be due to inhibition of melanin formation as shown by exposure of larval sturgeons to a concentration of 40.0 mg l^{-1} (Shmalgauzen 1973, cited in Falk-Petersen 1985).

C2. BIOACCUMULATION

The only reliable data relate to a study conducted on the mud crab, *Panopeus herbstii* (Key and Scott 1986). The authors measured the uptake of a radiolabelled (^{14}C) phenol concentration of 2.0 mg l^{-1} over a 96 hour exposure period as well as depuration rates following transfer to clean water. The highest rate of uptake was observed in the gills followed by the hepatopancreas and muscle. A maximum total tissue concentration of 17.5 mg kg^{-1} was recorded, which is equivalent to a dry-weight BCF of 8.75. Following 96 hours depuration, phenol was rapidly but incompletely lost. The maximum total tissue concentration recorded after the depuration period was 9.09 mg kg^{-1}. This concentration represents 48% depuration (i.e. half-life is somewhat greater than 96 hours).

Table C1 Toxicity of phenol to saltwater organisms

Species	Life stage	Test type	Analysis	Temp (°C)	Salinity (‰)	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
ALGAE										
Nitzschia palea (Diatom)	-	S[1]	nominal	-	-	7.6	4 h	185.0	EC50 (Assimilation of inorganic ^{14}C)	1
Skeletonema costatum (Diatom)	-	-	nominal	19.5-20.6	-	7.7-9.0	5 d	49.6	EC50 (Total cell count)	2
								49.8	EC50 (Total cell volume)	
Skeletonema costatum	-	-	-	20.0	25	-	-	>16.0	EC50 (Cell division)	9
Thalassiosira pseudonana								>16.0	EC50 (Cell division)	9
Glenodinium halli								>16.0	EC50 (Cell division)	9
Isochrysis galbana								>16.0	EC50 (Cell division)	9
Champia parvula (Red alga)	Mature female	S[2]	nominal	22-24	30	-	14 d	21.6	LOEC (Growth - dry weight)	3
	Mature female							28.8	LOEC (Sig. reduced number of cystocarps - evidence of reproduction)	3
	Tetrasporo-phyte							10.4	LOEC (growth - dry weight)	3
	Tetrasporo-phyte							60.0	LOEC (Sig. reduced number of tetrasporangia - evidence of meiosis)	3
Ceramium strictum (Red alga)	Mature	-	-	23-24	20	8	7 d	≅40.0	EC50 (production of cystocarps)	10
ECHINODERMS										
Strongylocentrotus droebachiensis (sea-urchin)	Fertilised eggs	-	measured	5	filtered seawater	-	96 h	>30.0[4]	EC50 (All deviations from control[5])	7

Species	Life stage	Test type	Analysis	Temp (°C)	Salinity (‰)	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
MOLLUSCS										
Crassostrea virginica (Eastern oyster)	Eggs	SS	nominal	24	seawater	-	48 h	58.3	EC50 (Normal development)	11
Crassostrea virginica (Eastern oyster)	Juvenile	F	-	20	30	-	96 h	>2000.0	EC50	12
Mercenaria mercenaria (Clam)	Eggs 2d	SS	nominal	24	seawater	-	48 h	52.6	EC50 (Normal development)	11
							12 d	55.0	LC50	
Cardium edule (Cockle)	Adult	S	-	15	-	-	48 h	>500.0	LC50	13
ANNELIDS										
Orphryotrocha diadema (Polychaete worm)	-	S	nominal	21	seawater	-	48 h	100-330	LC50	14
ARTHROPODS - CRUSTACEANS										
Tisbe battagliai (Copepod)	Copepodid	S	measured	20	30	8. ±0.1	24 h	171.74	LC50	4
Palaemonetes pugio (Grass shrimp)	-	-	nominal	21±1	15	8.1±0.1	24 h	53.0	LC50	5
							48 h	11.0	LC50	5
							96 h	7.4	LC50	5
Penaeus aztecus (Brown shrimp)	Juvenile	F	-	20	30	-	96 h	>2000.0	EC50	12
Crangon crangon (European brown shrimp)	Adult	S	-	15	-	-	48 h	23.5	LC50	13
Pandalus montagui (Pink shrimp)	Adult	S	-	15	-	-	48 h	17.5	LC50	13
Artemia salina (Brine shrimp)	-	S	nominal	24.5	seawater	-	24 h	LC50 157.0		16
							48 h	LC50 52.6		

Species	Life stage	Test type	Analysis	Temp (°C)	Salinity (‰)	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
Neomysis mirabilis (Mysid shrimp)	47-52 mm	S	nominal	-	-	-	7-10 mins	0.05 1.0 5.0 10.0	31.3% avoidance[6] 53.1% avoidance[6] 95.8% avoidance[6] 50.0 % avoidance[6]	8
Panopeus herbstii (Mud crab)	Adult	SS	nominal	25	25	-	96 h	52.8 1.0-32.0	LC50 No sig. effects on oxygen consumption	6 6
Carcinus maenas (Shore crab)	Adult	S	-	15	-	-	48 h	56.0	LC50	13
FISH										
Solea solea (Sole)	45±2.5 g	SS	measured	6	22	8.±0.1	96 h	14.23	LC50	4
Platichthys flesus (Flounder)	56±2.5 g	SS	measured	6	5	8.±0.1	96 h	19.99	LC50	4
Phoxinus phoxinus	-	F	nominal	5.0±0.7	0.6%	7.7±0.1	96 h	10.0	LC50	15
Mugil auratus (Grey mullet)	125 g	F[3]	measured[3]	12±0.5	37.5±0.3	-	8 d	0.5	Normal behaviour. No changes in gross pathology or blood chemistry	6
							8 d	5.0	Excited swimming. Temporary increase in blood glucose and enzymes	6

Species	Life stage	Test type	Analysis	Temp (°C)	Salinity (°/oo)	pH	Exposure time	Concn (mg l^{-1})	Effect	Ref
							8 d	7.5	Excited, sensitive to light, later depressed activity. Severe changes in blood chemistry. 10% mortality	6
							5 h	10.0	Loss of equilibrium. Haemorrhaging. Severe changes in blood chemistry. 25% mortality	6
Mugil auratus (Grey mullet)	125 g	F[3]	measured[3]	12±0.5	37.5±0.3	-	1 h	25.0	Convulsions and suffocation. Severe changes in blood chemistry. 50% mortality	6
Gadus morhua (Cod)	Fertilised eggs	-	measured	5	filtered seawater	-	96 h	>30.0[4]	EC50 (All deviations from control[5])	7
	6 days post-hatch							10.0-30.0	inhibition of body pigmentation	7
Hypomesus olidus (Pond smelt)	Juvenile	S	nominal	-	-	-	7-10 mins	0.1	No avoidance[6]	8
								1.0	51.7 % avoidance[6]	
								10.0	57.1 % avoidance[6]	
								20.0	98.2 % avoidance[6]	
Opistocentrum ocellatus (saltwater fish)	Juvenile	S	nominal	-	-	-	7-10 mins	0.1	No avoidance[6]	8
								1.0	37.5 % avoidance[6]	
								10.0	79.7 % avoidance[6]	
								20.0	96.9 % avoidance[6]	

Notes:

LC50	Median lethal concentration
EC50	Median effect concentration
ft	Flow-through
s	Static
ss	Semi-static
mi	Minutes
h	Hours
d	Days
w	Weeks
mo	Months
m	measured
n	nominal
-	Data not seen or not reported

1	Test conducted in scintillation vials.
2	Test solution renewed on days 7 and 11
3	Despite constant dosing the measured concentration dropped to 50% of the initial value. This was apparently due to proliferation of phenolic bacteria. The dosing system was therefore constantly adjusted to compensate for this
4	Highest concentration tested
5	Effects defined as all deviations from control, including mortality, pathology and inhibition of cleavage and differentiation
6	As measured by a significant difference between percentage of test animals in pollutant free zone of test vessel and control

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APPENDIX D MAMMALIAN TOXICOLOGY

Phenol is of moderate acute oral toxicity and has historically been used as a general disinfectant and topical anaesthetic/antipruritic. Oral exposure is mainly from mouthwashes and phenolic compounds in food. Toxicity following skin contact or inhalation is normally associated with occupational exposure (Deichmann and Keplinger 1981).

Phenol confers an unpleasant medicinal taste to water and a characteristic odour. Threshold values reported in the literature vary considerably (0.016 -16.7 mg l^{-1}). This range probably reflects the use of different water types in the tests including chlorinated waters in which chlorophenols will form. Chlorophenols have much lower taste and odour thresholds and often distort values for pure phenol. An odour threshold concentration for pure phenol derived by WRc, is approximately 10 µg l^{-1} (>99% purity) in distilled water at 40 °C. The taste threshold concentration varied from >2 µg l^{-1} to 13 µg l^{-1} in a repeat experiment.

Phenol is readily halogenated during the chlorination of water to form a range of mono-, di- and tri- chlorophenols. These compounds do not appear to have substantially different toxicities but do impart an unpleasant medicinal taste to the water at concentrations an order of magnitude below that for phenol (US EPA 1994). Microbiological action in the gut is thought to breakdown some of these chlorophenol compounds to phenol itself but there are no accurate data for exposure via this route.

Phenol is readily absorbed through the skin, lungs and gastrointestinal (G.I). tract and is rapidly distributed throughout the body. Highest levels have been reported in liver, heart, kidneys, lungs, blood and muscle (WHO 1994). Metabolism of phenol principally occurs in the liver, lung and G.I. mucosa. Hydroxylation by the cytochrome p450 mixed function oxidase system to catechol and hydroquinone may occur. Conjugation of phenol or these metabolites with sulphate or glucuronic acid is the principle mechanism of detoxification. In humans 90% of an oral dose has been reported to be excreted in urine within 24 hrs as the sulphate conjugate of phenol. A glucuronide conjugate of phenol and both sulphate and glucuronide conjugates of the oxidative metabolite hydroquinone are the three other principal metabolites of phenol produced in humans (Capel *et al.* 1972). Other species, notably cat and pig show different toxicokinetics. Metabolites including catechol and 1,4-benzoquinone have been identified following *in vitro* incubation with rat and mouse microsomes respectively (Sawahata and Neal, 1983). The rat may therefore not be a good model for phenol metabolism in humans.

Phenol is of moderate acute toxicity for mammals. Oral LD_{50} values in the range 300-600 mg kg^{-1} bw have been reported for rats and mice. Effects of acute phenol toxicity include swelling, necrosis and haemorrhage of the mucous membranes of the throat and oesophagus. Myocardial necrosis and liver changes are systemic effects but death is due to central nervous system (CNS) depression (Diechmann and Keplinger 1981). Gastrointestinal irritation may occur following acute exposure to moderate doses. Jarvis *et al.* (1985) reported a significantly higher incidence of gastrointestinal illness and other symptoms following accidental contamination of source water with phenol. Concentrations in the drinking water were not measured but were probably 10 µg l^{-1} or higher. The survey was, however, conducted by post and required voluntary self reporting which will have allowed considerable variation in the accuracy of reporting.

There are no adequate long term exposure studies or reproductive toxicity studies available for phenol. Data from Heller and Pursell (1938) indicate that the fecundity and development of offspring was unaffected in rats by doses of up to 5000 mg l^{-1} in drinking water (~500 mg kg^{-1} bw) administered for 3 generations.

A National Toxicology Program (NTP) study (1983) investigated the teratogenic potential of phenol in rats and mice. Phenol, dissolved in distilled water was administered by gavage to female rats on days 6 - 15 of pregnancy. Groups of 20 to 22 animals were given doses of 0, 30, 60 and 120 mg kg^{-1} day^{-1} and the animals were sacrificed on day 20. Detailed examinations did not find any dose related signs of maternal toxicity nor were any clinical signs of phenol toxicity noted in the dams. A highly significant reduction in foetal body weights was found in the highest dose group. No relationship between dose and implantation could be established since implantation occurred before dosing began. The highest foetal NOAEL in this study was recorded as 60 mg kg^{-1} day^{-1}.

The same methodology was used with mice given doses of 0, 70, 140 and 280 mg kg^{-1} day^{-1}. At the highest dose 4/36 mice died and significant clinical signs of toxicity including tremors and reduced body weight gain, were seen in survivors. As in the rat study, a significant dose related reduced foetal body weight was noted at the highest dose level, also an increased incidence of cleft palate. The highest (no observed adverse effects level) NOAEL in this study was 140 mg kg^{-1} day^{-1}.

Animal studies have demonstrated covalent binding of phenol and its metabolites to plasma proteins whilst reactive metabolites, formed by peroxidases, have been reported to bind readily to proteins and DNA (WHO 1994). However, there are inconclusive data from *in vitro* tests for mutagenicity. Phenol was shown to be negative in Ames tests using a wide range of *S. typhimurium* strains, however, weak mutagenic activity was found using *S. typhimurium* TA98 after S9 metabolic activation. Positive results have been found in three mammalian *in vitro* test systems (Chinese hamster V79 lung cell/HPRT mutation test; mouse lymphoma L5178Y cell/TK mutation test; sister chromatid exchange in Chinese hamster ovary cells). However these results have not always been reproducible. Other mammalian *in vitro* tests for mutagenicity have proved negative (WHO 1994).

A National Cancer Institute study (NCI) (1980) using both rats and mice and dosing via drinking water for 103 weeks concluded that phenol was not carcinogenic to mice or rats under the experimental conditions. There is some evidence to indicate that phenol may be a promoter and/or weak skin carcinogen in specially inbred mice when exposed following initiation with dimethylbenzanthracene (WHO 1994). No human carcinogenicity data, nor any indication of a potential tumorigenic problem has been found despite extensive occupational and medicinal exposure over many decades.

The US Health Advisory values for phenol for one day, ten day and longer term are set at 6 mg l^{-1} based on a 10 kg child drinking one litre of water a day. The RfD (reference dose) and lifetime levels set for a 70 kg adult drinking two litres of water a day are 0.6 mg kg^{-1} day^{-1} and 4 mg l^{-1} respectively. These values are based on the health effects of chronic exposure as reported in the NTP teratology study and NCI carcinogenicity study. However, the aesthetic

effects of phenol contamination of drinking water will become apparent an order of magnitude below these values. Any standards set should be based on aesthetic factors and should also consider the formation of chlorophenols in chlorinated drinking water which will cause aesthetic problems at levels an order of magnitude lower than for phenol itself.

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