EUROPEAN COMMISSION
DIRECTORATE-GENERAL
Joint Research Centre

Institute for Health and Consumer Protection
European Chemicals Bureau
I-21020 Ispra (VA) Italy

PHENOL

Revised Edition

CAS No: 108-95-2

EINECS No: 203-632-7

Summary Risk Assessment Report

November, 2006 **EUR 22522 EN/2**

The mission of the IHCP is to provide scientific support to the development and implementation of EU polices related to health and consumer protection. The IHCP carries out research to improve the understanding of potential health risks posed by chemical, physical and biological agents from various sources to which consumers are exposed.

The Toxicology and Chemical Substances Unit (TCS), commonly known as the European Chemicals Bureau (ECB), provides scientific and technical input and know-how to the conception, development, implementation and monitoring of EU policies on dangerous chemicals including the co-ordination of EU Risk Assessments. The aim of the legislative activity of the ECB is to ensure a high level of protection for workers, consumers and the environment against dangerous chemicals and to ensure the efficient functioning of the internal market on chemicals under the current Community legislation. It plays a major role in the implementation of REACH through development of technical guidance for industry and new chemicals agency and tools for chemical dossier registration (IUCLID5). The TCS Unit ensures the development of methodologies and software tools to support a systematic and harmonised assessment of chemicals addressed in a number of European directives and regulation on chemicals. The research and support activities of the TCS are executed in close co-operation with the relevant authorities of the EU MS, Commission services (such as DG Environment and DG Enterprise), the chemical industry, the OECD and other international organisations.

European Commission
Directorate-General Joint Research Centre
Institute of Health and Consumer Protection (IHCP)
European Chemicals Bureau (ECB)

Contact information:

Institute of Health and Consumer Protection (IHCP)
Address: Via E. Fermi 1 – 21020 Ispra (Varese) – Italy
E-mail: ihcp-contact@jrc.it
Tel.: +39 0332 785959
Fax: +39 0332 785730
http://ihcp.jrc.cec.eu.int/

European Chemicals Bureau (ECB)
E-mail:esr.tm@jrc.it
http://ecb.jrc.it/

Directorate-General Joint Research Centre
http://www.jrc.cec.eu.int

EUR 22522 EN/2
ISSN 1018-5593
Luxembourg: Office for Official Publications of the European Communities, 2006

Printed in Italy

PHENOL

Revised Edition

CAS No: 108-95-2

EINECS No: 203-632-7

SUMMARY RISK ASSESSMENT REPORT

EXPLANATORY NOTE

This report is the revised edition of the the European Risk Assessment Report (RAR) on phenol that has been prepared by Germany in the context of Council Regulation (EEC) No. 793/93 on the evaluation and control of existing substances and published in 2006 on the European Chemicals Bureau website (European Risk Assessment Report Vol. 64, EUR 22229 EN)[1].

Afterwards, the Rapporteur has brought up some changes, mainly on the consumer aspects of the Human health part of the Risk assessment.

The present version incorporates those changes in a consolidated text.

With respect to the previous version of the Summary RAR the changes are the following:

- The wording of the **conclusion (iii)** for dermally exposed consumers was extended (see Section 5.2.1).

- Section 4.1.1.2 has been modified taking into account the more precise wording for the disinfectant scenario. For example, the term “cleaner” was replaced by “disinfectant”.

- The same has been done for Section 4.1.3.2 introductory §.

- Section 4.1.3.2.3 - Irritation/Corrosivity and Section 4.1.3.2.5 - Repeated dose toxicity are now focusing on the dermal route,

- A reference to the Cosmetics Directive Amendment of November 2005 regarding phenol has been included.

- Editorial change: the date of the 29th ATP has been amended (April instead of August)

-

[1] European Chemicals Bureau – Existing Chemicals – http://ecb.jrc.it

PHENOL

Revised Edition

CAS No: 108-95-2

EINECS No: 203-632-7

SUMMARY RISK ASSESSMENT REPORT

Final report, November 2006

Germany

The rapporteur for phenol is the Federal Institute for Occupational Safety and Health.

Contact point:

Bundesanstalt für Arbeitsschutz und Arbeitsmedizin (BAuA)
Anmeldestelle Chemikaliengesetz
(Federal Institute for Occupational Safety and Health Notification Unit)
Friedrich-Henkel-Weg 1-25
44149 Dortmund (Germany)
fax: +49(231)9071-2679
e-mail: chemg@baua.bund.de

Date of Last Literature Search:	**1997 (Environment)** **2003 (Human Health)**
Review of report by MS Technical Experts finalised:	**2005**
Final report:	**November, 2006**

PREFACE

This report provides a summary, with conclusions, of the risk assessment report of the substance phenol that has been prepared by Germany in the context of Council Regulation (EEC) No. 793/93 on the evaluation and control of existing substances.

For detailed information on the risk assessment principles and procedures followed, the underlying data and the literature references the reader is referred to the comprehensive Final Risk Assessment Report (Final RAR) that can be obtained from the European Chemicals Bureau[2]. The Final RAR should be used for citation purposes rather than this present Summary Report.

[2] European Chemicals Bureau – Existing Chemicals – http://ecb.jrc.it

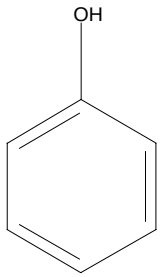
CONTENTS

TABLES

1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTITY OF THE SUBSTANCE

CAS No: 108-95-2
EINECS No: 203-632-7
IUPAC name: phenol
Synonyms: carbolic acid, monohydroxybenzene, phenylalcohol
Molecular weight: 94.11 g/mol
Empirical formula: C_6H_6O
Structural formula:



1.2 PURITY/IMPURITIES, ADDITIVES

Commercial phenol obtained using the cumene method has a purity of > 99.8% and a water content of maximum 0.05% (Phenolchemie GmbH, 1991). Phenol resulting from the cumene method may typically contain the following impurities in the ppm range: mesityloxide, 2-methylbenzofuran, cumene, acetophenone, dimethlyphenylcarbinol, acetone, alpha-methylstyrene, cyclohexanol, hydroxyacetone, sec-butanol, isopropanol, 2-phenylbutene (2) (IARC, 1989).

1.3 PHYSICO-CHEMICAL PROPERTIES

Table 1.1 Physico-chemical properties

Physical state	Phenol is a weak acid. Pure phenol is colourless to light pink crystalline solid. Pure phenol absorbs easily water from air and liquefies.	
Melting point	40.9°C	CRC (1991/92) Ullmann (1991) Kirk-Othmer (1982)
Boiling point	181.8°C at 1,013 hPa	Kirk-Othmer (1982) CRC (1991/92)
Density	1.132 g/cm^3 at 25°C 1.05 g/cm^3 at 50°C	Kirk-Othmer (1982) Ullmann (1991)
Vapour pressure	0.2 hPa at 20°C	Ullmann (1991).
Surface tension	71.3 mN/m at 20°C (0.118% solution in water)	CRC (1991/92)
Water solubility	84 g/l at 20°C (above 68.4°C completely miscible with water)	Ullmann (1991) Sorensen and Arlt (1979)
Partition coefficient	logPow 1.47 HPLC method	Butte et al.(1981)
Flash point	82°C	CHEMSAFE
Auto flammability	595°C	CHEMSAFE
Flammability	not highly flammable	test A.10 not conducted [1)] test A.12 not conducted because of structural reasons
Explosive properties	not explosive	no test because of structural reasons
Oxidising properties	no oxidising properties	no test because of structural reasons
dissociation constant	pKa = 9.89 at 20°C	Lide, D.R. (1994)

1) It is possible to predict the probable behaviour of phenol in such a test on the basis of knowledge of the melting point and the low flash point. Phenol will melt and only be ignitable as a result of a prolonged effect of the flame. After the ignition source has been removed, the flame will go out after a short time. Therefore phenol should be excluded from the possibility of being “highly flammable”.

Odour and taste threshold in water

Phenol and especially most of its reaction products with chlorine (2- and 4-chlorophenol, 2,4- and 2,6-dichlorophenol, 2,4,6-trichlorophenol) have an unpleasant taste and odour. The occurrence of phenol in drinking water is unacceptable, if the substance or one of the reaction products after drinking water chlorination can be detected by taste and odour.

For phenol a threshold for odour perception in air of 184 µg/m^3 and a threshold for taste and odour in water of 150 µg/l has been reported (Verschueren 1996).

Chlorophenols generally have very low organoleptic thresholds. The taste threshold in water for 2-chlorophenol, 2,4-dochlorophenol and 2,4,6,-trichlorophenol are 0.1, 0.3 and 2 µg/l (WHO, 1996).

From these values it can be concluded that phenol in drinking water will normally not give raise to taste and odour problems. But, drinking water containing only a few µg/l of phenol may be

unacceptable after chlorination due to the low threshold values for chlorophenols. Therefore, the Federal Environmental Agency of Germany recommends an aesthetic guide value (*ästhetischer Leitwert*) of 1 µg phenol per litre of drinking water in order to guarantee the option to chlorinate water if necessary without deteriorating its aesthetic quality with respect to taste and odour.

1.4 CLASSIFICATION AND LABELLING

In Germany phenol is classified as belonging to water-hazard class 2 (water-polluting). In the general administrative provisions to the Federal Immission Control Act - Technical Instructions on Air Quality Control (TA-Luft of 27.06.1986) - phenol is listed in Annex E and classified according to class I.

Classification and labelling according to the 29th ATP of Directive 67/548/EEC[3]:

R 23/24/25	Toxic by inhalation, in contact with skin and if swallowed
R 34	Corrosive: Causes burns
R 48/20/21/22	Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed
Muta. Cat. 3, R 68	Possible risks of irreversible effects

Specific concentration limits:

$c \geq 10\%$	T	R23/24/25-48/20/21/22-34-68
$3 \leq c < 10\%$	C; Xn	R20/21/22-34-68
$1 \leq c < 3\%$	Xn	R36/38-68

Annex I of Directive 67/548/EEC does not currently contain any environmentally relevant classifications for phenol.

[3] The classification of the substance is established by Commission Directive 2004/73/EC of 29 April 2004 adapting to technical progress for the 29th time Council Directive 67/548/EEC on the approximation of the laws, regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances (OJ L 152, 30.04.2004, p.1).

2 GENERAL INFORMATION ON EXPOSURE

Phenol is mainly produced synthetically, the most important method being the Hock method on the basis of cumene. The method for the production of phenol, which takes toluene as the starting point, is also of industrial significance. In 1989 the cumene method accounted for about 93% of the production capacity for phenol in Western Europe and the toluene method for about 7%. Phenol can also be obtained by processing coal-tar fractions. Within the EU approximately 15,000 tonnes/annum of phenol are obtained by processing coal-tar fractions.

According to available data there are 32 production and/or processing sites of phenol within the EU. Taking into account the quantities provided in the IUCLID data sets and actual statements of some companies, the resultant quantity of phenol produced in the EU amounts to 1,819,100 tonnes/annum (12 companies). Most of the companies, where phenol is only processed on site, bought phenol from production companies in the EU. In addition, a quantity of 113,400 tonnes/annum is annually imported. 290,000 tonnes/annum of phenol are exported to non-EU member states. The quantity used in the EU therefore amounts to approximately 1,642,500 tonnes/annum.

Phenol is mainly used as an intermediate in organic synthesis. In this, phenol essentially serves as a raw material for the production of bisphenol A, phenol resins, alkylphenols, caprolactam, salicylic acid, nitrophenols, diphenyl ethers, halogen phenols and other chemicals.

A small non-quantifiable part serves as a component in cosmetics and medical preparations. In Germany, phenol is no longer used as a disinfection component in laundry, cleaning, scouring and care agents.

In the Danish product register (2002) the quantity of used phenol is given as 1,378 tonnes/annum. The following product types are described: intermediate, adhesive, binder, impregnating agent, paints, lacquers and varnishes and solvents.

Phenol is also listed in the Swedish product register. In 1993, approximately 15,500 tonnes of the substance were registered as intermediates, binders, in paints and lacquers, flooring, hardeners, insulating materials, adhesives and other products. The Swedish product register (2000) gives the information that there are 5 consumer products that contain phenol. 3 products have a phenol content of maximum 0.1%, 2 products (hardeners for adhesives) have a phenol content of 1 – 5%.

The Norwegian product register for 1994 cites the use of 3,785 tonnes phenol in approximately 100 products. The substance is essentially used as a raw material and additive, binder and adsorbing agent in the manufacture of chemical products and woodworking. More recent data from the Norwegian product register (2002) give the information that phenol is contained in 208 products, containing a total quantity of 2,272 tonnes/annum.

It is not clear from the product registers which quantity of the substance is used as an intermediate in the manufacture of products such as phenol resins and binders and how much remain unchanged in the final product.

The product data base of BgVV is listing some phenol-containing products used by consumers: primers (content <1.0%) and two-component adhesives (content < 2.5%). The exact number of paints/primers being on the market and containing phenol is not known.

The following table shows the main industrial and use categories and the mass balance of phenol for the European market.

Table 2.1 Use categories of phenol according to the Technical Guidance Document

Main category (MC)	Industrial category (IC)	Use category (UC)	Mass balance [in %]
Non-dispersive use (1 b and 3)	Chemical industry (3)	Intermediate (33)	about 100
Wide dispersive use (4)	Personal/domestic (5)	Cosmetics (15) Pharmaceuticals (41) Biocides, non-agricultural (39) Adhesives (2) Impregnation agents (31)	small non-quantifiable part

3 ENVIRONMENT

3.1 ENVIRONMENTAL EXPOSURE

3.1.1 General Discussion

3.1.1.1 Releases into the environment

Phenol is released from a number of man-made sources. The primary sources of environmental phenol are automobile exhaust (direct emission and photochemical degradation of benzene), human and animal metabolism and different combustion processes. From industrial sources, it enters the environment from production and processing operations. Releases also occur due to the waste water from cooking plants and low-temperature carbonisation plants using hard coal and brown coal, from refineries, from pulp manufacture and landfill leachate.

3.1.1.2 Degradation

From the results of standard biodegradation tests it can be concluded that phenol is readily biodegradable. From available investigations on the biodegradation of phenol in surface waters a rate constant of 0.05 d^{-1} was determined. In soils phenol is biodegraded with an experimentally determined rate constant of 0.1 d^{-1}. From this value a rate constant for the biodegradation in sediments of 0.01 d^{-1} was derived.

In biological treatment plants, phenol is estimated to be removed by 87.4% by biodegradation. From site-specific influent and effluent concentrations elimination rates in the range of > 95% to 99% can be calculated for industrial sewage treatment plants. For municipal waste water treatment plants an elimination of 82% was found.

Hydrolysis at environmental conditions is not likely due to the chemical structure of the substance.

In the atmosphere phenol reacts with photochemicaly formed hydroxyl radicals with an estimated half-life of 14 hours ($kdeg_{OH}$ = 0.051 h^{-1}). Products of this degradation are catechol and ring cleavage products. In addition to the photochemical degradation due to hydroxyl radicals, the degradation through NO_3 radicals may also play an important role in the atmosphere. A half-life of approximately 44 minutes ($kdeg_{NO3}$ = 0.95 h^{-1}) can be calculated for phenol from the experimentally determined rate constant and the derived mean NO_3 radical concentration. A half-life of 42 minutes is calculated for the degradation of phenol in the atmosphere ($k_{deg\text{-}air}$ = 1.0 h^{-1}) in consideration of the cited degradation constants for the photochemical degradation with OH- and NO_3 radicals.

3.1.1.3 Distribution

A Henry constant of 0.022 Pa m^3/mol at 20°C is calculated indicating that phenol is only slightly volatile from an aqueous solution.

On the basis of the measured $logP_{ow}$ value of 1.47, the K_{oc} value is calculated as 82.8 l/kg. This value does not indicate a significant potential for geoaccumulation. Consequently, the substance can be washed out of the soil into the ground water by rain water depending on the elimination in soil by degradation and distribution.

With a Mackay I fugacity model the hydrosphere was identified as target compartment (98.8%).

3.1.1.4 Accumulation

From the available test results it can be stated that phenol has only a low bioaccumulation potential. An experimentally determined BCF of 17.5 is used for the assessment.

3.1.2 Aquatic compartment (incl. sediment)

3.1.2.1 Release during production and processing of phenol

For all production sites in the EU site specific data are available for the calculation of the $Clocal_{water}$. 96% of the used quantity of phenol in the EU (approximately 1,572,310 of 1,642,500 tonnes/annum) is covered by site specific calculations of the $Clocal_{water}$. The default emission value from the TGD (0.7%) is used for the remaining tonnage.

Local concentrations from 0.0 to 11.49 µg/l are calculated for all 32 sites of production and processing of phenol in the EU. The 90 percentile of the local concentrations is 2.83 µg/l.

A release of approximately 63.27 tonnes/annum phenol into the hydrosphere (859.85 tonnes/annum release to industrial WWTPs) results for production and further processing at known sites in Europe. For the quantity of 70,190 tonnes/annum of phenol processed at unknown sites the default values from the TGD were used to estimate the emission to water. The emission to waste water is 1.64 tonnes/day (491.33 tonnes/annum release to industrial WWTPs) and the emission to surface water is 61.9 tonnes/annum.

3.1.2.2 Release through the use of products containing phenol

A non-quantifiable part of phenol is used as a constituent of disinfectants and medical preparations (e.g. in skin-peeling preparations). In the case of such use of the substance, release of the total utilised quantity into the municipal waste water can be assumed. It is not possible to undertake an estimation of the $Clocal_{water}$ for these areas of use since no use quantities are known.

In Nordic product registers the presence of phenol in different products is described. For most products only very small amounts of phenol are given. Only for two product types a significant amount of unreacted phenol is indicated: for the product group adhesives and binders and for solvents. The information from the Danish product registers on the amount of phenol contained in adhesives and binding agents was extrapolated to an EU tonnage. Although it is unclear whether this tonnage really addresses unreacted phenol, a generic exposure scenario is estimated for the releases of unreacted phenol from these products to estimate very roughly whether a possible concern for the local aquatic environment is resulting. For the scenario “processing of

binders" a $Clocal_{water}$ of 2.1 µg/l is resulting. For the scenario "release during the service life of the binders" the $Clocal_{water}$ is estimated to 3.5 µg/l.

In addition, the Danish product register gives the information that an amount of 500 tonnes/annum of phenol is used as solvent. If the use in DK is proportional to the consumption in the EU, the EU tonnage would be 40,000 tonnes/annum. No further information on the type and application area of the indicated solvents could be obtained from the Nordic product registers. The European producers of phenol organised in the Phenol Producers Association tried to trace the use of phenol in solvents. They confirmed that to their knowledge phenol is not used in solvents in significant amounts and that an estimate of 40,000 tonnes/annum in Europe would be totally unrealistic. Therefore, the data basis is judged to be too scarce to quantify the amounts and environmental releases of phenol from the use in solvents.

3.1.2.3 Release from other areas

Environmental releases of phenol also occur from the waste water from cooking plants and low-temperature carbonisation plants, from refinery waste water, from pulp manufacture and from landfill leachate. In addition, release of phenol as a product of animal metabolism has to be expected. However, for all these areas it is not possible to estimate a $Clocal_{water}$ due to insufficient data.

Releases from municipal wastewater occur as phenol is a product of human metabolism. If the waste water is purified in a municipal WWTP a $Clocal_{water}$ of 2.52 µg/l can be calculated. For direct discharge of the waste water into the receiving stream a $Clocal_{water}$ of 20 µg/l is to be expected.

3.1.2.4 Data on occurrence in the hydrosphere

Only very few investigations are available with regard to the occurrence of phenol itself in the hydrosphere. They mostly contain only data on the quantity of steam-volatile phenols or on the Phenol Index. The available monitoring data are, in part, relatively old and cannot be assigned to the individual emission sources or the measured values.

3.1.2.5 Sediment

A local water concentration ($Clocal_{water}$) of approximately 2.83 µg/l is calculated from the estimation of exposure for a typical company (90 percentile of the local concentrations). In accordance with the TGD, the sediment concentration can be estimated from this water concentration. A sediment concentration of approximately 0.0073 mg/kg sediment was calculated for phenol.

In 1996 phenol was analysed in sediment of the river Oder (East Germany). The occurrences of phenol in the sediment at different sites are in the range of 0.015 to 45.5 µg/kg.

3.1.3 Atmosphere

3.1.3.1 Release during production and processing

Direct releases into the atmosphere occur during production and processing. The $PEClocal_{air}$ can be calculated for the individual sites by using the currently available emission data of individual production and/or processing companies. Where no site-specific data were available, the $PEClocal_{air}$ calculation was performed using the "default values" of the TGD.

For all production sites in the EU site specific data are available for the calculation of the $PEClocal_{air\text{-}annual}$. 96% of the used quantity of phenol in the EU (approximately 1,572,310 of 1,642,500 tonnes/annum) is covered by site specific calculations of the $PEClocal_{air\text{-}annual}$. The default emission value from the TGD (0.1%) is used for the remaining tonnage.

The calculated $PEClocal_{air\text{-}annual}$ was in the range of 0.027 µg/m³ to 152 mg/m³. The total releases into the atmosphere were estimated to 535.5 tonnes/annum.

3.1.3.2 Release from diffuse sources

Diffuse releases of phenol occur from photochemical degradation of benzene, as a result of vehicle exhaust fume and from further combustion processes. A total release of 96,294 tonnes/annum was estimated for these sources.

3.1.4 Terrestrial compartment

Phenol may enter the soil as a result of the spreading of sewage sludge and liquid manure from livestock farming. For the spread of liquid manure derived from livestock farming over agricultural areas it is not possible to estimate a total release to soil due to insufficient data. A release of phenol into the soil via the spreading of sewage sludge from municipal wastewater treatment plants was estimated to be approximately 5.7 tonnes/annum. Further diffuse release of phenol into the soil is to be expected as a result of deposition from the atmosphere.

A soil concentration (arable soil) of 1.35 µg/kg soil or a soil pore water concentration of 0.85 µg/l result from the deposition of phenol from the atmosphere. If the application of sewage sludge is considered in addition to the deposition rate, a soil concentration (arable soil) of 2.13 µg/kg soil or a soil pore water concentration of 1.35 µg/l is calculated.

The ground water concentration can be calculated in accordance with the TGD as the soil pore-water concentration under the agriculture soil. For a typical company and the use of sewage sludge, a soil pore water concentration of 0.94 µg/l is calculated.

3.1.5 Secondary poisoning

As phenol has only a low bioaccumulation potential it is not required to carry out a risk characterisation for secondary poisoning.

3.1.6 Regional concentrations

All releases, from point sources and diffuse sources, are considered in the determination of a regional background concentration. The summarised local emissions for the production and processing of phenol as well as the diffuse releases of phenol from human metabolism, photochemical degradation of benzene, vehicle exhaust fumes and further combustion processes were distributed between the regional and continental area at a ratio of 10% to 90%.

The following regional environmental concentrations result from the calculations:

$PECregional_{aquatic}$	= 2.41 µg/l
$PECregional_{air}$	= 0.026 µg/m³
$PECregional_{agr.soil}$	= 0.172 µg/kg
$PECregional_{natural\ soil}$	= 0.59 µg/kg

3.2 EFFECTS ASSESSMENT

3.2.1 Aquatic compartment (incl. sediment)

Short and long-term tests are available with fish, invertebrates and algae. With regard to short-term exposure of animals and algae the available valid LC/EC_{50} values point to similar susceptibility of sensitive taxa in fish and invertebrates (crustaceae), compared to a somewhat lower overall sensitivity of algae.

The lowest valid effect value was found in a 60-day fish early life stage test with *Cirrhin mrigala.* A MATC related to survival and growth of 77-94 µg/l was found, that corresponds to a NOEC of 77 µg/l. In a 16-day test with *Daphnia magna* an EC_{10} of 0.46 mg/l was found for the endpoint growth. Although this parameter is not a standardised endpoint the EC_{10}-value was used as long-term effect value for aquatic invertebrates. For the green algae *Selenastrum capricornutum* a 96-hour EC_{50} of 61.1 mg/l was found. NOEC values for algae are only available from tests with longer exposure duration (8 – 14 days).

With an assessment factor of 10 a Predicted No Effect Concentration (PNEC) of 7.7 µg/l was derived from the NOEC for *Cirrhina mrigala.*

3.2.1.1 Effects on microorganisms

The derivation of a PNEC for microorganisms is based on the result from a test on nitrification inhibition, as this was the most sensitive endpoint. With an assessment factor of 10 a PNEC of 2.1 mg/l was derived from the 24-hour EC_{50} of 21 mg/l for *Nitrosomonas* spec.

3.2.1.2 Effects assessment for the sediment

There are no results from sediment tests with benthic organisms available. According to the physico-chemical properties currently known, there is nothing indicating that phenol accumulates in sediment. Therefore a quantitative risk assessment seems not to be necessary for this compartment.

3.2.2 Atmosphere

Data on biotic or abiotic effects in the atmosphere are not available. Because of the short half-life adverse effects are not to be expected.

3.2.3 Terrestrial compartment

Short-term tests with earthworms and plants are available. Using an assessment factor of 1,000 a PNECsoil of 136 µg/kg dw was derived from the lowest effect value, the 14-day LC_{50} of 136 mg/kg dw found for Eisenia fetida.

3.2.4 Secondary poisoning

As phenol has only a low bioaccumulation potential it is not required to carry out a risk characterisation for secondary poisoning.

3.3 RISK CHARACTERISATION

3.3.1 Aquatic compartment (incl. sediment)

The risk assessment for aquatic organisms resulted in a $PNEC_{aqua}$ of 7.7 µg/l. The $PNEC_{microorganism}$ was determined to 2.1 mg/l.

3.3.1.1 Waste-water treatment plants

Production and processing of phenol

A WWTP effluent concentration above the $PNEC_{microorganisms}$ of 2.1 mg/l results for 8 of 32 sites. For these sites the calculations are based on default emissions and default WWTP flow rates of 2 000 m^3/day. Since the ratio of $Clocal_{effl}$ /PNEC > 1, there is an indication of a risk to the microorganism population of the industrial WWTPs.

Site specific data are available for 24 of 32 sites and for those the WWTP effluent concentrations are lower than the $PNEC_{microorganisms}$ and the ratio of $Clocal_{effl}$ /PNEC are < 1. There is currently no indication of a risk to the microorganism population of these industrial WWTPs.

Conclusion (iii).

For 8 out of 32 sites the $Clocal_{eff}/PNEC_{microorganism}$ ratio is > 1. For all these sites the $Clocal_{eff}$ is based on default values and could possibly be lowered by site-specific and traceable exposure data. However, it is not expected to obtain exposure data for all these sites with reasonable efforts and time expenditure.

Conclusion (ii).

This conclusion applies to all sites with $Clocal_{effl}$ /$PNEC_{microorganism}$ ratio is < 1 and also to municipal waste water treatment plants.

Use of products containing phenol

For the release of phenol from processing and use of binders $C_{effluent}$ of 21.41 µg/l and 35 µg/l were estimated. Both values are below the PNECmicroorganism of 2.1 mg/l.

Result

Conclusion (ii).

3.3.1.2 Aquatic environment

Production and processing of phenol

A regional background concentration of 2.41 µg/l for phenol in the hydrosphere is calculated based on all releases of phenol into the environment. This background concentration is added to the $Clocal_{water}$, thus obtaining the PEC_{local} concentrations for the individual point sources.

Taking into consideration the PNECaqua of 7.7 µg/l, there is only one site for which a PEC/PNEC ratio above 1 (1.8) is calculated. The calculation is based on site-specific emissions into the WWTP, site-specific volume of the WWTP and default elimination rates and dilution factor. However, there are indications from non-representative measured effluent concentrations of this site that the actual emissions may be significantly lower than the estimated concentrations. In addition, the default dilution factor of 10 applied for releases into the sea is considered a conservative approach. As a weight of evidence, it can therefore be concluded that there is not an unacceptable risk arising from this site and that there is no need for further information and/or testing.

Result

Conclusion (ii).

Use of products containing phenol

A PEClocal of 4.51 µg/l is estimated for the release of phenol from processing of binders. A PEC/PNEC ratio of 0.59 is resulting for this scenario.

For the release of phenol from the use of binders a PEClocal of 5.91 µg/l was roughly estimated. A PEC/PNEC ratio of 0.77 is resulting for this life-cycle step.

Result

Conclusion (ii).

3.3.1.3 Sediment

Since no effect values for sediment-dwelling organisms are available, it is not possible to perform a quantitative risk assessment for this compartment. Considering the low adsorption potential of phenol, the risk assessment for surface water covers also the sediment compartment.

Result

Conclusion (ii).

3.3.2 Atmosphere

On account of the atmospheric half-life ($t_{1/2}$ = approximately 42 minutes), abiotic effects on the atmosphere, such as global warming and ozone depletion, are not to be expected in the case of phenol.

The calculated concentration in air amounts to 18 µg/m^3 for a typical company (90 percentile of the local concentrations). In consideration of all known sources, a regional air load of 0.026 µg/m^3 results for phenol. Since no data are available on the ecotoxicological effect of the substance through exposure via air, it is not possible to carry out a quantitative assessment for this compartment. Considering the low atmospheric half-life of phenol and that there are no indications of specific toxicity in plants, the performance of further tests is not considered necessary.

Result

Conclusion (ii).

3.3.3 Terrestrial compartment

Releases into the terrestrial compartment are to be expected as a result of deposition from the atmosphere and the spreading of sewage sludge on soils, which are used for agriculture. The deposition rate results from the calculations for a typical company as well as from further diffuse releases into the atmosphere. If there is no spreading of sewage sludge on the soil in the immediate vicinity of a typical company, a soil concentration (arable soil) of 1.35 µg/kg soil or a soil pore water concentration of 0.85 µg/l results. A regional background concentration of 0.59 µg/kg is calculated from all of the releases of phenol into the environment. This background concentration relates to soils that are not contaminated as a result of the spreading of sewage sludge or are not located in the immediate vicinity of a point source (production/processing of phenol).

In consideration of the $PNEC_{soil}$ of 136 µg/kg, a PEC/PNEC < 1 results for soils without direct entry of phenol (i.e. without the spreading of sewage sludge), and a risk to terrestrial organisms is not to be expected.

3.3.3.1 Groundwater

The ground water concentration can be calculated in accordance with the TGD as the soil pore water concentration under the agriculture soil. For a typical company (90 percentile of the local concentrations) and the use of sewage sludge with a concentration of 1.69 mg/kg (dry weight) soil pore water concentration of 0.94 µg/l is calculated. With an odour and taste threshold value of 1 µg/l for drinking water proposed by the Federal Environmental Agency of Germany, no risk for this compartment has to be assumed.

Result

Conclusion (ii).

3.3.4 Secondary poisoning

As phenol has only a low bioaccumulation potential it is not required to carry out a risk characterisation for secondary poisoning.

3.3.5 Unintentional releases

3.3.5.1 Waste Water treatment plants

A WWTP effluent concentration of 0.0252 mg/l results for the continuous release of phenol into the environment via municipal WWTPs.

In consideration of the $PNEC_{microorganisms}$ of 2.1 mg/l, a ratio of $Clocal_{effl}$ /PNEC of 0.012 results for the diffuse source of release of phenol as a product of human metabolism via municipal WWTPs. Since the ratio of $Clocal_{effl}$ /PNEC < 1, there is currently no indication of a risk to the microorganism population of the municipal WWTPs.

Conclusion (ii).

3.3.5.2 Aquatic compartment (incl. sediment)

In the case of the release of phenol as a product of human metabolism, water concentrations of 22.57 µg/l result for direct discharges of municipal waste water into a receiving stream and 5.1 µg/l for indirect discharges into the receiving stream via municipal WWTPs. With regard to Europe it is assumed that approximately 70% of the population release their waste water into the receiving stream via municipal WWTPs and that 30% discharge directly into a receiving stream.

Taking into consideration the $PNEC_{aqua}$ of 7.7 µg/l, a PEC/PNEC ratio > 1 results for the direct discharges of phenol as a product of human metabolism without purification of the municipal waste water in a biological treatment plant. However, this emission path is not the subject of this risk assessment, but further investigations, i.e. measurement of the phenol content in the influent of municipal WWTPs or in untreated municipal waste water and/or monitoring of the phenol content in streams of direct discharges should be considered by the responsible authorities.

It was not possible to provide an estimation of exposure for the aquatic environment with regard to the areas relating to the coking, gasification and liquefaction of coal, refineries and pulp manufacture.

It was not possible to estimate the exposure to the aquatic environment from landfills without landfill leachate collecting system.

Conclusion (i).

3.3.5.3 Atmosphere

There are considerable unintentional diffuse sources for the release of phenol into the atmosphere. Although these releases are not subject of this risk assessment, they had to be considered to derive a realistic background concentration.

A regional background concentration of 0.026 µg/m^3 in the atmosphere is calculated from all of the releases of phenol into the environment. From the qualitative risk characterisation it can be concluded that no unacceptable risk for the atmosphere arises from diffuse emissions of phenol.

Conclusion (ii).

3.3.5.4 Terrestrial compartment

Releases into the terrestrial compartment are to be expected as a result of spreading of sewage sludge on soils, which are used for agriculture. The sewage sludge concentration results from the release of phenol into the municipal waste water as a product of human metabolism. The resultant soil concentrations for phenol amount to 0.0021 mg/kg or 0.0013 mg/l soil porewater, if both sewage sludge application as well as deposition is considered.

In consideration of the $PNEC_{soil}$ of 136 µg/kg, a PEC/PNEC ratio < 1 results for soils on which sewage sludge is spread.

Conclusion (ii).

Phenol may enter the soil as a result of the spreading of liquid manure from livestock farming. For the spread of liquid manure derived from livestock farming over agricultural areas it is not possible to estimate a total release to soil.

It was not possible to estimate the exposure to the terrestrial environment from landfills without landfill leachate collecting system.

Conclusion (i).

4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1 Occupational exposure

Phenol (vapour pressure: 20 Pa) is mainly used as a chemical intermediate in synthesis. Approximately 65% of the produced phenol is processed further to organic chemicals, for example, to bisphenol A, caprolactam, salicylic acid, diphenyl ether, alkyl phenols, nitrophenols and other chemicals. 30% is used to manufacture phenol resins and a non-quantifiable part serves as a component in cosmetics and medical preparations.

For detailed information see Section 2 (general information on exposure).

Based on the available information the following scenarios are regarded to be relevant for occupational exposure:

- Production of phenol and further processing as a chemical intermediate in the large-scale chemical industry (Scenario 1)
- Production of phenolic resins (Scenario 2)
- Use of phenolic resins (Scenario 3)

The exposure as a result of phenol vapours from cosmetic products and medical preparations at room temperature is assessed as low due to the relatively low vapour pressure of the pure substance, the low concentration of phenol (< 2% phenol) and the circumstance, that works with aerosols formed and processes at elevated temperatures are not probable.

Occupational exposure limit values (OEL) and short term exposure levels (STEL) are: (ARIEL, 2002)

OEL		
Finland, Spain, United Kingdom	20	mg/m^3
Germany, Belgium, France, Switzerland, USA (NIOSH/OSHA, AGGIH)	19	mg/m^3
The Netherlands	8	mg/m^3
Austria, Ireland, Italy	7.8	mg/m^3
Sweden, Denmark, Norway	4	mg/m^3
STEL		
Finland, United Kingdom	39	mg/m^3
Germany, Switzerland	19	mg/m^3
Sweden	8	mg/m^3

The exposure assessment is based on measured data and literature data, expert judgement and estimations according to the EASE model (Estimation and Assessment of Substance Exposure). The exposure levels should be regarded as reasonable worst case estimates representing the highly exposed workers.

The results for the different scenarios are summarised in **Table 4.1**.

4.1.1.1.1 Inhalation exposure

For the large scale chemical industry, it is assumed that the production and further processing of phenol is mainly performed in closed systems. Exposure occurs if the systems are breached for certain activities, e.g. filling (Scenario 1).

For production of phenolic resins (Scenario 2) there is a lack of information concerning the processes and companies involved. Since no measurement data are available EASE estimates are taken for exposure assessment. The typical exposure situations are assumed to be similar to Scenario 1.

The manifold uses of phenolic resins are clustered in Scenario 3. From the available data it is seen that relevant exposure occurs at open handling of phenol containing materials, at processes at elevated temperature (processing of phenolic resins in foundries, hardening in furnaces) and during spray-techniques. Surface coating (spraying) is performed in different industries (metalworking/mechanical engineering, woodworking and plastics processing industries). Relatively high exposure levels were measured if compressed-air spray guns and brushes were used (coatings for the preservation of tanks, drums and floors).

4.1.1.1.2 Dermal exposure

For the assessment of dermal exposure, it has to be considered that phenol and its preparations containing ≥ 3% phenol are classified as corrosive. It is expected, that daily repeated skin contact is avoided when pure corrosive substances and preparations classified as corrosive are handled. For phenol, the situation is more complex: beside the corrosive effect, phenol has local anaesthetic properties; therefore afflicted persons described reduced experience of pain after dermal contact with phenol.

According to the revised TGD (Technical Guidance Documents), for classified corrosives, it is not necessary to assess the risk from repeated dermal exposure (only occasional exposure). More important might be dermal exposure to non-corrosive preparations (< 3% phenol). In this case dermal exposure should be taken into account, i.e. repeated dermal exposure cannot be neglected. Quantitative information on dermal exposure is not available. The EASE model is used for assessing dermal exposure.

Due to the low melting temperature of phenol (41°C), transfer and drumming in the large-scale chemical industry (Scenario 1) are performed at temperatures of > 60°C and dermal contacts are avoided. In case of the handling of solid phenol, dermal exposure is assessed in consideration of the corrosive effect of phenol. Bagging of the solid is regarded to be the activity with highest dermal exposure.

For Scenario 2 the handling of corrosive phenol and corrosive resins (≥ 3% phenol) as well as bagging non-corrosive resins have to be taken into consideration.

The Scenario 3 “use of phenolic resins” is subdivided into activities without the formation of aerosols (Scenario 3a) and spray-techniques (Scenario 3b). Additionally, it has to be considered, that dermal contacts to resins classified as corrosive (≥ 3% phenol) is limited to occasional events with rather small skin areas exposed and that skin contacts to non-corrosive resins (< 3% phenol) may occur repeatedly on a daily scale. For the purpose of risk assessment, the higher exposure levels regarding exposure to non-corrosive resins are taken forward.

4.1.1.1.3 Summary of exposure data

The results are summarised in **Table 4.1**.

Table 4.1 Summary of exposure data

Exposure scenario	Duration and frequency of activities relevant for exposure	Inhalation exposure Shift average [mg/m³]	Dermal exposure Shift average [mg/person/day]
Production and further processing / Formulation			
1) Production and further processing as a chemical intermediate	shift length (assumed), daily	3.3	21 [1, 2)]
2) Formulation of phenolic resins	shift length (assumed), daily	20 [1, 3)]	90 [4, 5)]
Use of formulations			
3a) Use of phenolic resins (novolaks, resols)	shift length (assumed), daily	5	13 [1, 6)]
3b) Spraying techniques			300 [4, 7)]

1) Exposure assessment based on model estimates (EASE model)
2) Due to the high melting temperature of phenol, transfer and drumming of the substance are performed at temperatures of > 60°C as a liquid and dermal contacts are avoided.
3) Typical value is comparable to Scenario 1: 3 mg/m³
4) Exposure assessment based on expert judgement, analogy consideration.
5) Dermal contact to phenol and preparations labelled as corrosive (≥ 3 – 15%) is restricted to occasional events and leads to 21 mg/person/day.
6) Dermal contact to phenol and preparations labelled as corrosive (≥ 3 – 15%) is restricted to occasional events and leads to 3 mg/person/day.
7) Dermal contact to phenol and preparations labelled as corrosive (≥ 3 – 15%) is restricted to occasional events and leads to 75 mg/person/day.

4.1.1.2 Consumer exposure

Phenol is used in paints and polishes, products used for floor covering materials, glues, 2-component adhesives and printing inks (Swedish product register, 1995; Berufsgenossenschaft der Bauwirtschaft, 1995). The product data base of the Federal Institute for Risk Assessment (BfR) is listing some phenol-containing products used by consumers: primers (content < 1.0%) and two-component adhesives (content < 2.5%). The exact number of paints/primers being on the market and containing phenol is not known. In the US, 14,000 exposures to phenol-containing products have been reported between 1988 and 1990. The content of phenol was reported to amount up to 26% (Spiller et al., 1993).

4.1.1.2.1 Inhalation exposure

Floor waxes, polishes

30 g of such products containing 2.5% of phenol will be used every day for a period of 0.144 hours. The EPA-SCIES all-purpose liquid cleaner scenario was taken for estimation using the following defaults: air exchange rate 0.2, room volume 20 m³, house volume 408 m³. The estimate revealed a peak room concentration of ~ 12.7 mg/m³, an average concentration during use of ≈ 4.0 mg/m³ and an average concentration of 1.1 mg/m³ after use.

The results of the exposure estimation are given in the **Table 4.2**, for a female active user and a ten year old child as bystander.

Table 4.2 Exposure to phenol by use of polishes/floor waxes

	Room air concentration (mg/m³)	Inhalation rate (m³/h) (1)	Bodyweight (5th percentile) (2)	Estimated exposure (mg/kg bw)	Duration of stay (h) (3)	Exposure (mg/kg)
User during use (female, moderate activity)	12.7	1.6	45	0.45	0.14	0.063 during use
Bystander (child, 10 years, during use, light activity)	4	1	25	0.16	0.14	0.022 during use
After use (female, light activity)	1.1	1	45	0.024	20	0.48 per day
After use (child, 10 years, light activity)	1.1	1	25	0.044	15	0.7 per day

1) EPA (1997);
2) AIHC (1994);
3) Behörde für Arbeit, Gesundheit u. Soziales (1995)

From this estimation, the active user will be exposed to a maximum of 0.063 mg/kg, and to 0.48 mg/kg/day after use. This means that exposure during use can be neglected; however, the daily use of products should be mentioned as the most important source of exposure.

Bystanders, e.g. children, may also be exposed to amounts of about 0.7 mg/kg/bw/day.

Use of phenol containing disinfectants

For estimation exposure due to the disinfectants a weekly use has been assumed. The EPA-SCIES estimate reveals an average concentration in room air of 0.08 mg/m³ after use. This means that a female person staying for 20 hours in that room would have an exposure of 0.018 mg/kg/day, a child staying 15 hours 0.048 mg/kg/day.

Table 4.3 Exposure to phenol by use of disinfectants

	Room air concentration (mg/m^3)	Inhalation rate (1)	Bodyweight (5^{th} percentile) (2)	Estimated exposure (mg/kg bw)	Duration of stay (h) (3)	Exposure (mg/kg)
User during use (female, moderate activity)	10.2	1.6	45	0.363	0.14	0.05 during use
Bystander (child, 10 years, during use, light activity)	3.4	1	25	0.136	0.14	0.019 during use
After use (female, light activity)	0.08	1	45	0.0016	20	0.036 per day
After use (child, 10 years, light activity)	0.08	1	25	0.0032	15	0.048 per day

1) EPA (1997);
2) AIHC (1994);
3) Behörde für Arbeit, Gesundheit u. Soziales (1995)

Use of other products

There are no quantitative data on consumer exposure from the use of phenol containing printing inks and 2-component-adhesives. As a worst case, it is assumed that the exposure is about 10 times lower than that due to floor waxes (mentioned above); the exposure would amount to about 0.02 mg/kg per event.

Cigarette smoke

In a non-ventilated room having a volume of 50 m^3, the smoke from 10 cigarettes (main share of phenol in the side-stream) will result in a phenol concentration of 0.06-0.08 mg/m^3 (Kuwata et al., 1980), resulting in human exposure of about 0.02 mg/kg bw/day (respiratory volume 19 m^3 within 20 hours).

4.1.1.2.2 Dermal exposure

Phenol vapours are absorbed by the dermal route, thus contributing to the total dermal exposure.

Disinfectant solutions

Dermal exposure can occur by putting hands into disinfectant solutions, which can lead to an exposure of 52.5 mg/event (assuming 420 cm^2 · 25 mg/cm^3 (concentration of phenol in the product) · 0.5 (a dilution factor of 2, arbitrary value) · 0.01 (thickness of layer on the skin). The dermal exposure may account for 0.9 mg/kg bw per event.

Floor waxes, polishes

For water based waxes it is assumed that hands will be immersed in the solution for short time. Waxes can also be applied by immersing wiping cloth into a solution containing phenol for further cleaning actions. In this scenario, the maximum possible concentration on the wiping cloth will be similar to that in the cleaning solution. Using wiping cloth with hands will result in contact of 210 cm^2 25 mg/cm^3 (maximum concentration of phenol on wiping cloth)

0.01 (thickness of layer on skin) 0.5 (dilution factor) revealing an exposure of 26.25 mg/event corresponding to 0.44 mg/kg bw per event.

Cosmetics

In EU member states the use of phenol and its alkali salts in soaps and shampoos is permitted in concentrations up to 1% (calculated as phenol); such products must be labelled containing phenol (EU Cosmetics Directive 76/768/EEC and amendments).

For dermal exposure of phenol from soap, the normal case scenario has been assumed as follows: the frequency of use is 6 times per day using 0.8 g of soap. Thus, a consumer using soap is exposed to 800 mg soap product · 6 times/day = 4.800 mg product/day. 4.800 mg product · 0.01 (fraction of phenol) · 0.01 (retention on the skin) = 480 µg/day which corresponds to about 0.01 mg/kg bw/day.

Based on the SCCNFP-guideline the daily exposure with the use of phenol containing shampoo (one event per day) is calculated at 0.08 g/day (8 g/day and retention factor on skin 0.01), there from 1% (fraction of phenol) leads to 800 µg/day. Related to the exposed surface area of 1,430 cm^2 (hands and 1/2 of the head) the external dermal exposure has been estimated to be 0.56 µg/cm^2/day and referring to a body weight of 60 kg 13.3 µg/kg bw/day.

Thus, use of soaps and shampoos containing 1% phenol for their intended purposes will result in a total external dermal exposure of a consumer of 0.021 mg/kg bw/day.

4.1.1.2.3 Oral exposure

Phenol has also been detected as a contaminant in whipped cream dispensers (Sahenk et al., 1978). There is no detailed information available. Therefore this scenario would not be taken forward to the risk characterisation.

4.1.1.2.4 Exposure via medical treatment

The medicinal product, Labiosan Med ® (marketed in a package size of 9 g) contains 0.5% phenol (Hänselwerk, 1995). Application of 300 mg ointment per day to the lips will result in a human exposure of 0.02 mg/kg bw/day (assuming a 100% absorption).

Phenol is also used as a preservative in pharmaceutical preparations for parental administration in a concentration up to 0.5% (Danish Medicines Agency, 2003).

In insulin preparations used by many diabetics throughout the EU, phenolic compounds are used as a preservative in concentrations of 2.15 mg/ml (metacresol 1.5 mg and phenol 0.65 mg/ml). Insulin is dosed on a individual basis, but at an average daily dose for an adult of about 40 IE of insulin/day a diabetic will inject about 0.6 mg metacresol and 0.26 mg phenol (0.004 mg/kg bw) subcutaneously each day. (Lægemiddelkataloget; www.lmk.dk).

Total phenol exposure of the consumer

Chronic exposure by use of phenol-containing consumer products may occur via the inhalation and dermal route. During the application of floor waxes/polishes, and disinfectants consumers may be exposed via inhalation to maximum average concentrations of about 4 mg/m^3

(<10 minutes) with possible peak values of 12.7 mg/m^3 and 10.2 mg/m^3, respectively. The average concentration after use of floor waxes was calculated to be 1.1 mg/m^3.

Considering all consumer products together, it can be assumed that chronic inhalation exposure may not exceed 0.48 mg/kg bw/day and 0.7 mg/kg bw/day, respectively for female adults and 10-year-old children. A very rare acute exposure by using high amounts of paints containing phenol as a conservation agent may lead to higher values. However, the frequency of occurrence of acute exposure cannot be assessed exactly because there is not sufficient information on the number of phenol-containing products available on the market. It is assumed to be low.

Dermal exposure of the consumer via cosmetics (soaps and shampoos) can be assumed to be about 0.021 mg/kg bw/day. The dermal exposure from use of phenol containing floor waxes and disinfectants can account, respectively 0.44 mg/kg bw/event and 0.9 mg/kg bw/event.

4.1.1.3 Humans exposed via the environment

In accordance with the TGD, humans exposed via the environment, such as e.g. via food, drinking water and the air, is to be determined for phenol. As a "worst case" scenario, a point source (90 percentile of the local concentration in water and air) and application of sewage sludge from municipal waste water is used in the calculation. This result is compared with a second calculation, which is based on the regional background concentrations. The resultant daily doses for the substance are a DOSEtot of 46.4 µg/kg bodyweight and day for the local scenario and a DOSEtot of 0.15 µg/kg bodyweight and day for the regional background concentrations.

4.1.2 Effects Assessment

Phenol is well absorbed via gastrointestinal and respiratory tract and the dermal route in animals and humans. Concerning the oral route a high absorption was measured in rats, sheep and pigs with 90, 85, and 84% of the orally administered phenol dose of 25 mg/kg bw after 8 hours. Volunteers exposed to phenol concentrations of 6-20 mg/m^3 via inhalation absorbed 60 to 88% of the substance. After dermal application of phenol to rats, 40% of the applied dose was excreted in the urine by 4 hours, 70% by 12 hours and the excretion was essentially complete (with 75%) by 24 hours. Distribution of phenol in body tissues occurs rapidly. Phenol is metabolised to sulfate and glucuronide conjugates. Excretion via urine is the main elimination pathway of phenol metabolites in humans and animals for the different exposure routes. The ratio of sulfate/glucuronide conjugates excreted in urine is dose-dependent with a capacity-limited sulfatation at high dosages in rats and mice. Cats showed a poor glucuronidation of phenol, only conjugation with sulfate occurred. Small amounts of conjugated hydroquinone were only detected in the metabolic profiles for humans and rats. Metabolism predominantly occurs in liver, gut and kidneys. For risk assessment purposes the rates of oral and inhalation absorption are assumed to be 100%, whereas for dermal exposure the rate was set to 80%.

Signs and symptoms of acute toxicity of phenol in humans and experimental animals are similar regardless of the route of administration. Acute doses of phenol can produce symptoms of toxicity within minutes of administration thus a rapid absorption occurs. Oral toxicity of phenol in humans leading to death is reported for doses as low as 140-290 mg/kg bw. Absorption from spilling phenolic solutions on the skin of humans seems to be very rapid, and death resulted from collapse within 30 minutes to several hours. Death has resulted from absorption of phenol

through a skin area of 64 inch², too. For animals, oral LD_{50} values of 340 mg/kg bw are reported (rats), of approximately 300 mg/kg bw (mice), and of less than 620 mg/kg bw (rabbits). A dermal LD_{50} value of 660-707 mg/kg bw was determined for female rats. LC_{50} values are not available; however, rats are reported to tolerate phenol concentrations as high as 236 ppm (900 mg/m³) for 8 hours, resulting in ocular and nasal irritation, loss of co-ordination, tremors, and prostration. Based on the frequent reports on human experience with occupational exposure to phenol in earlier times phenol is classified as "toxic" and labelled with "R 23/24/25 (Toxic by inhalation, in contact with skin and if swallowed)".

Initial skin contact with phenol produces a white wrinkled discoloration with no experience of pain due to the local anaesthetic properties of phenol. Phenol causes severe chemical burns; occasionally skin necrosis is seen with solutions as dilute as 1%. Eye irritation in rabbits caused by a 5% aqueous phenolic solution was irreversible after an observation period of 7 days. Thus, local irritation caused by phenolic solutions cannot be assessed properly. Based on the corrosive properties phenol is labelled with the R-phrase "R 34, causes burns".

Phenol did not cause any signs of skin sensitisation in tests with guinea pigs (modified Buehler Test) and mice (Mouse Ear Swelling Assay), and there is no evidence of allergic contact dermatitis in humans.

Long-term exposure to phenol has shown effects on the nervous system and liver (in humans and animals), and on hematopoietic and immune system, kidneys, and skin (animals).

A 28-day administration of phenol in the drinking water (4.7, 19.5 and 95.2 mg/l) induced anaemia in CD-1 mice at concentrations of 19.5 mg/l (6.2 mg/kg bw/day). Furthermore, it was shown to induce T- and B-cell suppressive effects (reduced lymphocyte proliferation response to mitogens, antibody levels and T-cell dependent humoral immunity) in mice at low dosages (6.2 mg/kg bw and above). A significantly dose-dependent reduction of erythrocyte numbers by 32% was already seen at the lowest dose of 4.7 mg/l (1.8 mg/kg bw/day, LOAEL). Rats exposed to phenol containing drinking water did not show any alteration of the T-cell dependent humoral response up to 5,000 ppm (301 mg/kg bw/day).

In the 103-weeks cancer NIH studies (1980), F344 rats and B6C3F1 mice were administered drinking water containing 2,500 or 5,000 ppm phenol (equivalent to an assumed phenol uptake of 200 and 450 mg/kg bw/day for rats and 281 and 375 mg/kg bw/day for mice) for 103 weeks. Treated animals showed reduced body weights (rats at high dose, mice at both doses) and reduced water consumption (both species and sexes at both doses). No other relevant toxic effect was seen related to non-neoplastic lesions. Because the reduction of body weight gain was attributed to the reduced water consumption, the NOAEL from this study is 450 mg/kg bw/day for rat and 375 mg/kg bw/day for the mouse, respectively. Haematology parameters were not examined in these studies.

In a study on potential neurotoxicity of phenol male and female Sprague-Dawley rats were treated for 13 weeks via drinking water with phenol concentrations of 200, 1,000 or 5,000 ppm, corresponding to an average intake of, respectively18, 83 and 308 mg/kg bw/day for males and 0, 25, 107 and 360 mg/kg bw/day for females. The Functional Observation Battery evaluation did not reveal any findings of neurotoxicological significance following qualitative or quantitative measurements throughout treatment or following recovery except a significant decrease in body temperature noted for males in the 1,000 and 5,000 ppm groups at the week 13. The motor activity test indicated a significant reduction in total group mean activity counts at the week 4 and 8 for the 5,000 ppm females and for females of the 1,000 and 5,000 ppm group at week 17 during recovery. At week 13 total mean activity counts were significantly higher in

1,000 and 5,000 ppm females. Thus, the NOAEL for neurotoxicity on rats was 200 ppm (18 mg/kg bw/day for males, 25 mg/kg bw/day for females). Dysfunctions of the nervous system including tremor, convulsions, loss of co-ordination, paralysis, reduced motor and spontaneous activity, and reduced body temperature have been reported in other studies.

Specific effects of phenol on the respiratory tract were investigated in a 14 day-inhalation study in F344 rats with a study design similar to OECD TG 412 (nose-only exposure for 10 exposures, 5 days/week, 6 hours/day). No adverse effects were seen at phenol concentrations up to 25 ppm in the respiratory tract or in any other organ system (local and systemic NOAEC of 96 mg/m^3). This value is used in the risk characterisation as NOAEC for local effects after inhalation.

Prolonged dermal exposure of rabbits to phenol induced epidermal hyperkeratosis and ulceration. As dermal NOAEL for systemic effects the dose of 1.18% (130 mg/kg bw/day) was derived from the 18-day rabbit study, whereas the NOAEL for local effects was 2.37% (260 mg/kg bw/day).

Other limited repeated dose studies reported unscheduled deaths after inhalation (100-200 mg/m^3, hamster), dermal exposure (783 mg/kg bw/day, rabbit) or gavage (120 mg/kg bw/day, rat) to phenol, but no treatment-related mortalities have been seen after long-term exposure of phenol within the drinking water at dosages up to 450 mg/kg bw/day in rats and 375 mg/kg bw/day in mice. In some studies, mortalities were associated with growth retardation or respiratory distress. Liver damage has also been observed in rats repeatedly exposed to phenol by inhalation and the oral route (enlarged liver, elevated levels of liver enzymes and liver cell degeneration). Necrosis of renal tubules and papillary hemorrhage have been reported in rats after repeated oral administration.

Limited data are available on chronic effects of phenol in humans from oral, dermal or inhalation exposure indicating reduced spontaneous activity, muscle weakness, pain and disordered cognitive capacities. In phenol-exposed workers (mean exposure duration 13.5 ± 6.55 years) elevated activities for serum transaminases (especially ALAT) and increased clotting time were observed at a time weighted average concentration of 21 mg/m^3 indicating hepatotoxicity after chronic inhalation (LOAEC for systemic effects).

Based on all findings classification as “harmful” and labelling with the R-phrases R 48/20/21/22 “Danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed” has been agreed.

The following overall N(L)OAELs/NOAECs are recommended for risk assessment purposes. Oral administration: LOAEL of 1.8 mg/kg bw/day from the mouse study on subacute toxicity (Hsieh et al., 1992). Inhalation administration: NOAEC of 0.0963 mg/l for local effects (96 mg/m^3) from the 14-day rat study (CMA, 1998a), whereas a LOAEC of 0.021 mg/l for systemic effects (21 mg/m^3) was derived from a time weighted average exposure of workers (Shamy et al., 1994). Dermal administration: A NOAEL for systemic effects of 1.18% (≈ 130 mg/kg bw/day) was derived from the 18-day rabbit study (Deichmann et al., 1950), whereas the NOAEL for local effects was 2.37% (≈ 260 mg/kg bw/day) in the same study.

Phenol did not induce gene mutations in the vast majority of bacterial tests similar to OECD TG 471. In mammalian cell cultures positive effects were found for chromosomal aberrations, micronuclei, and gene mutations (hprt locus, Na+/K+ locus) in mouse lymphoma assays and in several indicator tests (SCE, UDS, DNA strand breaks and DNA adducts). A test for induction of aneuploidy was negative. *In vivo*, negative results were found in rodents for chromosomal aberrations, DNA strand breaks and DNA adducts. *In vivo* micronucleus tests showed negative

and weakly positive results. The frequency of micronuclei is extremely low even in doses which correspond to the LD_{50}. The induction of micronuclei at high doses may be based on an indirect mode-of-action (hypothermia). Drosophila tests were negative. Phenol is classified as a mutagen category 3 and labelled with R 68 "Possible risks of irreversible effects".

In 103-weeks cancer studies on F344 rats and B6C3F1mice with oral administration (drinking water, 2,500 and 5,000 ppm, ≈ 200 and 450 mg/kg bw/day for rats and 281 and 375 mg/kg bw/day for mice, respectively) phenol was not carcinogenic for both species and sexes. A medium-term study in a transgenic mouse model did not give any indication on treatment-related proliferative responses. Phenol was shown to act as a promoter in skin cancer bioassays in mice. A weak carcinogenic effect was observed after long-term skin application of a 10% solution of phenol in benzene (without initiation), but was considered less relevant. The test solution was strongly irritative, and it contained the carcinogen benzene. Some concern may be derived from weakly positive *in vivo* mutagenicity data and from the phenol metabolite hydroquinone classified as a suspected carcinogen (Category 3). This concern is considered to be of minor significance, as long term studies revealed no relevant indication for carcinogenicity. In conclusion, phenol is considered not to be carcinogenic in animals.

There are no data revealing an association of phenol exposure to increased tumour rates in humans. No firm conclusion on risk levels could be drawn from a case-control study on respiratory cancer of workers exposed to phenol.

No data are available on reproductive toxicity of phenol in humans. Phenol was investigated for impairment of reproductive performance and fertility in a two-generation (drinking water) reproductive toxicity study in rats according to OPPTS 870.3700 and OECD TG 416. No adverse effects on reproductive capability and fertility were revealed for either sex across two generations up to and including the highest dosages tested (5,000 ppm, according to 300 (males) and 320 (females) mg/kg bw/day). At this dose reduced water intake, decreased body weight and body weight gain including organ weight impairment were observed in animals of the P0 and F1 generation. No effects on sperm parameters or on estrous cyclicity were revealed. Effects observed during this study were confined to the observation of impaired offspring viability and offspring growth delay during the preweaning period for the groups of the highest tested concentration level. No substance specific embryotoxic or teratogenic potential was revealed for phenol in studies with mice and rats with oral (gavage) administration. From the overall assessment of all available data a NOAEL/developmental toxicity of 93 mg/kg bw/day is derived which is based on the observations upon offspring performance and development in the rat two-generation study. From the evaluation of the available data base on animal investigations there is at present no indication that phenol is a reproductive toxicant.

4.1.3 Risk Characterisation

4.1.3.1 Workers

4.1.3.1.1 Introduction to occupational risk assessment

Exposure to phenol is to be expected during the handling of pure phenol and phenolic resins. The routes to be considered in connection with the workplace are inhalation against phenol vapour

(especially during the hardening process of phenolic resins at elevated temperatures (≤ 180°C)), and dermal contact with the solid substance and its formulations.

Risk estimations are based on studies in humans and animals. The corrosive properties and the serious systemic toxicity might be addressed as the most prominent effects phenol.

For toxicological endpoints with relevant quantitative data MOS values are calculated as quotient of experimental NOAEL (or LOAEL) from animal studies or human case reports and workplace exposure levels. Scientifically based assessment factors describe stepwise the extrapolation of animal data to the worker population. The value of the minimal MOS, as decision mark between **conclusion (ii)** and **(iii)**, results from the multiplicative combination of the different assessment factors and the uncertainty factor. Minimal MOS values may be different for each toxicological endpoint. In a parallel procedure, which gives identical but more direct results, a "critical exposure level" is identified for each endpoint, indicating concern if occupational exposure levels exceed this value.

For risk assessment purposes, for oral uptake and inhalation an absorption percentage of 100%, for dermal contact of 80% is taken forward. For interspecies extrapolation of oral or dermal data, metabolic rate scaling results in lower effective dose levels (in mg/kg bw/day) for humans compared to experimental animals, e.g. for mice the scaling factor is 7, for rats 4, and for rabbits 2.4.

For inhalation the principle of metabolic rate scaling implies that a specific inhalation exposure level (in mg/m^3) is toxicologically equivalent in experimental animals and humans. To maintain this toxicological equivalence, the experimental air concentrations need to be corrected with a factor of 1.5 to reveal the corresponding occupational exposure levels. An additional uncertainty factor is determined which takes into account several aspects, as for instance the reliability of the data base, the biological relevance of the observed effects, the slope of the dose-response curve or the variability of the human population. By definition uncertainty factors have to be based on expert judgement.

Acute toxicity

Systemic effects (inhalation)

Conclusion (iii)

The assessment of acute systemic inhalation toxicity of phenol is mainly based upon the results of an epidemiological study (Shamy et al., 1994). It is reported that a time-weighted phenol exposure of about 21 mg/m^3 resulted in changes of haematological and clinical chemistry parameters, some of them indicating some degree of hepatotoxicity. Not being able to propose more specific adjustment factors, this level of 21 mg/m^3 is taken as starting point of acute risk characterisation in combination with a minimal MOS of 1. Recognising the borderline character of the decision, **conclusion (iii)** is reached for Scenario 2 (formulation of phenolic resins) with the exposure level of 20 mg/m^3. The typical exposure level for Scenario 2 is lower than the reasonable worst case of 20 mg/m^3 (see occupational exposure assessment) and does not lead to concern.

Systemic effects (dermal)

Conclusion (iii)

Dermal contact to liquid or solid phenol causes severe acute symptoms of local and systemic toxicity in humans and animals. Death has occurred in humans which have been exposed by skin contact. Absorption from spilling phenolic solutions on the skin of humans seems to be rapid.

Although a dermal rabbit study is available, the assessment of acute dermal toxicity is preferentially based on the human evidence by inhalation (study of Shamy et al., 1994). The inhalation level of 21 mg/m³ was taken as starting point in combination with a minimal MOS of 1. Assuming a breathing volume of 10 m³, the effective phenol concentration of 21 mg/m³ is converted into the internal dose of 210 mg/person. Based on the assumption of 80% dermal absorption the external starting point for dermal risk characterisation is 263 mg/person.

Conclusion (iii) is reached for Scenario 3b (use of phenolic resins, spraying techniques) for acute dermal toxicity.

Combined exposure

Conclusion (iii).

Combined exposure (dermal contact, inhalation) is expressed as the total internal body burden. The scenario-specific MOS values and the minimal MOS rely upon the same rationale as outlined in the previous section on acute dermal toxicity. For combined exposure **conclusion (iii)** is reached for Scenarios 2 and 3b. For Scenario 2 this conclusion is mainly based on inhalation exposure; for Scenario 3b the most prominent risk factor is dermal contact.

4.1.3.1.2 Irritation/Corrosivity

Acute inhalation

Conclusion (ii).

On the background of the corrosive properties of phenol local effects in the respiratory tract following inhalation are expected.

The NOAEC of 96 mg/m^3 obtained from a 14-day inhalation starting point for the assessment of inhalative local effects. Assessment factors for the identification of the minimal MOS are: (a) 1.5 for physiological differences between humans at rest and workers, (b) 1.3 for differences in study duration (from 6 hours daily to 8 hours of occupational exposure which give a minimal MOS of 2. Thus the corresponding critical exposure level calculates to 48 mg/m³ (96 mg/m³/2). The highest exposure level of 20 mg/m³ results in the lowest MOS of 4.8. There is no concern with respect to local tissue damage after singular exposure.

Sensory irritation

Conclusion (ii).

Sensory irritation is reported from animal data for phenol including a RD_{50}-value of 630 mg/m^3 (166 ppm). This value is multiplied with the factor of 0.03 for prediction of an exposure level with a minimal or low degree of sensory irritation in humans (Alarie 1981) and calculates to the according air concentration of 19 mg/m^3 (630 mg/m^3 · 0.03) for phenol. This value of 19 mg/m^3 is chosen as starting point concerning respiratory depression

For evaluation of the resulting MOS values no further aspects have to be taken into account, an uncertainty factor does not seem necessary. The corresponding minimal MOS is considered to be 1. The critical exposure level thus calculates to 19 mg/m^3.

The highest identified inhalative exposure values are described for the short-term concentration of Scenario 1 with an exposure value of 17.8 and Scenario 2 (formulation of phenolic resins) with an exposure value of 20 (see **Table 4.37**), which both reveal a borderline risk situation. Based on the combined interpretation of the RD_{50} data and human experience **conclusion (ii)** is applied for these occupational exposure scenarios with respect to sensory irritation of phenol.

Single dermal contact/Contact to the eyes

Conclusion (iii).

Phenol has extreme corrosive properties on the skin and eyes. Initial skin contact of humans with phenol produces a white wrinkled discoloration with the affected area turning brown and subsequently becoming gangrenous. Ten percent solutions regularly produce corrosion, and occasionally skin necrosis is seen with solutions as dilute as 1%. Phenol has local anaesthetic properties; therefore the afflicted persons described no experience of pain after dermal contact with phenol.

The formulations handled at the workplace contain up to 15% phenol. For the purpose of risk assessment it is assumed that skin/eye contact with corrosive solutions will cause severe lesions. It is realised that control measures exist for phenol, which should be able to reduce skin and eye exposure, if complied with. However, since a warning effect by local pain may be diminished leading to a weak effect of warning and single exposures might lead to irreversible damage at the skin and eye, concern is expressed for all scenarios in which phenol or its corrosive preparations are handled.

For those phenol preparations which are classified and labelled as irritating to the skin/eyes, **conclusion (ii)** is proposed on the grounds that control measures exist which can minimise exposure and risk of irritation, thereby reducing concern. However, these controls must be implemented and complied with to reduce the risk of skin/eye irritation.

4.1.3.1.3 Sensitisation

Dermal

Conclusion (ii).

Phenol did not cause any signs of skin sensitisation in tests conducted with animals. Likewise there is no evidence of allergic contact dermatitis in humans. Therefore, there is no concern with respect to skin sensitisation at the workplace.

Inhalation

Conclusion (ii).

No information on respiratory sensitisation is available. Phenol is not suspected to be a potent respiratory sensitiser in humans since during all the years of use no notice of specific case reports has been given. No concern with respect to respiratory sensitisation is expressed.

4.1.3.1.4 Repeated dose toxicity

Local effects by inhalation (RDT)

Conclusion (ii).

A 14-day rat inhalation study is available. No adverse effects were seen in the respiratory tract or in any other organ system until the highest tested value of 96 mg/m^3 phenol. This value is used as starting point for MOS calculation.

Some additional guidance results from the Shamy study (see above) where no local effects in the respiratory tract are reported. Combined evaluation of experimental data and human experience may support the conclusion that long-term exposure to about 20 mg/m³ should not result in substantial local effects in the respiratory tract.

Local effects by dermal contact (RDT)

Conclusion (ii).

Single dermal contact was considered to be of concern (**conclusion (iii)**) because the warning effect normally related to the corrosivity of substances might be reduced because of the local anaesthetic property of phenol. It is considered probable, that the isolated experience of skin corrosion at a specific workplace results in the avoidance of daily repeated skin contact. No additional concern.

Systemic effects by inhalation (RDT)

Conclusion (iii).

The assessment of repeated dose toxicity of phenol (systemic effects by inhalation) relies on human and experimental evidence. The most reliable experimental dataset is the 14-day rat inhalation study on phenol. Exposures up to 96 mg/m³ did not result in systemic adverse effects. Special emphasis is given to the results of a human study especially revealing elevated serum levels of liver enzymes and increased clotting time in workers occupationally exposed to a time-weighted phenol concentration of 21 mg/m³. As a LOAEC this value is taken forward to risk characterisation. The minimal MOS of 6 is calculated using an assessment factor of 3 for an LOAEC/NOAEC extrapolation and a further uncertainty factor of 2, especially accounting for the uncertainties of the study-specific exposure level leading to the biochemical changes reported. The corresponding critical exposure level calculates to about 4 mg/m³ (21 mg/m³/6).

There is concern for repeated dose toxicity (inhalation, systemic effects) for all three workplace exposure scenarios. The concern is clear-cut for formulation of phenolic resins (Scenario 2). For the exposure Scenarios 1 and 3 any conclusion proves to be a borderline decision. With reference to the overall interpretation of the data, putting special emphasis to the limited epidemiological results reported by Shamy et al., **conclusion (iii)** is proposed for Scenarios 1 and 3 as well. It is acknowledged that available toxicity data on phenol do not allow a reliable assessment of repeated dose toxicity (inhalation, systemic effects) for the exposure range below 20 mg/m³.

Systemic effects by dermal contact (RDT)

Conclusion (iii).

A dermal rabbit study with substantial limitations shows a NOAEL of 130 mg/kg/day which corresponds to a human dose of 9,100 mg/person/day (130 mg/kg/day · 70 kg) Because of the substantial limitation of the relevance of the dermal NOAEL from the rabbit study, dermal risk assessment is based on the worker experience for the inhalatory route (Shamy et al., 1994, see sections above).

As outlined above the LOAEC for workers is 21 mg/m³. Assuming a breathing volume of 10 m³, the phenol concentration of 21 mg/m³ is converted into the internal dose of 210 mg/person/day. Because of the assumption of 80% absorption by the dermal route, the dose of 263 mg/person/day is taken as external starting point for systemic dermal risk assessment. For calculation of the minimal MOS an assessment factor of 3 for an LOAEC/NOAEC extrapolation and a further uncertainty factor of about 2, especially accounting for the uncertainties of the study-specific exposure level leading to the reported biochemical changes, is proposed. Overall, based on the starting point of the LOAEC of 263 mg/person/day, a minimal MOS of about 6 is proposed. Based on the limited reliability of the worker study, this minimal MOS may not be interpreted as a strict line for reaching conclusions. The corresponding critical exposure level calculates to about 44 mg/person/day (263 mg/person/day divided by 6).

For non-corrosive preparations of phenol repeated dermal exposure levels for Scenarios 2, 3a and 3b have been calculated. For the activities described by Scenario 1 a daily dermal exposure is not assumed. For systemic effects by repeated dermal contact, concern is expressed for Scenario 2 (formulation of phenolic resins) and 3b (use of phenolic resins using spraying techniques).

Systemic effects by combined exposure (RTD)

Conclusion (iii).

For all three occupational scenarios concern has already been expressed for systemic effects following chronic inhalation of phenol. For Scenarios 2 and 3b (spraying techniques) in addition there is a relevant contribution to total systemic health risks by dermal exposure. Overall, **conclusion (iii)** is reached for all occupational scenarios for systemic effects by combined exposure.

4.1.3.1.5 Mutagenicity

Conclusion (ii).

Phenol is positive with respect to various genetic effects in mammalian cell cultures. In general, relatively weak effects are induced. Taking further into account that the frequency of micronuclei in mouse bone marrow cells is extremely low even in doses which correspond to the LD_{50} and the occupational exposure levels are low in comparison to that high experimental exposure levels a substantial mutagenic risk for workers is not anticipated to occur. Recognising the classification as a mutagen category 3, but putting emphasis on semi-quantitative potency considerations, it is proposed to reach no concern.

4.1.3.1.6 Carcinogenicity

Conclusion (ii).

There are no data revealing an association of phenol exposure to increased tumour rates in humans. Oral long term studies on rats and mice showed no effect of phenol on tumour induction. In conclusion phenol is considered not to be a carcinogen in animals. No concern is expressed.

4.1.3.1.7 Reproductive toxicity

Fertility impairment and developmental toxicity

Conclusion (ii).

Phenol was investigated in a two-generation rat study for impairment of reproductive performance and fertility. The observed effects were predominant at exposures that were also toxic to the dams. From the overall assessment of the available animal studies phenol was not identified to possess any specific properties adverse to reproduction.

4.1.3.1.8 Summary of conclusions for the occupational risk assessment of phenol

As result of the occupational risk assessment for phenol, concern is raised for the following toxicological endpoints: acute and repeated dose toxicity (systemic effects) after inhalation, dermal and combined exposure; irritation/corrosivity after single dermal/eye contact. **Table 4.4** summarises the occupational exposure scenarios with concern for phenol.

Special emphasis should be given to the corrosive effect of phenol and its corrosive preparations following dermal contact and contact to the eye. Because of its local anaesthetic properties, the pain following contact with the corrosive substance may be diminished leading to a weak effect of warning and possibly to more intensive local damage of the skin and eye. In order to make risk managers aware of this problem, **conclusion (iii)** is proposed for corrosivity following dermal contact and contact to the eyes for all scenarios.

Putting special emphasis on the human experience reported by Shamy et al. (1994) a critical inhalation exposure level near 4 mg/m³ was proposed. The confidence in this critical exposure level is rather limited because of uncertainties both of the underlying exposure assessment and of the pathological interpretation of the reported significant changes of clinical chemistry and haematology parameters. The overall interpretation of data still supported to express concern for all exposure scenarios with repeated inhalation.

In view of the outcome of the risk characterisation, i.e. the exposures associated with **conclusion (iii)** and the actual national occupational exposure limits, it is recommended to conclude on the necessity to reconsider these values.

Table 4.4 Summary of exposure scenarios with concern for phenol

Scenarios	Acute toxicity systemic		Irritation, corrosivity		Repeated dose toxicity, systemic	
	Inhalation	Dermal	Eyes	Skin	Inhalation	Dermal
1. Production and further processing			iii		iii	
2. Formulation of phenolic resins	iii		iii		iii	iii
3a. Use of phenolic resins (no spray techniques)			iii		iii	
3b. Use of phenolic resins (spraying techniques)		iii				iii

Note Blank fields: conclusion (ii)
Conclusion (iii) already results from inhalative and dermal exposure, therefore no specific concern for the combined exposure Scenario is indicated

Tables 4.5 and **4.6** try to visualise the risk profile of phenol for inhalation and dermal contact. The risk situations (defined by exposure scenario and the critical exposure level for a specific toxicological endpoint) are arranged in such a way, that the "high risk" situations principally are located in the left upper corner of the table, whereas the "low" risk situations are located in the lower right area of the table.

Table 4.5 Ranking of the critical exposure levels for phenol with respect to inhalative exposure at the workplace

Scenario	Exposure level in mg/m³	Repeated dose toxicity, systemic	Acute toxicity, systemic	Acute inhalation/ sensory irritation	Repeated dose toxicity, local	Acute toxicity, local
		Critical exposure level in mg/m³				
		4	21	19	24	48
2. Formulation of phenolic resins	20	iii	iii			
3. Use of phenolic resins	5	iii				
1. Production and further processing	3.3	iii				
	short term: 17.8					

Note Blank fields: conclusion (ii)

Table 4.6 Ranking of the critical exposure levels for phenol with respect to dermal exposure at the workplace

Scenario	Exposure level in mg/p/day	Repeated dose toxicity, systemic	Acute toxicity, systemic
		Critical exposure level in mg/p/day	
		44	263
3b. Use of phenolic resins (spraying techniques)	300	iii	iii
2. Formulation of phenolic resins	90	iii	
1. Production and further processing	21		
3a. Use of phenolic resins (no spray techniques)	13		

Note Blank fields: conclusion (ii)

4.1.3.2 Consumers

4.1.3.2.1 Consumer exposure

Chronic exposure by use of phenol-containing consumer products may occur via the inhalation and dermal route. Consumers may be exposed via inhalation during the application of floor waxes/polishes, and disinfectants to maximum average concentrations of about 4 mg/m^3 (10 minutes) with possible peak values of 12.7 mg/m^3 and 10.2 mg/m^3, respectively. The average concentration after use of floor waxes was calculated to be 1.1 mg/m^3. This concentration will be used in the risk characterisation of chronic exposure. Yearly average dose rates were estimated up to 0.48 mg/kg bw/day for female adults and 0.7 mg/kg bw/day for 10-year-old children.

Dermal exposure of the consumer via cosmetics (soap, shampoo) is assumed to be in the order of about 0.02 mg/kg bw/day. The dermal exposure from use of phenol containing waxes and disinfectants can account to 0.44 mg/kg bw/event and 0.9 mg/kg bw/event, respectively.

No formal risk characterisation has been performed for use of phenol in medicinal products since exposure of this type is regulated under another EU legislation.

4.1.3.2.2 Acute toxicity

Following the exposure assessment, consumers are not expected to be exposed to phenol in the range of hazardous doses which can be derived from dermal toxicity figures (dermal LD_{50} value of 660-707 mg/kg bw). Therefore, the substance is of no concern for the consumer in relation to dermal toxicity.

There may be an acute inhalation exposure to phenol during the application of floor waxes with a maximum average concentration of about 4 mg/m³ (10 minutes) and a possible peak concentration of 12.7 mg/m^3. Because vapours penetrate the skin surface with an absorption efficiency approximately equal to that for inhalation it is impossible to differentiate whether possible detrimental health effects are related to dermal or inhalation exposure. Taking into account all assumptions being applied in the exposure estimation (short duration time, model scenario, worst case conditions) and the weakness of the information from the study on phenol-

exposed workers (Shamy et al., 1994) and the nature and severity of effects, it is concluded there should be no concern for consumers with respect to acute inhalation.

Conclusion (ii).

4.1.3.2.3 Irritation/Corrosivity

Corrosivity is the main effect at the site of contact. Skin and eyes can be severely affected when coming into contact depending on substance concentration (even a 1% phenolic solution is reported to have caused skin necrosis). According to the EU Cosmetics Directive 76/768/EEC and amendments the use of phenol and its alkali salts in soaps and shampoos is permitted in concentrations up to 1%; such products must be labelled "contains phenol".[4]

Following the exposure assessment, consumers are expected to be dermally exposed to phenol. Given the levels of the substance contained in consumer products (up to 2.5%) it can not be excluded that skin irritation will occur despite the short application times (10 minutes).

Conclusion (iii).

4.1.3.2.4 Sensitisation

There is no evidence for skin sensitising properties of phenol by animal tests as well as by human experience.

Conclusion (ii).

4.1.3.2.5 Repeated dose toxicity

Limited data available on chronic effects of phenol in humans from oral, dermal or inhalation exposure indicated reduced spontaneous activity, muscle weakness, pain and disordered cognitive capacities. Animal studies after repeated administration by these routes have also reported dysfunctions of the nervous system including tremor, convulsions, loss of co-ordination, paralysis, reduced motor and spontaneous activity, and reduced body temperature.

Repeated dose studies on animals have reported unscheduled deaths after inhalation (100-200 mg/m^3, hamster), dermal (783 mg/kg bw/day, rabbit) or gavage (120 mg/kg bw/day, rat) exposure to phenol, but no treatment-related mortalities were seen after long-term exposure of phenol within the drinking water at dosages up to 450 mg/kg bw/day in rats and 375 mg/kg bw/day in mice. In some studies, mortalities were associated to growth retardation or respiratory distress.

Anaemia and suppressive effects on the erythropoietic and granulopoeitic stem cells and bone marrow stromal cells were found in studies on mice, whereas no data are available for other species. Application of phenol in drinking water was shown to induce T and B-cell suppressive

[4] According to the amendments of the Cosmetics Directive 76/768/EEC by Directive 2005/80/EC of November 21, 2005, phenol is listed in Annex II (List of substances which must not form a part of the composition of cosmetic products).

effects (reduced lymphocyte proliferation response to mitogens, antibody levels and T-cell dependent humoral immunity) in mice at low dosages (6.2 mg/kg bw and above), however, no effect on T-cell dependent humoral response was found for rats. Atrophic changes of thymus or spleen were occasionally seen in rats repeatedly exposed to phenol by the oral route. No histomorphologic alterations of immune organs were seen in cancer studies on mice and rats.

Inhalation studies

The observations of elevated activities for serum aminotransferases (especially ALAT) and increased clotting time indicating hepatotoxicity in phenol-exposed workers allow to derive a LOAEL of 21 mg/m³ for systemic effects after chronic inhalation (Shamy et al., 1994). The changes in biochemical parameters resulting from occupational exposure to phenol are considered as indications of liver toxicity. In using the Shamy data considerations on interspecies variations are not necessary. No adverse effects on the respiratory tract were reported in a valid 14-day inhalation study on rats (CMA, 1998a). No remarkable differences between control and exposed animals for clinical observation, body weights, food consumption, clinical pathology, organ weights and macroscopic and microscopic post-mortem examinations, at termination and recovery were seen at phenol concentrations up to 96.3 mg/m³.

Dermal studies

Phenol absorption after repeated dermal applications on 18 days at concentrations of 1.18-7.12% aqueous phenol solutions produced tremors (≥ 2.37%) as well as epidermal hyperkeratosis and ulceration in rabbits at concentrations > 3.56%. Signs of systemic intoxication were described at concentrations of 5.93 and 7.12% (Deichmann et al., 1950). The NOAEL for systemic toxic effects was 1.18% (130 mg/kg bw/day). Because of limitations in the study design the relevance of the dermal NOAEL from this rabbit study is considered as questionable. Thus, for dermal risk assessment it is proposed to adjust the worker experience for the inhalation route. Assuming a breathing volume of 10 m³ and 100% absorption, the phenol concentration of 21 mg/m³ is converted into an internal dose of 210 mg/person corresponding to 3.5 mg/kg bw/day. This value has been taken for dermal risk assessment.

Taking into account the variability in the experimental data and the limited validity of some studies there is concern which has to be expressed in the magnitude of the MOS. Following the exposure scenarios there is no reason to assume a special risk for elderly. There may be concern on people suffering from special diseases like anaemia and for children, which has to be expressed in the magnitude of the MOS.

MOS for inhalation exposure scenario

During application of floor waxes the consumer may be exposed to an average concentration of 4 mg/m^3 (for 10 minutes). After application consumers may be exposed to phenol from floor waxes to a concentration of 1.1 mg/m^3.

Local effects

No adverse effects were seen in the respiratory tract of rats until the highest tested value of 96 mg/m³ phenol (CMA, 1998a). The margin of safety between the estimated exposure of 1.1 mg/m³ and the NOAEC (local) of 96 mg/m³ is considered to be sufficient. Thus, there is no concern for local respiratory effects of phenol after repeated inhalation.

Conclusion (ii).

Systemic effects

For systemic effects the value of 21 mg/m³ (LOAEC) from observations on phenol-exposed workers is used. The margin of safety between the estimated exposure of 1.1 mg/m³ and the LOAEC (human) of 21 mg/m³ is considered to be not sufficient.

Conclusion (iii).

This conclusion is based on observations that inhalation of phenol by workers leads to increased activities of ALAT and ASAT in the blood indicating hepatotoxicity. On the other hand, taking into account the limitations of this study (in regard to lacking data on individual exposure, ranges of exposure height, and daily exposure duration as well as on recording time points and duration) and the uncertainties inherent in the exposure estimation (worst case conditions) this scenario may be considered as a border-line case.

MOS for dermal exposure scenarios

Systemic effects

The dermal exposure from use of phenol containing waxes and disinfectants can account 0.44 mg/kg bw/event and 0.9 mg/kg bw/event, respectively. The margin of safety between the internal exposure level of 0.72 mg/kg bw (disinfectants) and the converted human LOAEL (dermal) of ~ 3.5 mg/kg bw/day is judged to be not sufficient taking into account a frequent exposure and that the MOS consideration is based on a LOAEL and the limitations of the human LOAEC used for the route-to-route extrapolation.

Conclusion (iii).

The dermal exposure of consumers due to cosmetics was calculated to about 0.02 mg/kg bw/day. The margin of safety between the estimated internal exposure level of 0.016 mg/kg bw/day and the converted human LOAEL (dermal) of ~3.5 mg/kg bw/day is judged to be sufficient even taking into account that the MOS consideration is based on a LOAEL and the limitations of the human LOAEC used for the route-to-route extrapolation.

Conclusion (ii).

Local effects

The calculation of a combined dermal exposure for consumers (use of cosmetics and phenol containing disinfectants) leads to an exposure of about 0.9 mg/kg bw/day. Local effects were observed in a rabbit study with repeated dermal application at >390 mg/kg bw (3.56% phenol) with epidermal hyperkeratosis and ulceration. As NOAEL for local effects 260 mg/kg bw/day (2.37%) was derived.

The margin of safety between the exposure level of 0.9 mg/kg bw/day and the NOAEL (dermal) of 260 mg/kg bw/day is considered to be sufficient.

Conclusion (ii).

4.1.3.2.6 Genotoxicity

Phenol is positive with respect to various genetic effects in mammalian cell cultures. In general, relatively weak effects are induced. *In vivo*, phenol is a weak inducer of micronuclei in mouse bone marrow cells. However, taking into account that the *in vivo* effects occurred at high doses and the low exposure values a risk for consumers with respect to this endpoint is not expected.

Conclusion (ii).

4.1.3.2.7 Carcinogenicity

Oral long term studies on rats and mice revealed no effects of phenol on tumour induction. A medium-term study on transgenic mice did not give any indication on treatment-related proliferation responses. Phenol was shown to act as a promoter in skin cancer bioassays in mice. A weak carcinogenic effect was observed after long-term skin application of a 10% solution of phenol in benzene (without initiation). However, it is considered less relevant because the test solution contained the carcinogen benzene. A possible concern due to positive *in vivo* mutagenicity data is considered to be of minor significance, as long term studies revealed no relevant indication for carcinogenicity.

Conclusion (ii).

4.1.3.2.8 Reproductive toxicity

No data are available on reproductive toxicity of phenol in humans. Phenol was investigated for impairment of reproductive performance and fertility in a two-generation (drinking water) reproductive toxicity study in rats. No adverse effects on reproductive capability and fertility were revealed for either sex across two generations up to and including the highest dosages tested (5,000 ppm, according to 300 and 320 mg/kg bw/day for males and females, respectively). No effects on sperm parameters or on estrous cyclicity were revealed. Effects observed during this study were confined to the observation of impaired offspring viability and offspring growth delay during the pre-weaning period for the groups of the highest tested concentration level. No substance specific embryotoxic or teratogenic potential was revealed for phenol in studies with mice and rats. Also, no indications for a substance-related specific fetotoxic potential are obtained from the overall assessment of the available data. Based on the results of the 2-generation study a NOAEL/developmental toxicity of 93 mg/kg bw/day can be used for risk characterisation.

Following the exposure assessment, the consumer may be exposed to phenol via inhalation and the dermal route. The daily dose rate of inhalation (0.7 mg/kg bw/day for children) and the estimated dermal body burden (20 µg/kg bw/day) are compared with the NOAEL from a 2-generation drinking water study. There are no reasons to assume that special concern can be derived from this procedure or from the available toxicokinetic information (absorption rate via different routes was set with 100% for inhalation and of 80% for the dermal route).

<u>MOS for the inhalation exposure scenario</u>

During application of floor waxes the consumer may be exposed to an average concentration of 4 mg/m^3 (for 10 minutes) which results in a daily dose of about 0.06 mg/kg bw/day. After application consumers may be exposed to phenol from floor waxes to a concentration of

1.1 mg/m^3 which corresponds to a daily dose rate of 0.48 mg/kg bw/day for adults and of 0.7 mg/kg bw/day for children.

Fertility

The results from the 2-generation study gave no indication for an impairment of fertility. Therefore, fertility is not considered to be a relevant endpoint.

Conclusion (ii).

Developmental toxicity

The calculation of the inhalation exposure of children due to floor waxes leads to a daily exposure of 0.7 mg/kg bw/day. The margin of safety between the estimated exposure level of 0.7 mg/kg bw/day and the NOAEL of 93 mg/kg bw/day is judged to be sufficient.

Conclusion (ii).

MOS for the dermal exposure scenario

Fertility

The results from the 2-generation study gave no indication for an impairment of fertility. Thus, fertility is not considered to be of concern in relation to dermal exposure via cosmetics and uptake from use of phenol containing waxes and cleaners.

Conclusion (ii).

Developmental toxicity

The calculation of the dermal exposure of consumers due to cosmetics and from phenol containing cleaners leads to an exposure of about 0.9 mg/kg bw/day. The margin of safety between the estimated exposure level of 0.9 mg/kg bw/day and the NOAEL of 93 mg/kg bw/day is judged to be sufficient taking into account the rate and the extent of dermal absorption (80%).

Conclusion (ii).

4.1.3.2.9 Humans exposed via the environment

Indirect exposure via the environment has been calculated for oral intake via plant shoot and drinking water. Following the local scenario data (at a point source) an intake of a total daily dose of 0.0464 mg/kg bw/day is calculated with a fraction of the $DOSE_{plant\ shoot}$ of 91%. Following the data for the regional scenario, the total daily dose is smaller ($1.5 \cdot 10^{-4}$ mg/kg bw/day) with the main contributions of the $DOSE_{drw}$ and $DOSE_{plant\ shoot}$ with fractions of, respectively 46% and 41%.

4.1.3.2.10 Repeated dose toxicity-oral intake

A NOAEL for oral administration has not been established. For establishing the MOS for an oral exposure, the LOAEL of the most sensitive species (drinking water study on mice) has been applied. This LOAEL of 1.8 mg/kg bw/day was derived from the subacute mouse study (Hsieh,

1992). Comparing the effect levels for effects on the hematopoietic and immune system mice seem to be more sensitive than rats (LOAEL mice 1.8 mg/kg bw/day versus NOAEL rats > 300 mg/kg bw/day). The effects described in mice as "low observed adverse effect" is anaemia and suppressive effects on the erythropoietic and granulopoeitic stem cells, and bone marrow stromal cells. These effects are considered to be serious health effects. The estimated total body burden with an assumed absorption of 100% is compared to that oral LOAEL.

MOS for the exposure scenario: Humans exposed indirectly via the environment

Local scenario

The calculated internal dose for local exposure is 0.0464 mg/kg bw/day. The margin of safety between the estimated exposure level of 0.0464 mg/kg bw/day and the oral LOAEL of 1.8 mg/kg bw/day is judged to be not sufficient, taking into account considerations on intra and interspecies variation, nature and severity of the effects and possible human populations at risk.

Conclusion (iii).

Regional scenario

The total calculated internal dose for regional exposure is $1.5 \cdot 10^{-4}$ mg/kg bw/day. The margin of safety between this estimated regional exposure level and the oral LOAEL of 1.8 mg/kg bw/day are judged to be sufficient.

Conclusion (ii).

4.1.3.2.11 Repeated dose toxicity-inhalation exposure

MOS for the exposure scenario: Humans exposed indirectly via the environment

Local scenario

For the local scenario data an air concentration of 0.018 mg/m^3 phenol is used (see **Table 4.3**). The NOAEC for local effects at the respiratory tract in the 14-day rat inhalation study (CMA, 1998a) was 96.3 mg/m³, whereas a LOAEC of 21 mg/m³ for systemic effects was derived from a time weighted average exposure of workers (Shamy et al., 1994). The margin of safety for local effects expressed by the magnitude between the calculated exposure of 0.018 mg/m^3 and the NOAEC of 96.3 mg/m^3 is high for local effects.

Conclusion (ii).

The margin of safety for systemic effects expressed by the magnitude between the calculated exposure of 0.018 mg/m³ and the LOAEC for systemic effects (21 mg/m³) is considered to be sufficient.

Conclusion (ii).

Regional scenario

Taking into account the even smaller air concentration in the regional scenario ($2.6 \cdot 10^{-5}$ mg/m^3) there is also no concern.

Conclusion (ii).

4.1.3.2.12 Genotoxicity

Phenol is positive with respect to various genetic effects in mammalian cell cultures. In general, relatively weak effects are induced. *In vivo*, phenol is a weak inducer of micronuclei in mouse bone marrow cells. Taking into account that the *in vivo* effects occurred at high doses and the low exposure values a risk for humans exposed via the environment is not expected.

Conclusion (ii).

4.1.3.2.13 Carcinogenicity

Oral long term studies on rats and mice revealed no effects of phenol on tumour induction. A medium-term study on transgenic mice did not give any indication on treatment-related proliferation responses. Phenol was shown to act as a promoter in skin cancer bioassays in mice. A weak carcinogenic effect was observed after long-term skin application of a 10% solution of phenol in benzene (without initiation). However, it is considered less relevant because the test solution contained the carcinogen benzene. A possible concern due to positive *in vivo* mutagenicity data is considered to be of minor significance, as long term studies revealed no relevant indication for carcinogenicity.

Conclusion (ii).

4.1.3.2.14 Reproductive toxicity

Phenol was investigated for impairment of reproductive performance and fertility in a two-generation (drinking water) reproductive toxicity study in rats. No adverse effects on reproductive capability and fertility were revealed for either sex across two generations. No substance specific embryotoxic or teratogenic potential was revealed for phenol in studies with mice and rats. Also, no indications for a substance-related specific fetotoxic potential are obtained from the overall assessment of the available data. A NOAEL/developmental toxicity of 93 mg/kg bw/day is used for risk characterisation, which is based on the 2-generation study (see Section 4.1.2).

Fertility

The results from the two-generation study gave no indication for an impairment of fertility. Therefore, fertility is not considered to be a relevant endpoint for indirect exposure via the environment.

Conclusion (ii).

4.1.3.2.15 Developmental toxicity

MOS for the exposure scenario: Humans exposed indirectly via the environment

Local scenario

The margin of safety between the internal exposure level of 0.0464 mg/kg bw/day and the NOAEL of 93 mg/kg bw/day is judged to be sufficient.

Conclusion (ii).

Regional scenario

The margin of safety between the regional exposure levels of $1.5 \cdot 10^{-4}$ mg/kg bw/day and the NOAEL of 93 mg/kg bw/day are judged to be sufficient.

Conclusion (ii).

5 RESULTS

5.1 ENVIRONMENT

Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

This conclusion applies to the industrial WWTPs at 8 out of 32 sites. For all these sites the $Clocal_{eff}$ is based on default values and could possibly be lowered by site-specific and traceable exposure data. However, it is not expected to obtain exposure data for all these sites with reasonable efforts and time expenditure. In addition, the concern cannot be removed by testing due to the result from an available respiration inhibition test with industrial activated sludge.

Conclusion (i) There is a need for further information and/or testing.

This conclusion applies to unintentional releases of phenol to:

- the aquatic compartment as a product of human metabolism. Water concentrations of 22.57 µg/l result for direct discharges of municipal waste water into a receiving stream. With regards to Europe it is assumed that approximately 30% of the population release their waste water direct into a receiving stream. Taking into consideration a PNECaqua of 7.7 µg/l, a PEC/PEC ratio > 1 results for the direct discharges of phenol as a product of human metabolism without purification of the municipal waste water in a biological treatment plant. This emission pathway is not the subject of this risk assessment, but further investigations, i.e. measurement of the phenol content in the influent of municipal WWTPs or in untreated municipal waste water and/or monitoring of the phenol content in streams of direct discharges should be considered by the responsible authorities;
- the aquatic environment from cooking, gasification and liquefaction of coal, refineries and pulp manufacture, as it was not possible to estimate the exposure from these areas;
- the terrestrial compartment as a result of the spreading of liquid manure from livestock farming. For the spread of liquid manure derived from livestock farming over agricultural areas it is not possible to estimate a total release to soil;
- the aquatic and terrestrial compartment from landfills without landfill leachate collecting system. It is not possible to estimate the exposure from this area.

Conclusion (ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

This conclusion applies to the production and industrial use of phenol and all environmental compartments, i.e.

Aquatic compartment (incl. sediment);

Terrestrial compartment;

Atmosphere;

Secondary poisoning.

5.2 HUMAN HEALTH

5.2.1 Human health (toxicity)

Workers

Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

There is reason for concern at the workplace.

For phenol risk assessment, three occupational exposure scenarios are defined: production and further processing (Scenario 1), formulation of phenolic resins (Scenario 2) and use of phenolic resins, the latter being divided in a sub-scenario without (3a) and with spraying techniques (3b).

For all dermal exposure scenarios corrosivity following skin contact and contact to the eyes gives reason for concern. It is known, that sensation of pain due to local exposure to phenol may be diminished possibly leading to less awareness and thus higher degrees of local damage. Special emphasis should be given by risk managers to all dermal exposure scenarios (Scenario 1, 2 and 3) when deciding on the possible need for further risk reduction measures.

For all scenarios concern is expressed with respect to systemic toxicity following repeated inhalation. No concern is reached for respiratory tract irritation. In addition, for Scenarios 2 and 3b, concern is expressed for systemic toxicity following repeated dermal exposure. With respect to acute toxicity, concern is indicated for Scenario 2 (only for inhalation) and for Scenario 3b (only for dermal contact).

Consumers

Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

Dermal exposure of consumers via disinfectants leads to **conclusion (iii)** because of systemic repeated dose toxicity and possible skin irritation.

In addition application of floor waxes leads to concern with respect to systemic repeated dose toxicity by inhalation.

Humans exposed via the environment

Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

There is concern for local indirect exposure via plant shoot.

5.2.2 Human health (risk from physico-chemical properties)

There are no significant risks from physico-chemical properties

Conclusion (ii) There is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already.

European Commission
DG Joint Research Centre, Institute of Health and Consumer Protection
European Chemicals Bureau

EUR 22522 EN/2 **European Union Risk Assessment Report**
phenol, **Volume 64, Revised Edition**

Editors: S.J. Munn, K. Aschberger, O. Cosgrove, S. Pakalin, A. Paya-Perez, B. Schwarz-Schulz, S. Vegro

Luxembourg: Office for Official Publications of the European Communities

2006 – 47 pp. – 17.0 x 24.0 cm

EUR – Scientific and Technical Research series; ISSN 1018-5593

The summary report provides the comprehensive risk assessment of the substance Phenol. It has been prepared by Germany in the frame of Council Regulation (EEC) No. 793/93 on the evaluation and control of the risks of existing substances, following the principles for assessment of the risks to humans and the environment, laid down in Commission Regulation (EC) No. 1488/94.

Part I - Environment

This part of the evaluation considers the emissions and the resulting exposure to the environment in all life cycle steps. Following the exposure assessment, the environmental risk characterisation for each protection goal in the aquatic, terrestrial and atmospheric compartment has been determined.

The environmental risk assessment concludes that there is concern for some industrial waste-water treatment plants. For aquatic, terrestrial and atmospheric compartments, and as regards secondary poisoning, there is no concern.

There is a need for further information and for testing regarding the unintentional release of phenol to the aquatic and terrestrial compartments.

Part II – Human Health

This part of the evaluation considers the emissions and the resulting exposure to human populations in all life cycle steps. The scenarios for occupational exposure, consumer exposure and humans exposed via the environment have been examined and the possible risks have been identified.

The human health risk assessment concludes that there is concern for workers, consumers and humans exposed via the environment with regard to irritation/corrosivity of skin and eye and systemic effects induced by repeated exposure.

For human health, as far as physico-chemical properties are concerned, there is no concern.

The conclusions of this report will lead to risk reduction measures to be proposed by the Commission's committee on risk reduction strategies set up in support of Council Regulation (EEC) N. 793/93.

EUROPEAN COMMISSION
DIRECTORATE-GENERAL
Joint Research Centre

The mission of the JRC is to provide customer-driven scientific and technical support for the conception, development, implementation and monitoring of EU policies. As a service of the European Commission, the JRC functions as a reference centre of science and technology for the Union. Close to the policy-making process, it serves the common interest of the Member States, while being independent of special interests, private or national.

European Commission – Joint Research Centre
Institute for Health and Consumer Protection
European Chemicals Bureau (ECB)

European Union Risk Assessment Report

phenol
Revised Edition

CAS No: 108-95-2 EINECS No: 203-632-7

Series: 1st Priority List

Publications Office
Publications.eu.int