European Union Risk Assessment Report

4-chloro-o-cresol

CAS No: 1570-64-5  EINECS No: 216-381-3

Institute for Health and Consumer Protection

European Chemicals Bureau

Existing Substances

European Chemicals Bureau

1st Priority List

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11
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RISK ASSESSMENT
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4-CHLORO-O-CRESOL

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RISK ASSESSMENT

Final report, 2002

Denmark

Rapporteur for the risk assessment report on 4-chloro-o-cresol is the Danish Environmental Protection Agency.

Responsible for the risk evaluation and subsequently for the contents of this report is the Rapporteur.

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Foreword

We are pleased to present this Risk Assessment Report which is the result of in-depth work carried out by experts in one Member State, working in co-operation with their counterparts in the other Member States, the Commission Services, Industry and public interest groups.

The Risk Assessment was carried out in accordance with Council Regulation (EEC) 793/93 on the evaluation and control of the risks of “existing” substances. “Existing” substances are chemical substances in use within the European Community before September 1981 and listed in the European Inventory of Existing Commercial Chemical Substances. Regulation 793/93 provides a systematic framework for the evaluation of the risks to human health and the environment of these substances if they are produced or imported into the Community in volumes above 10 tonnes per year.

There are four overall stages in the Regulation for reducing the risks: data collection, priority setting, risk assessment and risk reduction. Data provided by Industry are used by Member States and the Commission services to determine the priority of the substances which need to be assessed. For each substance on a priority list, a Member State volunteers to act as “Rapporteur”, undertaking the in-depth Risk Assessment and recommending a strategy to limit the risks of exposure to the substance, if necessary.

The methods for carrying out an in-depth Risk Assessment at Community level are laid down in Commission Regulation (EC) 1488/94, which is supported by a technical guidance document. Normally, the “Rapporteur” and individual companies producing, importing and/or using the chemicals work closely together to develop a draft Risk Assessment Report, which is then presented at a Meeting of Member State technical experts for endorsement. The Risk Assessment Report is then peer-reviewed by the Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE) which gives its opinion to the European Commission on the quality of the risk assessment.

If a Risk Assessment Report concludes that measures to reduce the risks of exposure to the substances are needed, beyond any measures which may already be in place, the next step in the process is for the “Rapporteur” to develop a proposal for a strategy to limit those risks.


This Risk Assessment improves our knowledge about the risks to human health and the environment from exposure to chemicals. We hope you will agree that the results of this in-depth study and intensive co-operation will make a worthwhile contribution to the Community objective of reducing the overall risks from exposure to chemicals.

Barry McSweeney
Director-General
Joint Research Centre

J. Currie
Director-General
Environment, Nuclear Safety and Civil Protection

---

1 O.J. No L 084, 05/04/199 P.0001 – 0075
OVERALL CONCLUSIONS/RESULTS OF THE RISK ASSESSMENT

CAS No.: 1570-64-5
EINECS No.: 216-381-3
IUPAC Name 4-chloro-2-methylphenol

Overall result of the risk assessment

(    ) i) There is a need for further information and/or testing.
(X) ii) There is at present no need for further information and/or testing or for risk reduction measures beyond those which are being applied already.
(    ) iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

With regards to the environment, the latest information from the manufacturers on release and monitoring data result in the conclusion that no further information and/or testing is needed as the endpoints do not give rise to concern beyond the need for classification and labelling.

With regard to effects on human health, an older positive in vivo mutagenicity assay was not confirmed by a test performed according to current guidelines. Other mammalian toxicity endpoints do not give rise to concern beyond the need for classification and labelling. While exposure can occur indirectly through the use of pesticides, PCOC itself appears to be used exclusively as an intermediate in the chemical industry.

Summary of conclusions

Environment


The documentation varies from original studies according to OECD test guidelines with GLP to literature references of varying quality. 4-Chloro-2-methylphenol (PCOC) is used in the industry as an intermediate in the synthesis of the phenoxy herbicides MCPA, MCPB, mecoprop (MCPP) and mecoprop-p (MCPP-P). From the industrial production, processing and formulation of phenoxy herbicides, PCOC is emitted to air and wastewater. The release is estimated based on TGD and relevant information about environmental releases during production and formulation obtained from the manufacturers. The produced pesticides contain PCOC as impurity (normally <1%). The use of the pesticides in the agriculture as herbicides results in exposure of soil to PCOC as an impurity and degradation product.

The emissions to surface water from production, processing and formulation sites are local and the number of sites are few. Sludge from the two major production sites is incinerated but one of the wastewater receiving sewage treatment plants (STPs) uses sludge for field application. Therefore, the contribution from sludge application is considered local.
PCOC has been found in water, soil, air and groundwater. In water, PCOC was observed mainly around emission sources, in air near fields applied with MCPA or MCPP, and in soil and biota after the application of the herbicides. The findings in groundwater are assumed to be the result of mobility and reduced degradation under anaerobic conditions.

As MCPA is transformed to PCOC and PCOC has a high vapour pressure, the atmosphere will receive a contribution from soil application of the above mentioned pesticides. PCOC has a low to medium adsorption to organic carbon and may be considered mobile in some soils.

According to an experiment, PCOC is primarily degradable by photolysis in clean water with a half-life of 4 days. However, a re-estimation of photolysis to typical EU surface water resulted in an estimated photolytic degradation half-life of 300-700 days and therefore photolysis is considered negligible. The available biodegradation data are somewhat conflicting but based on a judgement of the balance of evidence the “realistic worst-case” aerobic biodegradation half-life of PCOC in soil is estimated to be 21 days, whereas no biodegradation has been found under anaerobic conditions. The aerobic biodegradation half-life in surface waters is also estimated to be 21 days. The estimated half-life in biological wastewater treatment plants is 0.7 hour resulting in an estimated removal of 88%. The substance is therefore considered to be readily biodegradable (borderline).

PCOC has a bioaccumulation potential based on log K_{ow} 3.09 but BCF found in fish was low (≤30). The risk characterisation of secondary poisoning is therefore not performed.

The exposure assessment is primarily based on monitoring data from the two main manufacturing sites where all production and all processing of PCOC take place and where approximately 60% of the production volume is formulated. A worst-case environmental exposure scenario for a separate formulation site is included in the risk assessment.

PCOC is very toxic to aquatic organisms. The acute toxicity to fish LC_{50} (96h) was observed to be 2.3-6.6 mg/l. The EC_{50} (48h) to daphnids was 0.29-1.0 mg/l and the EC_{50} (96h) to algae was 8.2 mg/l and EC_{10} to algae (96h) was 0.89 mg/l. The NOEC (28 days) for fish was 0.5 mg/l for histopathological changes in kidneys and liver. NOEC (21 days) for Daphnia reproduction was 0.55 mg/l.

The PEC_{local, water}/PNEC_{aquatic organisms} <1.

The PEC_{STP} / PNEC_{microorganisms} <1.

There are no data available on toxicity to soil organisms. Using the equilibrium partitioning method, it was possible to estimate a PNEC_{soil} from PNEC_{aquatic organisms}. The resulting risk quotient indicated no potential risk to soil organisms, i.e. PEC_{soil}/PNEC_{soil} <1. In all above cases the conclusion for the aquatic and soil compartment does not differ regardless of whether the substance is considered readily or inherently biodegradable or whether an assessment factor of 10 or 100 is employed.

PEC_{air}: There are no effect data present and, therefore, no PEC/PNEC ratio can be calculated.
Human health

Information on mammalian toxicity includes both GLP studies done according to OECD guidelines, and older, unpublished citations of limited value. For most endpoints, there appears to be sufficient information to perform a preliminary risk assessment.

The most important sources of direct exposure are assumed to be during production (with predicted exposures of up to 0.7 mg/kg/day) or in conjunction with the use of phenoxy herbicides (containing PCOC as an impurity or breakdown product) where exposures of ca. 0.35 mg/kg/day may occur.

Indirect exposure is estimated as being several orders of magnitude lower than the above values at a regional level while consumer exposure to the substance as an impurity or breakdown product in lawn-treatment sprays may be as high as 0.07 mg/kg/event. Indirect human exposure \(1.2 \times 10^{-4}\) mg/kg/day arising from production and formulation is unlikely to give rise to local concerns.

PCOC is corrosive and toxic by inhalation but is only moderately toxic in acute tests by other routes. The substance is not a sensitiser. According to OECD screening test 422, PCOC did not cause reproductive effects in rats. Tests for repeated dose toxicity suggest a NOAEL of 200 mg/kg and a LOAEL of 800/mg/kg (slight liver toxicity and effects on blood parameters) implying a worst-case safety margin of about 285 at production sites.

PCOC was positive in an older mouse micronucleus test, but negative in a recent test performed according to current guidelines. It did not give rise to genotoxicity in the Ames test. On the basis of current knowledge, the substance cannot be considered a mutagen.
CONTENTS

1 GENERAL SUBSTANCE INFORMATION ................................................................. 4

1.1 IDENTITY OF THE SUBSTANCE ....................................................................... 4

1.2 PURITY/IMPURITIES, ADDITIVES ................................................................... 4

1.3 PHYSICO-CHEMICAL PROPERTIES ................................................................. 4
    1.3.1 Comments on physico-chemical data ......................................................... 5

1.4 CLASSIFICATION .............................................................................................. 5

2 GENERAL INFORMATION ON EXPOSURE ....................................................... 7

2.1 SUMMARY ....................................................................................................... 7

3 ENVIRONMENT .................................................................................................... 8

3.1 ENVIRONMENTAL EXPOSURE ...................................................................... 8
    3.1.1 General discussion ..................................................................................... 8
        3.1.1.1 Production ............................................................................................. 8
        3.1.1.2 Processing ............................................................................................. 9
        3.1.1.3 Formulation ........................................................................................... 9
        3.1.1.4 Environmental release ......................................................................... 9
            3.1.1.4.1 Production, processing and formulation ......................................... 10
        3.1.1.5 Agricultural use .....................................................................................

    3.1.2 Environmental fate ................................................................................... 12
        3.1.2.1 Degradation .......................................................................................... 12
        3.1.2.2 Aerobic biodegradation in water ............................................................ 13
        3.1.2.3 Aerobic biodegradation in soil ............................................................... 14
        3.1.2.4 Anaerobic degradation ......................................................................... 15
        3.1.2.5 Adsorption ............................................................................................ 15
        3.1.2.6 Bioaccumulation .................................................................................. 16
        3.1.2.7 Environmental distribution .................................................................. 16
        3.1.2.8 Summary .............................................................................................. 16

    3.1.3 Aquatic compartment ............................................................................... 17
        3.1.3.1 Measured data ....................................................................................... 17
        3.1.3.2 Aquatic exposure estimations ............................................................... 18
        3.1.3.3 Calculations of PEClocal-water ............................................................... 19
            3.1.3.3.1 PCOC released from production, processing and formulation .......... 19
            3.1.3.3.2 Agricultural use of phenoxy herbicides ........................................... 21

    3.1.4 Atmosphere ............................................................................................... 22
        3.1.4.1 Measured data ....................................................................................... 22
        3.1.4.2 Atmospheric exposure estimations ....................................................... 22

    3.1.5 Terrestrial compartment ........................................................................... 23
        3.1.5.1 Measured data ....................................................................................... 23
        3.1.5.2 Terrestrial exposure estimations ............................................................. 24

    3.1.6 Non compartment specific exposure relevant to the food chain ............... 27

3.2 EFFECTS ASSESSMENT: HAZARD IDENTIFICATION AND DOSE (CONCENTRATION) - RESPONSE (EFFECT) ASSESSMENT ......................................................... 28

    3.2.1 Aquatic compartment ............................................................................... 28
    3.2.2 Atmosphere ............................................................................................... 31
    3.2.3 Terrestrial compartment ........................................................................... 31
    3.2.4 Non compartment specific effects relevant to the food chain ................. 31
3.3 RISK CHARACTERISATION

3.3.1 Aquatic compartment

3.3.2 Atmosphere

3.3.3 Terrestrial compartment

3.3.4 Non compartment specific effects relevant to the food chain

4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1 General discussion

4.1.1.2 Occupational exposure

4.1.1.3 Consumer exposure

4.1.1.4 Indirect exposure via the environment

4.1.1.5 Combined exposure

4.1.2 Effects assessment: Hazard identification and Dose (concentration) - response (effect)

4.1.2.1 Toxicokinetics, metabolism and distribution

4.1.2.2 Irritation and Corrosivity

4.1.2.3 Sensitisation

4.1.2.4 Repeated dose toxicity

4.1.2.5 Mutagenicity

4.1.2.6 Carcinogenicity

4.1.2.7 Toxicity for reproduction

4.1.3 Risk characterisation

4.1.3.1 General aspects

4.1.3.2 Workers

4.1.3.3 Consumers

4.1.3.4 Humans exposed indirectly via the environment

4.1.3.5 Combined exposure

4.2 HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

4.2.1 Exposure assessment

4.2.2 Effects assessment: Hazard identification

4.2.2.1 Explosivity

4.2.2.2 Flammability

4.2.2.3 Oxidizing potential

4.2.3 Risk characterisation

5 RESULTS

6 REFERENCES

ABBREVIATIONS

Appendix A Photolysis of PCOC in surface water

Appendix B QSAR estimations on toxicity

Appendix C TGD estimations on exposure

Appendix D Estimation of inhalation and dermal exposure EASE-model

EUSES Calculations can be viewed part of the report at the website of the European Chemicals Bureau:
http://ecb.jrc.it
Tables

Table 3.1 Regional environmental release estimated according to TGD................................................................. 11
Table 3.2 Scenario on formulation ..................................................................................................................... 11
Table 3.3 PCOC concentration in on-site effluent before entering the STPs............................................................. 19
Table 3.4 Concentration in wastewater before treatment in municipal STP ............................................................ 20
Table 3.5 Local concentration during emission episode, PEClocal, river water ...................................................... 20
Table 3.6 Annual average concentration, PEClocal, river water, ann .................................................................... 21
Table 3.7 Predicted environmental local concentration in sediments (PEClocal, sed) .................................................. 21
Table 3.8 PECSTP, microorganisms .................................................................................................................... 21
Table 3.9 PEClocal, air calculations .................................................................................................................... 23
Table 3.10 Deposition from the atmosphere calculations ....................................................................................... 23
Table 3.11 Concentration in soil ........................................................................................................................ 25
Table 3.12 Concentration in sludge and in soil just after sludge application ........................................................... 25
Table 3.13 Removal rate constants ..................................................................................................................... 26
Table 3.14 Local concentration in soil ................................................................................................................ 26
Table 3.15 Local concentration in soil porewater .................................................................................................. 26
Table 3.16 Local concentration in groundwater ................................................................................................ 27
Table 3.17 Fish, short term .................................................................................................................................. 28
Table 3.18 Crustaceans, short term ...................................................................................................................... 28
Table 3.19 Algae ................................................................................................................................................... 28
Table 3.20 Higher plants, short term .................................................................................................................. 29
Table 3.21 Microorganisms .................................................................................................................................. 29
Table 3.22 Prolonged, fish ...................................................................................................................................... 29
Table 3.23 Long-term Daphnia, reproduction ....................................................................................................... 29
Table 3.24 PEC/PNEClocal for aquatic organisms ............................................................................................... 32
Table 3.25 PEC/PNEC microorganisms ............................................................................................................... 32
Table 3.26 PEC/PNEClocal, soil .......................................................................................................................... 33
Table 4.1 Calculated exposure data excl. agricultural spraying ............................................................................. 39
Table 4.2 Data on acute toxicity of PCOC .......................................................................................................... 42
Table A.1 Extrapolations according to Zsd and Z of the “standard EU environment” ............................................. 65
Table B. Comparison between QSAR predicted and experimental L(E)Co-values for fish and daphnia ............... 66
Table C.1 Concentration in wastewater before treatment in municipal STP ......................................................... 67
Table C.2 Predicted environmental local concentration in surface water during episode (PEClocal,water) ............... 68
Table C.3 The predicted annual average concentration in surface water (PEClocal, water, ann) ................................. 68
Table C.4 Predicted environmental local concentration in sediments (PEClocal, sed) .............................................. 69
Table C.5 PEClocal, air calculations ..................................................................................................................... 70
Table C.6 Deposition from the atmosphere calculations ....................................................................................... 70
Table C.7 Deposition from the atmosphere per kg of soil ..................................................................................... 71
Table C.8 Concentration in soil .......................................................................................................................... 71
Table C.9 Predicted environmental local concentration in sludge (PEClocal,sludge) .................................................... 72
Table C.10 Concentration in soil just after sludge application ................................................................................ 72
Table C.11 Removal rate constants ..................................................................................................................... 73
Table C.12 Initial concentration in soil after 10 years of deposition and sludge application ................................. 73
Table C.13 Estimated local concentration in soil ................................................................................................ 73
Table C.14 Local concentration in soil porewater ................................................................................................ 74
Table C.15 Local concentration in groundwater ................................................................................................ 74
1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTITY OF THE SUBSTANCE

CAS-No.: 1570-64-5
EINECS-No.: 216-381-3
IUPAC Name: 4-chloro-2-methylphenol
Synonyms: 4-chloro-o-cresol
            para-chloro-ortho-cresol (PCOC)
Trade Name: 4-chloro-o-cresol
Synonym: Chlorocresol
Molecular formula: C_7 H_7 Cl O
Structural formula:

\[
\begin{align*}
\text{OH} & \\
\text{Cl} & \quad \text{CH}_3
\end{align*}
\]

Molecular weight: 142.59

1.2 PURITY/IMPURITIES, ADDITIVES

Purity: approx. 97% w/w
Impurities:
- 2-chloro-6-methylphenol (OCOC, CAS: 87-64-9) <1.0%
- 2-methylphenol (OC, CAS: 95-48-7) <1.0%
- 2,4-dichloro-6-methylphenol (DCOC, CAS: 1570-65-6) <2.0%
- 4-chloro-2,6-dimethylphenol <0.5%
- 4-chlorophenol <0.5%
- 5-chloro-2-methylphenol <0.2%
Additives: None

1.3 PHYSICO-CHEMICAL PROPERTIES

Physical state: solid at 25°C, 1 atm
Melting point: 46-50°C (OECD 102, Qvist Lab. 1983)
Boiling point: 231°C (OECD 103, Qvist Lab. 1983)
            150-155°C at 20-25 hPa (BASF 1994)
Relative density: 0.4769 g/cm³ at 20°C (OECD 109, Qvist Lab. 1983)
            1.2 g/cm³ at 50°C (BASF 1994)
Vapour pressure: 26.66 Pa at 20°C (OECD 104, Dantest 1983)
            160 Pa at 70°C (BASF 1994)
Water solubility: 2300 mg/l (20°C), (OECD 105, Qvist Lab. 1983)
Octanol/water (K_ow): log K_ow = 3.09 (OECD 107, BASF 1994)
               pK_a 9.71 (BASF 1994)
Explosivity No information (unlikely, structural considerations)
Flammability Not flammable (EF 3.10, Quist Lab. 1983)
Oxidizing potential Non oxidizing (PC II Annex V EEC/831/79, Dantest 1983)
1.3.1 Comments on physico-chemical data

The physico-chemical data are present but with variations that may be attributed to different test methods, temperatures, etc. The results from tests performed by OECD guidelines have been used instead of handbook values and literature references when the test method has not been mentioned.

The melting point 50°C was found in an OECD 102, capillary method. The boiling point of 231°C has been found in an OECD 103, Siwoloboff method. The vapour pressure of 26.66 Pa (0.2 mmHg) at 25°C was found in an OECD 104 method and is used instead of the 3.2 Pa at 25°C mentioned by Seiber et al. (1986) who only states that the result was found in a laboratory study.

The water solubility was found to be 2,300 mg/l (0.23 w/w%) in an OECD 105 at 20°C. In a BASF study, a water solubility of 7,600 mg/l at 25°C is found after solution in hot water, centrifugation and GC analysis without reference to test method. A water solubility of 1,312 mg/l is mentioned by US-EPA (1996) without specifying the test method. A QSAR estimation by the EPIWIN model calculations (Meylan and Howard, Syracuse, 1995) results in an estimated water solubility of 1,765 mg/l. The water solubility of 2,300 mg/l which was observed in an OECD test is evaluated to be the most valid and is used in the risk assessment. The octanol/water partition coefficient log $K_{ow}$ is stated to be 2.78 according to US-EPA (1996) without specifying the test method. Hansch et al. (1995) found an experimental value of 2.63. QSAR calculations by fragment analysis (KOWWIN, Syracuse 1995; ClogP) estimated the log $K_{ow}$ to be 2.70 and 3.13, respectively. However, a test performed according to the OECD guideline resulted in log $K_{ow}$ to be 3.09 which is used in the risk assessment.

The explosive properties of the substance have not been tested. However, no reports of explosive properties were found in the available literature, nor does the chemical structure contain elements associated with explosivity. According to methods EF 3.10 and EF 3.10 mod. the substance does not burn, nor is it flammable in contact with water (Quist Laboratory, 1983). The substance was classified as non oxidizing according to the test method from the working group PC II Annex V EEC/831/79, sixth amendment of Dir. 67/548/EEC (Dantest, 1983).

1.4 CLASSIFICATION

Classification and labelling according to the 26th ATP of Directive 67/548/EEC:

Classification:  
T; R23 Toxic by inhalation  
C; R35 Corrosive, Causes severe burns  
N; R50 Dangerous for the environment,  
Very toxic to aquatic organisms

Labelling:  
T; C; N  
R: 23-35-50  
S: (1/2-)26-36/37/39-45-61

---

Specific concentration limits:

\[
\begin{align*}
C \geq 25\%: & \quad T; C; R23-35 \\
10\% \leq C < 25\%: & \quad C; R20-35 \\
5\% \leq C < 10\%: & \quad C; R20-34 \\
3\% \leq C < 5\%: & \quad Xn; R20-36/37/38 \\
1\% \leq C < 3\%: & \quad Xi; R36/37/38
\end{align*}
\]

Comments on classification

The substance may be regarded as a borderline readily biodegradable substance but it is noticed that the data are somewhat conflicting (cf. section on biodegradability). The log $K_{ow}$ is greater than 3 but available BCFs in fish are below 100. The closely related isomer 4-chloro-3-methylphenol recently has been classified in regard to the environment: N, R50 (EU classification).
2 GENERAL INFORMATION ON EXPOSURE

PCOC is used as an industrial intermediate in the production of pesticides. The environmental exposure is due to release of the substance from the manufacturing of the substance itself and when using the substance for manufacturing and formulation of the phenoxy herbicides: MCPA (4-chloro-2-methylphenoxy acetic acid), MCPP (4-chloro-2-methylphenoxy butyric acid), and mecoprop [2-(4-chloro-2-methylphenoxy)-propionic acid (MCPP)]. Furthermore, environmental exposure is due to the agricultural use of the above-mentioned pesticides. Mainly, because these pesticides are degraded in the environment to PCOC (main degradation product) but also because PCOC is an impurity in these pesticide formulations.

Production figure

In EU, PCOC has been produced in the United Kingdom (UK), in the Netherlands, and in Germany (BUA, 1994). In 1996, the major production took place in the UK by two major manufacturers. Other production sites in the EU with a production below 1,000 tons/year may exist but this data will not be available until 1998. In addition, PCOC may be produced as a non-isolated part of a continuous process which need not be reported under the Regulation. At least one such site has been identified in The Netherlands.

The current production of PCOC in the EU takes place in the UK by A.H.Marks and Nufarm. A part of the production volume of technical phenoxy acids is exported to be formulated elsewhere within and outside EU.

According to US-EPA, it is not known whether three different US manufacturers are still producing PCOC and using PCOC for manufacturing of MCPA, MCPB and MCPP (US-EPA 1996). The total production and consumption in the EU, import and export etc. could not be obtained. Therefore, two methods have been used for the estimation.

Based on the information from the two manufacturers of PCOC (pers.comm., 1997), the total production of PCOC in the EU is estimated to be approximately 15,000 tons/year. The latest figures from the ECDIN database (1995) indicate the value of production and consumption of the relevant pesticides to be approximately 22,000 tons in 1989.

PCOC constitutes the main molecular fraction of the herbicides. Therefore, the total annual use of PCOC in the EU is estimated using stoichiometric calculation to be approximately 15,000 tons. Recent data from manufacturers (confidential papers, October 1997) inform that the production of phenoxy acid herbicides in 1996 was 21,000 tonnes of which 13,000 was exported. This information supports the estimation of the production PCOC to be approximately 15,000 tonnes per year. The import is unknown but assumed to be 0.

2.1 SUMMARY

The tonnage of PCOC in the EU has been estimated to a total of 15,000 tons per annum based on the production volumes presented by the manufacturers and supported by the production and consumption figures of the herbicides MCPA, MCPB and MCPP in 1989. Main points of emissions are at manufacturing sites of the substance where PCOC is used as an intermediate for manufacturing of the phenoxyherbicides (i.e. PCOC processing and phenoxyherbicides formulation sites) and where these herbicides are used in agriculture.
3 ENVIRONMENT

3.1 ENVIRONMENTAL EXPOSURE

The environmental exposure assessments considered combine the relevant exposure scenarios for the substance and apply recommended assessment methods for deriving PEC local and regional according to TGD (1996) and EUSES ver. 1.0 (1997). During 1997 monitoring studies have been performed by the two main manufacturers at their production sites which are both located in the UK. The results are used in the exposure assessment but for confidentiality average values are used. The confidential report with the monitoring results has been submitted to the Competent Authorities.

The environmental exposure scenarios according to TGD would include:

a) Production of PCOC.
b) Processing: Manufacture of technical phenoxy acids (MCPA, MCPB and MCPP/MCPP-P).
c) Formulation of the phenoxy herbicides.
d) Agricultural use of herbicides.

In the TGD, “processing” covers all kinds of processes where the substance as such is applied or used, including application of use of preparations or articles containing the substance. In this risk assessment report, the term processing only covers the industrial transformation of PCOC into phenoxy acids. The term formulation covers the final stage in the manufacture of the plant protection products.

In this assessment report, the applied scenarios are based on the adoption of monitoring data from the two main manufacturers. The monitoring data covers the total emissions from the main production sites where production, processing and formulation takes place on the same manufacturing site. A part of the phenoxy acids are formulated elsewhere and therefore this scenario is included and the exposure estimations are based on TGD since monitoring data were not available.

<table>
<thead>
<tr>
<th>Scenario:</th>
<th>Exposure estimation based on:</th>
<th>Compartment of primary release:</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) Production of PCOC, processing and formulation of phenoxy herbicides at the same site</td>
<td>Monitoring data</td>
<td>Surface water</td>
</tr>
<tr>
<td>b) Formulation at another site</td>
<td>TGD/EUSES (generic)</td>
<td>Surface water</td>
</tr>
<tr>
<td>c) Agricultural use of herbicides</td>
<td>USES 1.0 (generic)</td>
<td>Agricultural soil</td>
</tr>
</tbody>
</table>

3.1.1 General discussion

3.1.1.1 Production

PCOC is produced and used in the production of pesticides and the exposure to the environment is related to the production of the pesticides and the degradation of the pesticides after application. The chlorophenols used in the production of MCPA, MCPB, MCPP and MCPP-P
are obtained by chlorination of o-cresol, a starting material in abundant supply in Europe as a product of coal tar distillation.

Although, chlorination occurs mostly at the 4-position, 6-chloro- and 4,6-dichloro-2-methylphenol are also produced when chlorine or alkali hypochlorites are used. Other cresol production ways are catalytic and thermal cracking of naphtha fractions during petroleum distillation, or direct production of o-cresols from methylation of phenol in the presence of catalysts (Fiege, 1986).

\[
\text{CH}_3\text{C}_6\text{H}_4\text{OH} + \text{HCl} \rightarrow \text{CH}_3\text{C}_6\text{H}_3\text{-(OH)(Cl)} + 2\text{H}^+
\]

The crude PCOC is purified by distillation.

### 3.1.1.2 Processing

Most of the o-cresol manufactured in Europe is chlorinated to 4-chloro-o-cresol (PCOC) the starting material of the chlorophenoxyalkanoic acids; 4-chloro-2-methylphenoxy acetic acid (MCPA), 4-chloro-2-methylphenoxy butyric acid (MCPB), 2-(4-chloro-2-methylphenoxy)-propionic acid (mecoprop, MCPP) and its isomer mecoprop-p (MCPP-P).

**MCPA**


MCPA acid is formed by condensation of PCOC with monochloroacetic acid.

**Mecoprop (MCPP) / Mecoprop-P (MCPP-P)**

CAS no.: 7085-19-0 (racemate), 16484-77-8 (MCPP-P). Molecular formula: C\(_{10}\)H\(_{11}\)ClO\(_3\).

Molecular weight: 214.7.

MCPP is formed by the condensation of PCOC with 2-chloropropionic acid. MCPP-P which is the biocidal active isomer is manufactured by a similar process in the same plant, but by using the single isomer S(-)2-chloropropionic acid.

**MCPB**


MCPB acid is manufactured by condensation of PCOC with gamma butyrolactone.

The phenoxy acids are produced as flaked solids, sold for further processing elsewhere or used on site for making fluids, aqueous salts or esters or formulations.

### 3.1.1.3 Formulation

MCPA, MCPB, and MCPP are formulated and sold as various kinds of phenoxy herbicides: acids, salts or esters. This takes place either at the manufacturer site or elsewhere.

### 3.1.1.4 Environmental release

Environmental release may take place from the production of PCOC, processing into phenoxy acids or from the formulation of the herbicides. All three processes may take place at the same
site or the technical phenoxy acids is sold for the final formulation to take place at a separate site. According to manufacturers (pers. comm. 1997) no PCOC leaves the production sites.

3.1.1.4.1 Production, processing and formulation

According to TGD, the main category is Ib (intermediates stored on-site), the industrial category is 3 (chemical industry: chemicals used in synthesis) and the use category 33 (intermediates). The release from production, processing and formulation of PCOC is estimated based on monitoring data or by using the Emission Scenario Document (TGD, part IV, 1996) on intermediates. For confidentiality, the average volumes are used.

Production

The local and regional values from production are based on the approximate production volume per production site according to the information given by the two main manufacturers covered by the Regulation (i.e. at each site 1/2 of the total EU production volume). All wastewater from these two industrial plants is treated in municipal biological Sewage Treatment Plants (STPs) before release to surface waters.

Processing

PCOC is used at the manufacturing site in the production of technical phenoxy acids and thus remains on-site. It has not been possible to obtain a precise figure for the number of plants producing and processing PCOC in the EU. However based on information from the main manufacturers, the total number of processing plants in the EU covered by the Regulation is two which is used in this risk assessment.

Formulation

The technical phenoxy acids are formulated into phenoxy herbicides. It has not been possible to obtain a precise figure for the number of plants formulating phenoxy herbicides (based on PCOC) in the EU. The two main manufacturers inform that they each formulate approximately 60% of the production volume of phenoxy acids on-site, that at least 30% of the production volume of phenoxy acids are exported out of the EU and approximately 5% to 10% of the production volume is formulated outside the two main manufacturing sites but within the EU.

As worst case, therefore, the approximate volume formulated elsewhere is estimated to be 10% of the total production volume and all formulated at one site.

Summary of annual total production, processing and formulation volumes

| Production: | 15,000 tonnes PCOC |
| Processing: | 15,000 tonnes PCOC used to produce 21,000 tonnes phenoxy acids, 30% exported for formulation outside EU |
| Formulation: | 60% of production volume formulated on-site (9,000 tonnes PCOC eqv.) and 10% of the total phenoxy acid volume formulated elsewhere within the EU (cf. below) |
Regional release

For regional release, based on the information provided by the major manufacturers it is estimated that each production site is located in one region. Two main manufacturers are known, therefore, at the specific site ½ of the total EU PCOC production volume occurs and is processed into phenoxy acids and that 60% of this volume is formulated into the final product. The total volume released in the region is averaged over the year (365 days).

According to TGD the following default regional releases would occur:

Table 3.1 Regional environmental release estimated according to TGD

<table>
<thead>
<tr>
<th>Life-stage</th>
<th>Production of PCOC</th>
<th>Processing (manufacture of phenoxy acids)</th>
<th>Formulation of phenoxy herbicides</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of sites</td>
<td>2 (~7500 t/yr)</td>
<td>2 (~7500 t/yr)</td>
<td>2 (~4500 t/yr)</td>
<td></td>
</tr>
<tr>
<td>Emission to:</td>
<td>Fraction</td>
<td>t/yr</td>
<td>kg/d</td>
<td>Fraction</td>
</tr>
<tr>
<td>Air</td>
<td>0.00001</td>
<td>0.075</td>
<td>0.205</td>
<td>0.00001</td>
</tr>
<tr>
<td>Wastewater</td>
<td>0.003</td>
<td>22.50</td>
<td>61.62</td>
<td>0.0005</td>
</tr>
<tr>
<td>Soil</td>
<td>0.00001</td>
<td>0.075</td>
<td>0.205</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

However, monitoring data from the two main manufacturers have been submitted. Because the monitoring data generally covers the total emissions from the specific production site, one scenario in the risk assessment is based on the actual monitored values and identified “site specific” and where monitoring data are not present, values according to TGD are calculated. For confidentiality, average values are used.

A scenario on formulation was included in the risk assessment, because 10% of the production volume is formulated outside the two main manufacturers. The number of sites where formulation takes place is unknown and therefore, 10% of the total production volume of the phenoxyherbicide is estimated to be formulated at one other site (worst case) and release based on the default estimation procedures of the TGD. According to the manufacturers the content of PCOC as an impurity in the technical acids is below 1% (AHMarks 1997b, Nufarm 1997) and therefore 1% is used in the assessment for formulation (e.g. the footnote to the following table).

Table 3.2 Scenario on formulation

<table>
<thead>
<tr>
<th>Emission to:</th>
<th>Specific site *</th>
<th>Formulation site **</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>t/yr</td>
<td>kg/d</td>
</tr>
<tr>
<td>Air</td>
<td>0.2</td>
<td>0.6</td>
</tr>
<tr>
<td>Wastewater</td>
<td>2.8</td>
<td>7.8</td>
</tr>
<tr>
<td>Soil</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

* Based on monitoring data from specific site including production, processing and formulation (cf. text above)

**According to a worst case consideration (cf. above) the PCOC content in the phenoxy acids is max. 1%. Thus, a fraction of 0.001 of the total phenoxy acid production volume is used as the input volume to the release scenario
3.1.1.5 Agricultural use

The following recommended application rates are taken from Pesticide Manual (9th ed., 1991):

- MCPA: 0.3-2.25 kg/ha.
- MCPP: 1.8-2.4 kg/ha.
- MCPB: 1.7-3.4 kg/ha.

By considering these figures and the consumption volumes of these three different phenoxy herbicides “a realistic worst-case” application rate of these herbicides of 2 kg/ha can be estimated.

PCOC is found as impurity in phenoxy herbicides: 0.2 to 0.5% in MCPA (HSDB, Seibert 1986, Technical product), 0.645-0.67% in MCPA-dimethylammonium, and 0.05-0.06% in MCPA-sodium-salt. Only Hattula (1978) reports on 4% PCOC as impurity in MCPA from Kemira Co, Finland, while Marks (1995) mentions <1.0% impurity in a letter to the Danish EPA.

Considering these reported percentages of impurities in the herbicides and confidential information regarding the impurities in formulated phenoxy herbicides from the Danish EPA’s Pesticides Division (pers. comm.), a general percentage of PCOC in these herbicides is today estimated to be 0.5 % according to "the realistic worst-case concept".

3.1.2 Environmental fate

3.1.2.1 Degradation

PCOC is not expected to spontaneously hydrolyse (Lyman 1982) at environmental conditions as covalent bound substituents to a benzol ring normally is hydrolytically stable.

Formation of PCOC by photolysis of MCPA

PCOC is a photolysis degradation product of MCPA and a possible degradation product of MCPP. The major photodegradation product of MCPA was PCOC. The concentration was increasing with time (Clapes et al., 1986; Benoit-Guyod et al., 1986). PCOC is further photodegraded to o-cresol and methylhydroquinon (Clapes et al., 1986). Photodecomposition is suggested by Clapes & Vicente (1986) by substitution of the chlorine by a hydroxyl group or by dehalogenation to 2-methylbenzene and 1,4-dihydroxy-2-methylphenol.

Freiberg & Crosby (1988) suggest photodecomposition of PCOC to 5-chlorosalicylaldehyde and o-cresol or 2-methylphenol.

In sunlight (>300 nm) or under 40 W UV blacklight (Soderquist & Crosby, 1975; Crosby & Bowers, 1985), MCPA is degraded to PCOC, 4-chlorosalicyl-aldehyde, 4-chlorosalicylic acid and 2-methylphenol.

Crosby & Bowers (1985) found in a photoreactor using a spray solution of 200 mg/l MCPA that after 3.5 days of UV-irradiation 25% of the applied MCPA was degraded and PCOC increased to a maximum of about 12% of applied amount. Irradiation of droplets in an open dish gave noticeable odour of PCOC and chemical analysis confirmed PCOC to be the principal constituent. The low residues recovered may be the result of volatilization following the irradiation of MCPA/MCPP.
In a field exposure, PCOC was removed by volatilization as it was formed. After 31 days outdoor exposure to sunlight in California, PCOC was 1.3% of original MCPA (Crosby & Bowers, 1985).

In a simulated field study on photolysis using MCPA-dimethylammonium (MCPA-DMA), droplets containing 14.7 g MCPA/l were created at the tip of a 1.0 µl syringe and irradiated for 5 days. The evaporation residues contained 36% PCOC (Freiberg & Crosby, 1986) and in the gas phase 92% PCOC. The degradation was 41% to 44% of the applied MCPA. After the application of a MCPA-DMA (480 µg/40 µl = 12 g/l) solution on a glass plate and irradiated for 6 days, it was found that 49% of the residues was PCOC.

A 71-hour irradiation of a 100 ppm MCPA solution with simulated sunlight (λ >290 nm) resulted in the formation of 6% PCOC of the applied amount (Soderquist & Crosby, 1975).

Photolysis of PCOC
The UV-spectrum of PCOC shows maximum absorption near 280 nm with an absorption tail reaching 300 nm. The effect of sunlight irradiation on aqueous solution of PCOC (100 mg/l = 0.7 mM) was studied (Soderquist & Crosby, 1975). The PCOC solution was sealed in a flask to prevent volatilization. The exposure to "summer sunlight" (not further defined or specified) in California resulted in a stated photolytic half-life of 2.5 days (which from the presented graph rather seems to be 4 days) (Crosby & Bowers, 1985).

In the Appendix "Photolytic degradation of PCOC in surface water" (cf. Appendix A), the estimation of the photolytic degradation in EU surface waters was performed by relating the sunlight intensity in California (40°N) to the EU (50°N) and according to TGD the dimensions of the "standard environment for the EU" employed in the regional model for calculation of PECregional, water, i.e. a water depth of 3 m and a concentration of suspended solids of 15 mg/l. This results in a half-life for photolytic degradation in water of 301 days (annual mean) and 715 days (winter). Thus, the photolytic degradation of PCOC in surface waters of the EU is concluded to be negligible.

Photolysis in the atmosphere
Experimental data on photochemical oxidation by OH-radicals are not available, however, using the model of Atkinson (1986), the degradation rate \( k_{OH} = 1.3 \cdot 10^{-11} \text{ cm}^3/\text{moles} \cdot \text{sec} \) and the averaged OH-radical concentration \( 5 \cdot 10^5 \text{ molecules/cm}^3 \), an atmospheric half-life of 30 hours can be calculated (BASF 1991).

3.1.2.2 Aerobic biodegradation in water
The toxicity to microorganisms of chlorophenols was studied as growth inhibition test on agar plates using bacterial isolates from a polluted and an unpolluted stream. PCOC in a volume of 0.01 ml and in the concentration 250 mM was observed to inhibit the growth of microorganisms (Milner and Goulder, 1986).

PCOC is not readily degradable according to the modified MITI (I) test. In the MITI test 0% degradation was found at a test concentration of 100 mg/l (MITI 1992). As other studies at lower concentrations find biodegradation the result is expected to be a consequence of toxicity to bacteria at the high-test concentration. (e.g. bacterial toxicity was observed in the Salmonella typhimurium mutagenicity test at 500 µg PCOC/plate).
In a pilot plant study on activated sludge using sludge back-feeding and sludge from a municipal STP, the aerobic degradation on a mixture of chlorinated phenolics was studied (Buisson et al., 1988). The concentration of PCOC in the influent and effluent was in the order of 100 to 200 ng/l. The temperature was 17.5°C. PCOC was found to be removed by adsorption and biodegradation, but the relative importance of these processes was not investigated. The removal was found to depend on the sludge age. The reported removal and recovery percentages at sludge age of 4, 6, and 9 days were 10 and 88, -20 and 115, 36 and 63, respectively. A low removal of PCOC was thus observed under the study conditions. Biodegradation was probable under these conditions allowing adaptation of the microorganisms to PCOC but the data from the pilot plant study are not quantifiable in order to estimate a degradation rate in STPs. A biodegradation half-life of PCOC can therefore not be deduced from this study. The observed low removal of PCOC may be caused by transformation of other present chlorophenolics to PCOC. Another possible explanation may be that the other chlorophenolics inhibited the biodegradation of PCOC. This indicates that an assessment of the removal or degradation of chemical substances in studies performed on each individual substance may overestimate the actual biodegradation and thus, the total removal in STP where several substances are present at the same time.

In a shake flask die away test using DOC analysis at 15°C over 10 days, the degradation of PCOC in seawater from an initial concentration of 18 µg/l PCOC had a half-life of 3 days. In a mixture using 3.6 µg/l PCOC and wastewater, the degradation was reduced and the half-life increased to 45 days, which like the study described above either suggests an inhibitive effect on the microorganisms active in the degradation of PCOC caused by the other organic compounds present in the mixture (0.1-0.5 mg/l) or that these other compounds were transformed to PCOC (Lindgaard-Jørgensen, 1989).

3.1.2.3 Aerobic biodegradation in soil

Several degradation studies confirm PCOC to be the primarily metabolite of MCPA (e.g. Bollag et al., 1967; Paasivirta et al., 1983; Duah-Yentumi & Kuwatsuka, 1980; Gaunt & Evans, 1971; Gamar & Gant 1971 and Oh et al., 1995). In experimental studies, it is found that PCOC by biodegradation of MCPP and MCPA is a biodegradation product which will be further degraded (Smith, 1985). The reported concentration of PCOC which is formed in the degradation process is mainly 2 to 5% of the applied amount of the phenoxy herbicide. In one study (Duah-Yentumi & Kuwatsuka, 1980) in a fairly acid soil with pH 5.3, a maximum of 2% to 55% of the applied MCPA was reached after 25 days depending of the pH of the soil. “A realistic worst case” seems to be in the order of 5% of the applied pesticide dosage based on the majority of studies. In acid soils, however, considerably higher concentrations may occur.

The degradation half-lives of PCOC in aerobic soil are reported from few studies to be 14 to 21 days. For instance, Kinkannon and Lin (1985) found a half-life of 14 days in a bioreactor. The reactor was filled with sandy loam covered with DAF sludge (not defined, 33 g/kg soil) and mixed into the top 20 cm soil. The sludge was spiked with 304 ppm PCOC and the degradation followed over 82 days.

The degradation of PCOC in loamy sand and sandy loam after application of 10, 200 and 2000 ppm PCOC was found to have a half-life of 14 days (Sattar, 1981). The degradation rates in sandy clay were 0.0378 d⁻¹ at the application of 10 mg/kg soil and 0.029 d⁻¹ at 1000 mg/kg soil. The half-lives calculated as \( T_{1/2} = 0.693/k \) were equivalent to 18 days and 24 days (Sattar, 1989). An estimated half-life for degradation of PCOC in soil seems to be approximately 21 days (k = 0.033 d⁻¹) according to the "realistic worst-case" concept.
This latter biodegradation half-life in soil is based on considerably more experimental evidence than the above mentioned degradation half-life in surface waters. However, the half-lives for degradation in soil and surface water seem generally to be somewhat conflicting, because degradation half-lives are generally said to be of the same order of magnitude (Boethling et al., 1995) or twice as long in soil than in surface water for a substance with a $K_p < 100$ (TGD, 1996). Based on these considerations, a general approximate half-life for aerobic biodegradation in surface waters as well as in soil of 21 days seems justified according to "the realistic worst-case concept".

**Biodegradation half-life, conclusion**

A biodegradation half-life in soil and in surface water of about 21 days would according to the TGD (cf. Chapter 3 Tables 5 & 6) indicate that the substance could be regarded as readily biodegradable in soil and surface water. By using this and the TGD concerning degradation rates for biodegradation in STPs (cf. TGD, Chapter 3, Table 4), an estimated degradation rate constant $K_{\text{STP}}$ of 1 h$^{-1}$ (half-life of 0.7 h) can be estimated.

There is, however, quite some uncertainty as regards the biodegradability of PCOC, also because of the uncertainty as regards the extrapolation from degradation half-lives in soil and surface water to a degradation rate in STP. Based on the available information, it therefore seems to be justifiable to include for information the EUSES model estimations employing half-lives equivalent with inherent biodegradability.

### 3.1.2.4 Anaerobic degradation

Anaerobic degradation in sewage sludge treatment pilot plant has been studied by Buisson et al. (1986). PCOC was resistant to chemical or biologically mediated changes (no abiotic or biotic degradation) during anaerobic degradation at 10 µg/l. PCOC was also resistant to anaerobic degradation in soil. Buisson et al. (1986, 1990) and Kirk & Lester (1988) found no anaerobic degradation within 32 days.

### 3.1.2.5 Adsorption

In an experimental adsorption/desorption study from New Zealand on an acid soil (pH 5.2 and 3.6% organic carbon), a low adsorption coefficient, $K_d$ 0.008, was observed and the resultant $K_{\text{oc}}$ 0.22 was calculated (Bhamidimarri & Petrie, 1992). Buisson et al. (1986) observed some adsorption of PCOC to sludge from a municipal STP, but $K_d$ or $K_{\text{oc}}$ was not given and could not be estimated based on the available information. Based on an estimated $K_{\text{oc}}$ of 400, calculated from $K_{\text{ow}}$ (TGD: log $K_{\text{oc}}$ = 0.81 log $K_{\text{ow}}$ + 0.10), PCOC may exert a medium adsorption in soil and may partition to sediments and particulate materials in water. PCOC has a pKa of 9.71 (Weast et al. 1986) and the adsorption may be sensitive to pH. The adsorptive capability will increase and the leaching potential will decrease in increasingly alkaline soils due to a greater presence of the ionic form under alkaline conditions. Thus, in acid soils leaching may be expected.

Various QSAR estimations give a Koc of 14 to 700. Analogue substances like chlorobenzenes have Koc's of 83 to 389, chlorophenols approximately 51 and cresols 22 to 49. Due to the lack of an experimental Koc in non-acidic soils which are more representative for most soil types in the EU, the calculated value of $K_{\text{oc}} = 400$ l/kg has therefore been used in the risk assessment. The adsorption to sediments is unknown but based on an estimated $K_{\text{oc}}$ of 400, PCOC is expected to adsorb to sediments and particulate materials in the water column depending of the pH.
3.1.2.6 Bioaccumulation

A log $K_{ow}$ of about 3 indicates that bioaccumulation may occur.

However in a bioaccumulation test on orange red killifish, *Oryzias latipes*, during 42 days at 25°C after OECD 305C and a concentration of 2 µg/l, a BCF of 6.4 to 14 was found, and at 20 µg/l a BCF of 8.2 to 28 was found. The fish average lipid content was 4.9% (MITI 1992).

In a 28-day study on rainbow trout, *Salmo trutta*, Hattula (1979) found BCF 6.9 to 4.3 at concentrations 0.5 to 1.5 ppm (average 6.6 at 0.5 ppm, 4.7 at 1.0 ppm and 4.3 at 1.5 ppm). In a short-term study (24 hours) the BCF was 8.2. Based on the log $K_{ow}$ and the time to reach equilibrium in a BCF fish study: $t_{95} = \frac{3.0}{k_2} = \frac{3.0}{(-0.414 \log K_{ow} + 1.47)}$ (cf. OECD Test Guideline 305, 1996), an estimated $t_{95} = 16$ hours is obtained, i.e. steady state in the above mentioned bioconcentration studies can be assumed and the measured BCF (fish) values therefore regarded as reliable.

Based on the above mentioned data, a "realistic worst-case" BCF for fish is 30.

3.1.2.7 Environmental distribution

Volatilisation

PCOC may form rapidly from sun irradiated water solutions or irradiated deposits or volatilize from water. In a study (Seiber et al., 1986) around flooded rice fields in California treated with MCPA at 0.87 kg active ingredient/ha, it was observed that air samples contained more PCOC than MCPA. Several kilometres from the fields 30 ng/m$^3$ PCOC was found. The highest normalised flux was found on the day of spraying and decreased with time. The daily averaged volatilisation flux for day 0, 1, 2 and 3 was 1.27, 0.43, 0.27 and 0.24 ng/cm$^2$/h.

The volatilisation was evaluated by Henry's Constant, $H = \frac{VP}{SOL}$ (VP 3.2 Pa, SOL 4 g/l at 25°C) by Seibert et al. (1986). However, using VP 26.66 Pa and SOL 2.3 g/l resulted in $H = 1.65$ Pa m$^3$/mol. All results indicate that volatilisation may be expected from water and wet soil.

Performing a Mackay fugacity level I calculation (Mackay & Paterson, 1990) based on physiochemical properties results in an environmental compartment distribution of 33% in air, 56% in water, 6% in soil and 5% in sediment.

3.1.2.8 Summary

The environmental exposure assessments considered combine the relevant exposure scenarios for the substance and apply recommended assessment methods for deriving PEC local and regional, i.e. the applied environmental exposure scenarios are:

Scenario                                      Compartment of primary release                
   a) Production of PCOC, processing and formulation of phenox herbicides at the same site (monitoring data) surface water  
   b) Formulation at another site (generic) surface water  
   c) Agricultural use of herbicides (generic) agricultural soil
Performing a Mackay fugacity level I calculation (Mackay & Paterson, 1990) results in an environmental compartment distribution of 33% in air, 56% in water, 6% in soil and 5% in sediment.

4-Chloro-2-methylphenol (PCOC) may be released to the environment in wastewater and air effluent from its production and its use as a chemical intermediate in the synthesis of phenoxy herbicides (MCPA, MCPB, MCPP). Environmental release is also present via degradation at points of herbicide application and sites of subsequent environmental transport.

Considering the reported percentages of impurities in the herbicides, a general impurity percentages in these herbicides according to the "realistic worst-case" approach of 0.5 % is used.

Soil exposure to PCOC from degradation and impurities of the herbicides is estimated to be maximum 5% of the herbicide application rate of 2 kg/ha. It is noted that there will be a prolonged exposure of PCOC after the application of herbicides, which are continuously broken down via PCOC.

If released to the atmosphere, PCOC may be physically removed by settling or washing out in precipitation. Model calculations indicate that the photochemical degradation in the atmosphere is rapid with an estimated half-life rate of 1.25 days whereas photodegradation in surface water is negligible.

Available data suggest that PCOC is biodegradable in soil and surface waters under aerobic conditions with an approximate half-life of 21 days (k = 0.03 d\(^{-1}\)) and resistant to anaerobic degradation. By using the estimation tables of the TGD concerning degradation rates for biodegradation in STPs, a degradation rate constant K\(_{\text{STP}}\) of 1 h\(^{-1}\) can be estimated. The available experimental data on biodegradation are somewhat conflicting and the estimated half-lives are quite uncertain.

The adsorption coefficient in soil (K\(_{\infty}\)) is calculated to be 400 l/kg. Volatilisation is expected to be slow. Henry’s Law constant is calculated to be 1.65 Pa m\(^3\)/mol.

The hydrolysis of PCOC in water is estimated to be negligible.

A "realistic worst-case" BCF for fish is 30.

### 3.1.3 Aquatic compartment

#### 3.1.3.1 Measured data

Historical data exist from a Danish production site of phenoxyherbicides, which is no longer operating. The effluent from a municipal wastewater treatment plant receiving wastewater from the plant had PCOC as the dominant chlorophenol. The effluent from the manufacturer (600 m\(^3\)/d) contained 2,400 µg/l PCOC (1.4 kg PCOC/d). The effluent was diluted before entering a biological STP to 3% of the total wastewater loading and the PCOC concentration in the influent was thus reduced to 72 µg/l. The effluent had a mean concentration of PCOC of 4 µg/l (64 g/d), resulting in a total removal of 94% of the inflow concentration (Folke & Lund, 1983, Folke 1984). These values can, however, not be used in this risk assessment because no information about the representativeness of this single measured value is available. Furthermore, no information about the concentration of PCOC in the wastewater nor of the dilution in the receiving river was present.
Monitoring results from the effluent from the two main manufacturing sites ranged from <1 mg/l to 45 mg PCOC/l in the raw effluent leaving the production sites including production, processing and formulation. The average concentration was 34 mg/l during a one year measuring period (1996 to 1997) of one manufacturing site but during a one-week period from both manufacturers in October 1997 values around 18 mg/l was measured.

The raw effluents from the manufacturing sites were diluted with municipal STP influent before STP treatment. Dry weather flow at the municipal STPs varied from 130,000 m³/day to 200,000 m³/day.

The measured concentrations in STP effluent varied from below detection level to 3.6 µg/l at one of the two main production sites. In the two STP effluents receiving wastewater from the main production sites, the same variation in measured concentrations was observed. Because the detection limit is approximately 3.6 µg/l, the level before dilution in the receiving rivers is estimated to be <3.6 µg/l (A.H. Marks and Nufarm, October 1997).

Of more recent surface water monitoring data, a single sample from approximately 1.3 km downstream from a municipal STP discharge was sampled and analysed. The municipal STP received wastewater from a PCOC production site. PCOC could not be measured above a detection limit of 0.2 µg/l, and therefore, it was concluded that PCOC concentration was below 0.2 µg/l. However, because this value cannot be validated as regards representativeness according to the TGD, the figure cannot be used directly in the assessment.

In a monitoring programme during September 1997, 2 km downstream in the receiving river during the production period, the measured concentrations ranged from <0.2 to 3.0 µg/l. The instrument detection limit was observed to be 0.2 µg/l. The limit of detection (LOD) was found to be 3.6 µg/l and the limit of quantification (LOQ) 12 µg/l. During a factory “shut down” period, no PCOC was detected in the river. The analytical method was modified which reduced the LOD to 0.7 µg/l and LOQ to 2.4 µg/l. It was concluded considering the measure error that the concentration in surface water was less than 3.6 µg/l.

After the application of MCPA to a rice field in California, PCOC was measured in the water during 4 days. The concentrations found were relatively constant confirming the continuous transformation of MCPA to PCOC and the intermediate nature of PCOC. PCOC concentrations of 1.4, 1.3, 1.2 and 1.3 µg/l were measured on day 0, 1, 2, 3 after application, respectively (Seiber et al., 1986).

3.1.3.2 Aquatic exposure estimations

The local emissions and concentrations rely on the monitoring results from the two main manufacturers. For comparison, the local emission from production, processing and formulation is also estimated according to the TGD (cf. Appendix C for TGD calculation methods and Appendix D for the results of the model calculations employing EUSES v.1.0).

The observed removal in a Danish STP was 94% (including adsorption, evaporation and degradation) (Folke 1984). However, it is not known how representative this percentage of removal is for biological STPs of the EU in general, but the observed removal figure is in general accordance with calculated removal figures employing EUSES, i.e. the SIMPLETREAT model using the environmental fate data mentioned in section 3.1 (cf. also appendices with print out of results from EUSES model calculations). Thus in the estimations, the TGD based removal value of 88% (cf. TGD erratum, 19 February, 1997), is used.
The results from the manufacturer monitoring programmes are inconclusive regarding the removal in STP as the measured values are approximately at the detection level after the dilution into the municipal sewage flow (130,000 to 200,000 m$^3$/day) (Marks, 1997b; Nufarm UK 1997b). However, a rough estimate of the removal in the municipal STP can be calculated. The average emission to STP wastewater is 7.8 kg/day (cf. below). The concentration in the STP effluent was measured below 3.6 µg/l and using the highest average dry weather STP flow rate of 200,000 m$^3$/day then $3.6 \times 10^{-6} \text{ g/l} \times 200 \times 10^6 \text{ l/d} = 720 \text{ g/day}$ leaves the STP (worst case). The estimated removal would then be: $1-(720/7800) = 91\%$. The result supports the above made estimation.

PEC$_{\text{regional-water}}$

As regards the calculation of the PEC$_{\text{regional-water}}$; cf. the appendices with the results of model calculations employing EUSES v.1.0.

When PCOC is released from production, processing and formulation of herbicides:

PEC$_{\text{regional-water}}$: $1.69 \times 10^{-4} \text{ mg/l}$.

PEC$_{\text{regional-water}}$ is not estimated in the pesticide scenario.

3.1.3.3 Calculations of PEC$_{\text{local-water}}$

3.1.3.3.1 PCOC released from production, processing and formulation

The site-specific emissions and concentrations are based on average monitoring results from the two main manufacturers. The wastewater effluents from the production sites including production of PCOC, processing into phenoxy acids and formulation to phenoxy herbicides together with water from air scrubber etc. are collected and chemically treated in a main effluent treatment plant on-site prior to disposal via tanker or drain to the effluent system. The measured concentrations in the raw effluent are shown in the table below.

<table>
<thead>
<tr>
<th>PCOC (mg/l)</th>
<th>Minimum</th>
<th>Average</th>
<th>95% percentile</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific site 1</td>
<td>&lt;1</td>
<td>17</td>
<td>29</td>
<td>45</td>
</tr>
<tr>
<td>Specific site 2</td>
<td>16</td>
<td>18</td>
<td>34</td>
<td>45</td>
</tr>
</tbody>
</table>

At the municipal STP the raw effluent is mixed with the incoming domestic sewage water and passed to biological treatment beds before being discharged to the river. The STP dry weather flows varied from 130,000 to 200,000 m$^3$/day.

During 1 week in October 1997, the concentration in STP influent was measured to vary between 0.15 to 2.0 µg/l (n=6, detection limit (DL) 0.1 µg/l) (Nupharm, 1997b).

The concentration in one of the specific STP during 1 week in October 1997, the STP effluent varied 0.13 to 3.1 µg/l (n=6, DL 0.1 µg/l). During one month, September 1997 at the other STP, the measured concentration of PCOC in the STP effluent was <0.2 to 3.3 µg/l (n=13, instrument detection limit 0.2 µg/l).

In the risk assessment on specific sites where production, processing and formulation takes place, the average measured values where available are used (Marks, 1997; Nufarm, 1997,
1997b). For illustrative purposes Appendix C shows a comparison of environmental exposure estimates from main manufacturing processes: production, processing and formulation, respectively, calculated according to TGD with the measured values.

The local emission during episode to the aquatic compartment, \( E_{\text{local-water}} \), is calculated in EUSES by using the regional values in section 3.1.1.4 multiplied with the estimated release fractions. The release fraction for production was set to 0.5 due to two main manufacturers, 0.5 for processing as PCOC is not used elsewhere and 0.3 (0.5 \( \cdot \) 60%) for formulation as this part of the life cycle was performed at another site. The site-specific STP flow rate of \( 1.3 \cdot 10^5 \text{ m}^3/\text{d} \) is included, because this is the lowest flow of the STPs receiving wastewater from the two known manufacturers.

The release estimations from the formulation performed outside the specific sites are calculated according to TGD using 10% of total production and a PCOC content of the formulation of 1% (worst case). The default local wastewater volume is according to TGD: 2,000 m\(^3\)/d, and number of days of emission is according to TGD: 300.

The average local on-site monitoring results and the estimated results on a generic formulation site (10% of total production volume) are shown below.

### Table 3.4  Concentration in wastewater before treatment in municipal STP

<table>
<thead>
<tr>
<th>Life cycle stage</th>
<th>Based on</th>
<th>Emission (t/y)</th>
<th>Emission (kg/d)</th>
<th>( C_{\text{site effluent}}^b ) (mg/l)</th>
<th>( C_{\text{STP influent}}^c ) (µg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific site</td>
<td>monitoring</td>
<td>2.5(^a)</td>
<td>7.8(^a)</td>
<td>29 &amp; 34</td>
<td>43 &amp; 52(^c)</td>
</tr>
<tr>
<td>Formulation (10% of total)</td>
<td>TGD defaults</td>
<td>0.063</td>
<td>0.21</td>
<td>-</td>
<td>110</td>
</tr>
</tbody>
</table>

\(^a\) Estimated by using measured effluent concentration at each of the two sites times the respective volumes of wastewater at the sites (At these sites production, processing and formulation occurred (cf. the text))

\(^b\) Measured concentrations from each of the two sites' wastewater (from production, processing and formulation) before dilution at STP, 95% percentile

\(^c\) Average concentrations, estimated by using the average measured effluent concentration times the actual dry weather STP-dilution at each site. Measured concentration at one of the sites was one order of magnitude below the estimated concentrations but not much above the detection limit

For the site-specific scenario and in accordance with the principle of “realistic worst case”, a concentration (3.6 µg/l) from the upper range of the monitoring data of the STP effluents has been used (AHMarks, 1997b; Nufarm 1997b). This concentration is in general accordance with the estimated “realistic worst case” concentrations in the STP effluents without any dilution in the receiving river. Measured concentrations approx. 2 km downstream one of the sites were fluctuating between a few µg/l and one order of magnitude below.

### Table 3.5  Local concentration during emission episode, \( \text{PEC}_{\text{local, river water}} \)

<table>
<thead>
<tr>
<th></th>
<th>( C_{\text{local, effluent}} ) (mg/l)</th>
<th>( C_{\text{local, river water}} ) (mg/l)</th>
<th>( \text{PEC}_{\text{local, river water}} ) (mg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site specific (measurements)</td>
<td>0.0036</td>
<td>0.0036</td>
<td>0.0038</td>
</tr>
<tr>
<td>Formulation (estimations)</td>
<td>0.0126</td>
<td>0.0013</td>
<td>0.0014</td>
</tr>
</tbody>
</table>

\(^*\) Including \( \text{PEC}_{\text{regional, river water}} \) 0.000169 mg/l
The annual average concentration is estimated below to be:

<table>
<thead>
<tr>
<th>Table 3.6</th>
<th>Annual average concentration, PEC_{local, river water, ann}</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PEC_{local, water, ann}^{a} (mg/l)</td>
</tr>
<tr>
<td>Specific site^{b}</td>
<td>0.0037</td>
</tr>
<tr>
<td>Formulation (TGD default)</td>
<td>0.0012</td>
</tr>
</tbody>
</table>

^{a}Including PEC_{regional, water}
^{b}Based on monitoring data

Calculation of PEC_{local} for sediment

The local concentration in sediments during emission episode is calculated according to TGD (cf. Appendix C).

<table>
<thead>
<tr>
<th>Table 3.7</th>
<th>Predicted environmental local concentration in sediments (PEC_{local, sed})</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PEC_{local, water} (mg/l)</td>
</tr>
<tr>
<td>Specific site^{*}</td>
<td>0.0038</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.0014</td>
</tr>
</tbody>
</table>

^{*}Estimated using monitoring data

Calculation of STP concentration for evaluation of inhibition of microorganisms

For the risk characterisation of PCOC regarding microorganisms in STP, ideally the concentration in the aeration tank should be used. Because this value was not available, the dissolved concentration of PCOC is assumed to be equal to the effluent concentration according to the TGD:

PEC_{STP, microorganisms} = C_{effluent}

<table>
<thead>
<tr>
<th>Table 3.8</th>
<th>PEC_{STP, microorganisms}</th>
</tr>
</thead>
<tbody>
<tr>
<td>Life cycle stage</td>
<td>PEC_{STP, microorganisms} (mg/l)</td>
</tr>
<tr>
<td>Specific site^{*}</td>
<td>0.004</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.013</td>
</tr>
</tbody>
</table>

^{*}Estimated using monitoring data

### 3.1.3.3.2 Agricultural use of phenoxy herbicides

It is noted that the TGD does not include exposure scenarios for pesticides.

An initial exposure assessment for surface waters according to Annex VI of dir. 91/414/EEC, i.e. Directive 94/43/EC, is presented here. It is noted that the latter directive was annulled by a EU Court decision in 1996.

The aquatic environment close to areas where the herbicides are applied may receive direct exposure by overspraying (ponds, ditches, streams). It is assumed that direct application to a water body of 1 ha, 1/3 meter deep and density 1.0 takes place. The above described application would result in $10,000 \times 0.333 = 3,333 \times 10^3$ l. Using the application rate 2 kg/ha of e.g. MCPA,
and assuming PCOC as 0.5% impurity and 4.5% as degradation product, the initial concentration would be: 100/3,333 = 0.03 mg/l. It is noted that the duration of exposure will be prolonged due to continuous degradation of MCPA to PCOC.

The concentration in surface water based on indirect exposure via spray drift from a nearby agricultural application (2 kg phenoxy herbicide/ha, resulting in 100 g PCOC/ha, 3% spray drift) is estimated to be 0.0009 mg/l.

EUSES ver 1.0 (employing the country code specifications for the EU according to the TGD) (RIVM, 1994) which includes a pesticide scenario, calculates the initial concentration in a ditch to 0.0008 mg/l.

The PEC_{local, water} is calculated based on the yearly volume of PCOC which may be released to and within the environment whereas the estimations of PEC by direct application of the herbicides to water bodies are based on the steady state exposure where account of the continuous degradation from the herbicides is taken.

It is noted that surface waters besides exposure from STP receive contributions from possible incidences of direct application, spray drift, run off and atmospheric deposition.

### 3.1.4 Atmosphere

PCOC may enter the atmosphere from the production and processing. The use of MCPA, MCPB, and MCPP as agricultural herbicides results in the release of PCOC from evaporation and degradation to the atmosphere.

#### 3.1.4.1 Measured data

Air from storage, handling and use of PCOC at the production sites are connected to a scrubber system to reduce emissions. Monitoring results of the process contribution from PCOC production combined at one of the manufacturing sites is estimated to be <0.027 mg/m$^3$ based on the measured release 6.6$ \cdot 10^{-3}$ g/sec (0.57 kg/day). The concentration in air was below the detection level at production site boundary. At the other manufacturing site the maximum quantities released from the plant main fume scrubber was 5 mg/m$^3$ or 0.034 kg/hour (0.8 kg/d)

**Measurements in air at local STP and at 112 m from STP**

Based on the results a dispersion programme using a Gaussian dispersion model calculated short-term concentrations directly downwind (wind speed 3.76 m) at approximately 100 m. The maximum ground level concentration ranged 27.0$ \cdot 10^{-8}$ to 10.4$ \cdot 10^{-5}$ mg/m$^3$ with a time weighted average of 6.8$ \cdot 10^{-4}$ mg/m$^3$.

#### 3.1.4.2 Atmospheric exposure estimations

**PCOC used in production, processing and formulation**

The concentration in air is estimated according to TGD at a distance of 100 meters from the point source. In the calculation for PEC_{local} for air, both emissions from a point source as well as the emission from a STP are taken into account. The maximum from the two concentrations (direct and via STP) is used as the PEC_{local}. 

The annual average predicted environmental concentration in air, PEC_{local, air, ann}, is the local annual average added to the regional concentration in air.

PEC_{regional, air} = 7.8 \times 10^{-7} \text{ mg/m}^3 \text{ (EUSES)}

<table>
<thead>
<tr>
<th>Emission</th>
<th>E_{local, air}</th>
<th>E_{stp, air}</th>
<th>C_{local, air}</th>
<th>C_{local, air, ann}</th>
<th>PEC_{local, air, ann}</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific site*</td>
<td>0.57</td>
<td>0.047</td>
<td>0.000158</td>
<td>0.000130</td>
<td>0.000131</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.021</td>
<td>0.07</td>
<td>0.0014</td>
<td>0.000020</td>
<td>0.000016</td>
</tr>
</tbody>
</table>

*Estimated using monitoring data on total release to air and wastewater and TGD estimation methods

### Deposition from air

The deposition from air includes the emissions from the two sources (direct and STP). The total deposition from air during emission episode is calculated as:

\[
\text{DEP}_{\text{total}} = (E_{\text{local, air}} + E_{\text{stp, air}}) \times [F_{\text{ass}} \cdot \text{DEP}_{\text{std, aer}} + (1-F_{\text{ass}}) \cdot \text{DEP}_{\text{std, gas}}]
\]

The annual average total deposition flux is defined as:

\[
\text{DEP}_{\text{total, ann}} = \text{DEP}_{\text{total}} \times \frac{300}{365}
\]

| Site specific* | 0.57 | 0.047 | 0.00025 | 0.00020 |
| Formulation | 0.07 | 0.0014 | 0.00003 | 0.00002 |

*Estimations based on monitoring data

### 3.1.5 Terrestrial compartment

#### 3.1.5.1 Measured data

During monitoring groundwater in Denmark and among 10 chlorophenols searched for, PCOC was found in 5 measurements out of 825 (5 filters in two wells) in the concentration 0.040 µg/l to 8.79 µg/l (Groundwater monitoring, DGU, 1994). The detailed information below is from W.Brüsch (pers.comm 1995):

- Copenhagen county, 27 meter below terrain: 0.1 µg/l
- Copenhagen county, same as above 1 year later: 0.04 µg/l
- North Jutland county, 15 meter below terrain: 8.7 µg/l
- North Jutland county, 26 meter below terrain: 0.84 µg/l
- North Jutland county, 18 meter below terrain: 0.32 µg/l
A single finding of 0.13 µg/l is reported from 1992. No phenoxyacetic acids were found in the same measurements (Groundwater monitoring, DGU, 1994).

In a Danish study on leaching of pesticides from arable land, samples of stream water and soil water in the depth 80 cm below soil surface 1 m away from the stream bank were taken every 2nd week during 2 years. PCOC was found in the soil water at one location with clay soil at 0.03 to 0.08 µg/l in 6 out of 48 samples and in the stream water at 0.01 to 0.12 µg/l in 3 out of 47 samples. PCOC was observed to be present from April to October (Spliid & Mogensen, 1995).

The findings confirm that PCOC is present in the environment but being an intermediate in a degradation sequence with a relatively fast degradation rate, the amount is expected to be temporarily under aerobic conditions. Under anaerobic conditions, PCOC is considered persistent.

The evaporation from MCPA and MCPP and photodegradation of MCPA (and MCPP though no records) to PCOC is also a route to be considered but the contribution is not quantifiable.

Thus PCOC is found to be a degradation product (from MCPA and MCPP), which is further, degraded or transformed. The results from MCPA studies indicate 3% to 15% of applied amount found as PCOC after 20 to 25 days after application. In studies on MCPP, 2 to 3% of the applied amount of MCPP (1 mg MCPP/kg) was recovered after 20 days (Smith 1985). More recent studies on aerobic degradation indicated that PCOC was an intermediary degradation product from MCPA never present at more than 5% of the applied amount (Matt 1990 cited by AH Marks 1995). In a study on the degradation of MCPP, it was observed that one transient metabolite reached a maximum of 3.5% of applied. The metabolite was not identified (Saxena, 1988 cited by AH Marks, 1995).

### 3.1.5.2 Terrestrial exposure estimations

**Regional concentration in soil, PEC\textsubscript{regional, soil}**

As regards the calculation of the PEC\textsubscript{regional, soil}, cf. the appendices with the results of model calculations employing EUSES ver. 1.0.

**PCOC released from production, processing and formulation**

<table>
<thead>
<tr>
<th>Type of Soil</th>
<th>Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEC regional in agricultural soil</td>
<td>$2.7 \cdot 10^{-5}$ mg/kg ww</td>
</tr>
<tr>
<td>PEC regional in natural soil</td>
<td>$8.6 \cdot 10^{-7}$ mg/kg ww</td>
</tr>
</tbody>
</table>

**PCOC released from agricultural use**

PEC\textsubscript{regional, soil} (EU): not calculated

**Local concentrations (PEC\textsubscript{local, soil}) from production, processing and formulation**

The estimated concentration of PCOC in soil may be a result of atmospheric deposition and sludge application.

**Deposition from air**

The contribution from atmospheric deposition, D\textsubscript{air}, is derived by converting the total deposition flux DEP\textsubscript{total,ann} into concentrations (cf. Appendix C).

The contribution from wet and dry deposition from the atmosphere is not quantifiable but in the section on air, a calculation of total deposition has been estimated.
Sludge application

Sludge from both main production sites are incinerated or landfilled. Sludge from the municipal STPs treating the wastewater is incinerated at one STP and used for field application in the other STP. Therefore the estimations below have been performed to cover the sludge application scenario according to the realistic worst-case concept.

Only few data on measured concentrations of PCOC in sludge exist e.g. Buisson et al. (1984) measured 0.09 mg/kg dry weight in sludge from a municipal STP in the UK. During a week in October 1997, the sludge from the STP receiving effluent from a production site and where the sludge is used in field application has been analysed. The measured PCOC concentration varied between <1 to 2 µg/kg sludge (n=6, DL 1 µg/kg).

The concentration in soil will be high just after sludge application and reduced in time due to removal processes (degradation, volatilisation, leaching, etc.). Therefore, the concentration is averaged over a certain time period for different endpoints according to the TGD:

<table>
<thead>
<tr>
<th>Depth of soil (m)</th>
<th>Averaging time (days)</th>
<th>Sludge application (kg/m²/year)</th>
<th>Endpoint</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEC_{local, soil}</td>
<td>0.20</td>
<td>30</td>
<td>0.5</td>
</tr>
<tr>
<td>PEC_{local, agr. soil}</td>
<td>0.20</td>
<td>180</td>
<td>0.5</td>
</tr>
<tr>
<td>PEC_{local, grassland}</td>
<td>0.10</td>
<td>180</td>
<td>0.1</td>
</tr>
</tbody>
</table>

The concentration in dry sewage sludge, C_{sludge}, is based on monitoring data for the specific site. For the formulation site, C_{sludge} is estimated from the emission rate to water, the fraction of emission sorbed to sludge and the rate of sewage sludge production according to TGD.

The initial concentration in sludge and in soil after the first sludge application is tabulated below.

<table>
<thead>
<tr>
<th></th>
<th>C_{sludge, local, soil} (mg/kg)</th>
<th>C_{sludge, local, agr. soil} (mg/kg)</th>
<th>C_{sludge, local, grassland} (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site specific*</td>
<td>0.002</td>
<td>0.000003</td>
<td>0.000001</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.161</td>
<td>0.000237</td>
<td>0.000095</td>
</tr>
</tbody>
</table>

*Estimated using monitoring data from STP (C_{sludge})

The concentration in soil after sludge application for 10 consecutive years is assumed to be a “realistic worst case”. However based on the removal rates and the calculations in the Appendix C (calculation of PEC_{local, soil}), the estimated concentrations after 10 years of consecutive applications remain the same as after just one year.

Removal rates

The first order constant for removal from top soil, k, is derived by adding the biodegradation (k_{bio, soil}), volatilisation (k_{volat}) and leaching rate constant (k_{leach}) calculated for PCOC according to TGD.
Table 3.13 Removal rate constants

<table>
<thead>
<tr>
<th>Soil depth</th>
<th>( k_{\text{bio, soil}} ) (d(^{-1}))</th>
<th>( k_{\text{volat}} ) (d(^{-1}))</th>
<th>( k_{\text{leach}} ) (d(^{-1}))</th>
<th>( k ) (d(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.2 m</td>
<td>0.033007</td>
<td>0.000156</td>
<td>0.000197</td>
<td>0.033353</td>
</tr>
<tr>
<td>0.1 m</td>
<td>0.033007</td>
<td>0.000312</td>
<td>0.000393</td>
<td>0.033705</td>
</tr>
<tr>
<td>0.05 m</td>
<td>0.033007</td>
<td>0.000624</td>
<td>0.000785</td>
<td>0.034416</td>
</tr>
</tbody>
</table>

Local concentration in soil

The sum of both the concentration due to deposition and to the initial concentration from sludge application (cf. above) is used to estimate the local concentration in soil and averaged by the time periods in the table above. The results are tabulated below for the different endpoints.

For natural soil (soil depth 0.05 m), only the deposition from air is included because no sludge application is assumed.

\[ \text{PEC}_{\text{local, soil}} = \text{PEC}_{\text{local, soil}} + \text{PEC}_{\text{regional, natural soil}} \]

\( \text{PEC}_{\text{local, soil}} \), however, in most situations are equal to \( \text{C}_{\text{local, soil}} \) because the regional contribution is estimated to be lower than the estimated values. Except for the site-specific estimations, which is estimated to be lower than the regional contribution.

Table 3.14 Local concentration in soil

<table>
<thead>
<tr>
<th></th>
<th>( \text{PEC}_{\text{local, soil}} ) (mg/kg)</th>
<th>( \text{PEC}_{\text{local, agr. soil}} ) (mg/kg)</th>
<th>( \text{PEC}_{\text{local, grassland}} ) (mg/kg)</th>
<th>( \text{PEC}_{\text{local, natural soil}} ) (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific site *</td>
<td>0.0000002</td>
<td>0.0000014</td>
<td>0.0000011</td>
<td>0.00000088</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.000152</td>
<td>0.0000415</td>
<td>0.0000197</td>
<td>0.00000319</td>
</tr>
</tbody>
</table>

*Estimated using monitoring data on Csludge

Concentration in pore water

The concentration in soil porewater is calculated in the Appendix C.

Table 3.15 Local concentration in soil porewater

<table>
<thead>
<tr>
<th></th>
<th>( \text{C}_{\text{local, soil, porewater}} ) (mg/l)</th>
<th>( \text{C}_{\text{local, agr. soil, porewater}} ) (mg/l)</th>
<th>( \text{C}_{\text{local, grassland, porewater}} ) (mg/l)</th>
<th>( \text{C}_{\text{local, natural soil, porewater}} ) (mg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site specific*</td>
<td>0.00000003</td>
<td>0.000000020</td>
<td>0.000000015</td>
<td>0.00000012</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.00000212</td>
<td>0.00000577</td>
<td>0.00000274</td>
<td>0.0000044</td>
</tr>
</tbody>
</table>

*Estimated using monitoring data

Concentration in groundwater

The concentration in groundwater calculated for indirect exposure to humans through drinking water is initially assessed by the concentration in pore water in agricultural soil for a “realistic worst-case” estimation according to TGD.

\[ \text{PEC}_{\text{local, grw}} = \text{PEC}_{\text{local, agr. soil, porew}} \]

Sludge from the factory STP is either incinerated or disposed on landfill according to information from the main manufacturers (Marks, 1997; Nufarm, 1997). The basis for the
estimation of $\text{PEC}_{\text{local,grw}}$ is therefore the concentration in the influent to the municipal STP where sludge may be expected to be used on soil which is the situation at one municipal STP. If sludge is not applied, atmospheric deposition alone is considered relevant, except when phenoxy herbicides are used in agriculture. The atmospheric contribution is estimated to be insignificant.

<table>
<thead>
<tr>
<th>Table 3.16 Local concentration in groundwater</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PEC</strong>&lt;sub&gt;local, grw&lt;/sub&gt; (mg/l)</td>
</tr>
<tr>
<td>Site specific*</td>
</tr>
<tr>
<td>Formulation</td>
</tr>
</tbody>
</table>

*Estimated using monitoring data

Agricultural use of phenoxy herbicides

The concentration in soil calculated according to TGD as an average concentration in agricultural soil using soil depth 0.2 meter<sup>5</sup> and density 1700 kg/m<sup>3</sup> (wet soil) = 3400 ton soil/ha:

MCPA: 5% of 2 kg/ha = 100 g PCOC/ha = 0.029 mg PCOC/kg soil

MCPP: 3.5% of 2 kg/ha = 70 g PCOC/ha = 0.021 mg PCOC/kg soil

$\text{PEC}_{\text{soil,porewater}} = \text{PEC}_{\text{soil}} \cdot \text{RHO}_{\text{soil}} / (K_{\text{soil-water}} \cdot 1000)$ (mg PCOC/1 pore water)

$= 0.029 \cdot 1700 / (12.2 \cdot 1000)$

$= 0.004 \text{ mg PCOC/l porewater}$

**Conclusion**

The $\text{PEC}_{\text{soil}}$ is calculated to be at maximum 0.029 mg PCOC/kg agricultural soil using the TGD set-up (in this “pesticide scenario”).

By employing USES version 1.0 (“Pesticide scenario” by employing the country code file for the EU according to the TGD; RIVM et al., 1994), the initial concentration for PCOC in soil is 0.1143 mg PCOC/kg soil and the concentration over 28 days is 0.075 mg PCOC/kg soil.

For agricultural soils receiving atmospheric deposition, sludge application, and pesticides, the maximum $\text{PEC}_{\text{total, local, soil}}$ is estimated to be 0.03 mg PCOC/kg soil. The contribution from atmospheric and sludge application is negligible.

**3.1.6 Non compartment specific exposure relevant to the food chain**

In a specific monitoring study (Paasivirta et al., 1983), the content of PCOC in plants from garden near a railroad site in Northern Finland were measured two weeks after MCPA spraying and the following concentrations were observed: 0.2 ppb PCOC (fresh weight) in potatoes, 2.9 ppb in carrots, 52.9 ppb in green salad and 593 ppb in onions.

---

<sup>5</sup> In the TGD, a default soil depth of 0.2 m for the sludge application scenario is used. However, according to assessment schemes for agricultural pesticides such as those of EPPO, Germany, the Netherlands and Denmark, a default soil depth for pesticides which are applied as sprays, the soil default depth is four to eight times less. The present assessment has been conducted employing the soil depth recommended by the TGD. If the assessment was performed using the above mentioned soil depth, the $\text{PEC}_{\text{soil}}$ would increase accordingly (i.e. four to eight times).
3.2 EFFECTS ASSESSMENT: HAZARD IDENTIFICATION AND DOSE (CONCENTRATION) - RESPONSE (EFFECT) ASSESSMENT

3.2.1 Aquatic compartment

The following dose (concentration) - response (effect) results have been observed:

**Table 3.17** Fish, short term

<table>
<thead>
<tr>
<th>Organism</th>
<th>Toxicity</th>
<th>Method</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Lepomis macrochirus</em></td>
<td>LC₅₀ (96h, static)</td>
<td>2.3 mg/l</td>
<td>USEPA 1975, nominal conc.*</td>
</tr>
<tr>
<td></td>
<td>LC₅₀ (24 h, static)</td>
<td>3.8 mg/l</td>
<td></td>
</tr>
<tr>
<td><em>Oryzias latipes</em></td>
<td>LC₅₀ (96h, static)</td>
<td>6.3 mg/l</td>
<td>JIS K0102-86-71, not specified**</td>
</tr>
<tr>
<td><em>Brachydanio rerio</em></td>
<td>LC₅₀ (96h, static)</td>
<td>3 to 6 mg/l</td>
<td>OECD TG 203, not specified*</td>
</tr>
<tr>
<td><em>Brachydanio rerio</em></td>
<td>LC₅₀ (96h, static)</td>
<td>&gt;3.2 (LC₀) mg/l</td>
<td>OECD TG 203, nominal conc.</td>
</tr>
<tr>
<td></td>
<td>LC₅₀ (96h, static)</td>
<td>&lt;4.2 (LC₈₀) mg/l</td>
<td></td>
</tr>
<tr>
<td><em>Salmo trutta</em></td>
<td>LC₅₀ (24 h, static)</td>
<td>2.12 mg/l</td>
<td>Hattula et al. 1979</td>
</tr>
</tbody>
</table>

*Precipitation and use of open system noted (- the actual effect concentration may have been lower ?)  
Not specified whether measured or nominal concentrations (presumably nominal concentrations)

**Table 3.18** Crustaceans, short term

<table>
<thead>
<tr>
<th>Organism</th>
<th>Toxicity</th>
<th>Method</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Daphnia magna</em></td>
<td>EC₅₀ (48h, static)</td>
<td>0.29 mg/l</td>
<td>USEPA 1975, nominal conc.</td>
</tr>
<tr>
<td></td>
<td>NOEC (48h, static):</td>
<td>0.028 mg/l</td>
<td></td>
</tr>
<tr>
<td><em>Daphnia magna</em></td>
<td>EC₅₀ (48h, static)</td>
<td>1.0 mg/l</td>
<td>DIN34812 L11, not specified*</td>
</tr>
<tr>
<td></td>
<td>EC₅₀ (48h, static)</td>
<td>0.32 mg/l</td>
<td></td>
</tr>
<tr>
<td><em>Daphnia magna</em></td>
<td>EC₅₀ (48h, static)</td>
<td>0.63 mg/l</td>
<td>OECD TG 202, nominal conc.</td>
</tr>
<tr>
<td><em>Daphnia magna</em></td>
<td>EC₅₀ (48h, static)</td>
<td>&gt;0.56 mg/l</td>
<td></td>
</tr>
<tr>
<td></td>
<td>EC₉₀ (48h, static)</td>
<td>≤1.8 mg/l</td>
<td>OECD TG 202, nominal conc.</td>
</tr>
</tbody>
</table>

*Not specified whether measured or nominal concentrations (presumably nominal concentrations)

Range finding study for reproduction test
Full report submitted to CAs

**Table 3.19** Algae

<table>
<thead>
<tr>
<th>Organism</th>
<th>Toxicity</th>
<th>Method</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Scenedesmus subspicatus</em></td>
<td>EC₅₀ (72h, static)</td>
<td>15.0 mg/l</td>
<td>DIN38412 L9, not specified*</td>
</tr>
<tr>
<td></td>
<td>EC₅₀ (48h, static)</td>
<td>0.97 mg/l</td>
<td></td>
</tr>
<tr>
<td><em>E</em>₅₀ (96h, static)</td>
<td>EC₅₀ (48h, static)</td>
<td>8.2 mg/l</td>
<td>DIN38412 L9, not specified*</td>
</tr>
<tr>
<td></td>
<td>EC₅₀ (48h, static)</td>
<td>0.89 mg/l</td>
<td></td>
</tr>
</tbody>
</table>

*Not specified whether measured or nominal concentrations (presumably nominal concentrations)
Table 3.20  Higher plants, short term

<table>
<thead>
<tr>
<th>Organism</th>
<th>Toxicity</th>
<th>Method</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Lemna minor</em></td>
<td>EC50 (48 h, static)</td>
<td>cf below*</td>
<td>Blackman et al. 1955</td>
</tr>
</tbody>
</table>

* Method: pH of test medium: 5.1; light intensity: 320 ± 20 ft.candles (approx. 3,520 lux); effect endpoint chlorosis after 48 h exposure and 24 h in pure test medium; nominal concentration

Table 3.21  Microorganisms

<table>
<thead>
<tr>
<th>Organism</th>
<th>Toxicity</th>
<th>Method</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Pseudomonas putida</em></td>
<td>EC50 (17h):</td>
<td>DIN 38412, part 8</td>
<td>BASF 1994</td>
</tr>
<tr>
<td>Activated sludge:</td>
<td>EC50 (30 min):</td>
<td>Inhibition of oxygen consumption</td>
<td>BASF 1994</td>
</tr>
<tr>
<td></td>
<td>EC50 (30 min):</td>
<td>ISO 8192</td>
<td>Bayer</td>
</tr>
</tbody>
</table>

Table 3.22  Prolonged, fish

<table>
<thead>
<tr>
<th>Organism</th>
<th>Toxicity</th>
<th>Method</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Salmo trutta</em></td>
<td>NOEC (21 to 28d)</td>
<td>0.5 mg/l daily renewal of test medium, nominal conc.</td>
<td>Hattula et al. 1979*</td>
</tr>
</tbody>
</table>

* At the exposure concentration of 0.5 mg/l an average BCF of 6.6 was observed, i.e. cf. section 3.1.2.6, time for reaching 95 % equilibrium concentration within the exposure time for the acute study

Table 3.23  Long-term Daphnia, reproduction

<table>
<thead>
<tr>
<th>Organism</th>
<th>Toxicity</th>
<th>Method</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Daphnia magna</em></td>
<td>NOEC (21d)</td>
<td>0.55 mg/l OECD TG 202-II, semi-static, measured concentrations</td>
<td>PCOC Task Force 1997*</td>
</tr>
</tbody>
</table>

*Full report submitted to the CAs. Generally there was good agreement between the nominal and measured concentrations

Other effects

In an in vitro assay with human breast cancer cells, PCOC was observed to have an estrogenic activity with a potency $1 \times 10^{-6}$ of the potency of 17-β-estradiol (Körner et al., 1996; Körner et al., 1997).

Comments

The results from the above mentioned ecotoxicity tests are evaluated to be valid for use in this risk assessment. It was evaluated whether to exclude the studies of Bucaffuso et al. (1981) and Blackman et al. (1955) but the former results are in general accordance with the other acute studies on fish and therefore accepted as valid, whereas the latter results are not considered important in this effects assessment context. When evaluating the validity of ecotoxicity test results, it was considered whether standardized test methods have been followed, but also whether the effect concentrations are measured or nominal, from flow through, semi-static or static tests, from experiments with nominal concentrations only, but performed in closed systems or open systems, and whether or not solvent was used. When evaluating the test data validity
information about the physical-chemical and environmental fate related properties of the substance were also considered.

Most of the mentioned test results are from methods performed according to standard methods/guidelines; US-EPA 1975 ("Methods for acute toxicity tests with fish, macro-invertebrates and amphibians"), OECD 1981, DIN 318412 and ISO 8192. The higher plant and algae study was performed in bright light and PCOC may have been subject to photodegradation, which may have affected the result.

In a prolonged toxicity test, histopathological changes were found in livers and kidneys in fish exposed to 1 ppm PCOC or more (Hattula et al., 1979). The study was conducted before the adoption of test guidelines of OECD, DIN, ISO and US. However, the study seems well performed (cf. also US EPA AQUIRE data quality code 2) and also includes detailed histopathological examinations of various fish tissues using staining techniques.

In the *Daphnia magna* reproduction test, no significant effects were observed in mortality, length or reproduction at the highest concentration used. The study is performed according to OECD guideline 202-II 1984. The concentrations were measured and generally there was good agreement between the nominal and measured concentrations (88 to 106% of nominal in the two highest concentrations 0.32 and 0.56 mg/l). The study is valid and concludes that NOEC (21d) is 0.55 mg/l based on average measured concentrations.

The results from the long term-Daphnia reproduction test seem to be inconsistent with the acute results. One acute EC$_{50}$ and two NOECs (48h) are below the long term NOEC. This is rarely observed but may be caused by slight differences in the studies in medium, sensitivity of the Daphnia clone, etc. The observed EC$_{50}$-values from the three static tests based on nominal concentrations may not reflect the equivalent EC$_{50}$ if such had been based on measured concentrations. In range finding test of the Daphnia reproduction test only 5 Daphnias in 3 duplicate concentrations were used. The mortality was 0% after 48 h at 0 mg/l and 100% at 1.8 mg/l and 3.2 mg/l. EC$_{50}$ in the long-term test was >0.56 mg/l after 48 hours. The conclusion was that EC$_{50}$ (48h) is estimated to be between 0.56 and 1.8 mg/l. This suggests that the acute study by LeBlanc (1980) might be invalid. Another explanation could be the presence of a steep concentration/effect curve and that the acute EC$_{100}$ is approximately 3 times the long-term NOEC. However, since the result from the long-term test is valid and therefore used in the risk assessment, a conclusion on the probable reasons for the discrepancy is not drawn but left open.

QSAR derived acute effect concentrations for polar narcotic substances according to the TGD for fish and daphnids compared to the experimental data suggest that for fish an acute polar narcotic toxic action seems plausible. This is supported by the evaluation of the acute mode of toxic action towards fathead minnow of the closely structurally related substance 4-chloro-3-methylphenol (Russom et al., 1997). For daphnids, however, the difference between the experimental nominal and QSAR predicted EC$_{50}$-value is almost one order of magnitude (cf. Appendix B). However, the results from the acute preliminary Daphnia study and the Daphnia reproduction test employing measured concentrations indicate that the experimental EC$_{50}$-values based on nominal concentrations may be less correct (cf. above).

In a recent in vitro assay, PCOC was observed to have an estrogenic activity with a potency 1 · 10$^{-6}$ of the potency of 17β-estradiol (Körner et al., 1997). Estrogenic effects have been observed e.g. in wild fish from the UK rivers, especially downstream from STP discharges. The reason for the field observations in the UK is, however, not fully elucidated and no hard evidence exists as regards whether PCOC in effluents may cause such effects in situ. A recent study in the UK rivers indicates that other chemicals in this case may be responsible since chemicals with known
estrogenic effects were observed in the rivers and the effects observed on caged fish (Harries et al., 1997). As the caged fish were placed upstream related to the location of the STP receiving PCOC discharge there was no indication of PCOC to be responsible for the observed effects.

**PNEC-estimations**

In the acute studies, the most sensitive aquatic organism was *Daphnia magna*. Based on three studies, a geometric mean EC$_{50}$ (48h) of 0.63 ppm in acute tests is calculated. Using the acute value and an assessment factor of 1,000 would result in a PNEC$_{aquatic, organisms}$ of 0.00063 mg/l. However, the presence of an algae EC$_{10}$, a long term NOEC for fish and a Daphnia reproduction test justifies the use of an assessment factor of 10. Thus, the estimated PNEC-value according to the TGD is:

PNEC$_{aquatic, organisms}$: 0.5/10 = 0.05 mg/l.

The PNEC$_{STP, microorganisms}$ is obtained by using the EC$_{50}$ for inhibition of respiration of activated sludge microorganisms and an assessment factor of 100:

PNEC$_{microorganisms}$: 55/100 = 0.55 mg/l

Because of the absence of experimental data for sediment-dwelling organisms and because log K$_{ow}$ is less than 5, the PNEC$_{sed}$ is not calculated. The reason is that employment of the equilibrium partitioning method and the PEC$_{sed}$ - calculation method would result in the same PEC/PNEC-ratio for sediment dwelling organisms as for pelagic organisms.

### 3.2.2 Atmosphere

No results are available to support an effect assessment in the atmosphere. The atmospheric photochemical half-life is estimated to be 30 hours.

### 3.2.3 Terrestrial compartment

Since no ecotoxicological data are available for soil organisms the equilibrium partitioning method has been applied:

PNEC$_{soil}$ = (K$_{soil, water}$/RHO$_{soil}$) · PNEC$_{aquatic, organisms}$ · 1,000 = 0.36 mg/kg wet weight.

### 3.2.4 Non compartment specific effects relevant to the food chain

The observed BCF’s in fish were small (max. 30). Therefore, secondary poisoning is not likely.
3.3 RISK CHARACTERISATION

3.3.1 Aquatic compartment

Environmental risk related to production, processing and formulation

Aquatic organisms

Risk quotient by employing TGD and including connection to STP:

Table 3.24 PEC/PNEC(local) for aquatic organisms

<table>
<thead>
<tr>
<th>Life cycle stage</th>
<th>PEC(local, water (mg/l))</th>
<th>PNEC(aquatic organisms)</th>
<th>PEC/PNEC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site specific*</td>
<td>0.0038</td>
<td>0.05</td>
<td>0.08</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.0014</td>
<td>0.05</td>
<td>0.03</td>
</tr>
</tbody>
</table>

*Based on monitoring data

The local PEC/PNEC is <1 and a local risk for aquatic organisms is not indicated. Considering formulation outside the two main manufacturing sites, the worst-case approach using 10% of production volume at one formulation site did not indicate risk to aquatic organisms. The regional PEC/PNECs are clearly << 1 and therefore not presented here.

Because PCOC ready biodegradability is considered a borderline case, the PEC/PNEC ratio is calculated considering PCOC as inherently biodegradable. This increases PEC/PNEC with a factor of 4. Thus, the PEC/PNEC ratios are <1 even when PCOC is considered inherently biodegradable (cf. Appendix B).

Risk for microorganisms in STPs

Using the PNECmicroorganisms 0.55 mg/l and the estimated concentrations in STP effluent as the exposure concentration, the following ratios are found:

Table 3.25 PEC/PNEC microorganisms

<table>
<thead>
<tr>
<th>Life cycle stage</th>
<th>PECSTP, microorganisms (mg/l)</th>
<th>PNECmicroorganisms (mg/l)</th>
<th>PEC/PNEC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific site*</td>
<td>0.004</td>
<td>0.55</td>
<td>0.01</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.013</td>
<td>0.55</td>
<td>0.03</td>
</tr>
</tbody>
</table>

*Based on monitoring data

Based on these values, a local risk for effects on the microorganisms in STPs is not indicated.

Aquatic risk from agricultural use of phenoxy herbicides

The environmental risk assessment according to the pesticide scenario is not conducted based on a decision at the EU Technical meeting on risk assessment of existing substances (TM III, Nov.1996) referring to this part of the risk assessment being conducted by DGVI working group on risk assessment of plant protection products.
Conclusion of the risk assessment of aquatic compartment

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

3.3.2 Atmosphere

Based on the calculated Henry's Law constant and the data on evaporation from rice fields, and other laboratory studies, it is concluded that PCOC will volatilize at significant rates from open water and wet soil.

In the air, the substance will be exposed to photochemical degradation at a half-life of 30 hours and based on the solubility, PCOC is expected to be washed out by rain to soil and water.

PEC_{air}: No effect data exist and therefore no PEC/PNEC ratio can be calculated.

Conclusion of the risk assessment of atmosphere compartment

(X) 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.

3.3.3 Terrestrial compartment

No data are available on the terrestrial toxicity. Therefore, the equilibrium partitioning method described in the TGD is applied as a conservative approach, comparing PEC_{soil} to PNEC_{soil}.

PNEC_{soil} = 0.36 mg/l.

Risk to the terrestrial compartment from production, processing and formulation

<table>
<thead>
<tr>
<th>Table 3.26 PEC/PNEC_{local, soil}</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>Specific site*</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Formulation</td>
</tr>
</tbody>
</table>

*PEC specific site is estimated using monitoring data

Risk to the terrestrial compartment during agricultural use

The environmental risk assessment according to the pesticide scenario is not conducted based on a decision at a EU Technical meeting on risk assessment of existing substances (TM III, Nov.1996) referring to that this part of the risk assessment will be conducted by the DGVI working group on risk assessment of plant protection products.

Conclusion of the risk assessment of terrestrial compartment

(X) 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.

The risk ratios for the local risk are below 1 for the considered exposure scenarios (i.e. specific manufacturing site including production, processing and formulation of herbicides as well as the scenario employing a formulation of phenoxy acids). Based on the ratio PEC/PNEC soil estimated to be <1, no risk for terrestrial organisms is indicated.
3.3.4 Non compartment specific effects relevant to the food chain

The risk characterisation of secondary poisoning is not performed because the BCF for fish is max. 30, i.e. considerably below 100. Therefore, even though log \( K_{ow} \) is above 3, there are no indications for a bioaccumulative potential of the substance and thus no concern for secondary poisoning.

Conclusion of the risk assessment of non-compartment specific effects relevant to food chains

(X) 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.
4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1 General discussion

P-chloro-o-cresol (PCOC) is used in the chemical industry as an intermediate in the synthesis of chlorophenoxy herbicides, e.g. MCPA (4-chloro-2-methylphenoxy acetic acid), MCPB (4-chloro-2-methylphenoxy butyric acid), and mecoprop (2-(4-chloro-2-methyl-phenoxy)-propionic acid, MCPP). PCOC is no longer produced in Denmark.

PCOC is found as an impurity in the herbicides MCPA, MCPB, and mecoprop.

During production of PCOC and in the synthesis of other compounds (downstream uses) PCOC is released to the environment through emitted air and wastewater. As a degradation product and as an impurity PCOC will also be found at the application sites of the herbicides mentioned above.

PCOC was detected upon branches sprayed with MCPA, 2 weeks post application, at concentrations of 8,900 ppb; and upon potatoes, carrots, green lettuce and onions grown on fields adjacent to a treated railway bed in Northern Finland at concentrations of 0.2, 2.9, 52.9 and 593.0 ppb, respectively (Paasivirta et al., 1983).

Concentrations in the environment are estimated in Chapter 3.

The most important route of direct exposure is by inhalation in occupational settings in the production of the substance itself or during use in the synthesis of other compounds (downstream uses). Oral or dermal exposure during production is assumed to be of relevance only in the case of accidents.

Exposure to PCOC as an impurity in herbicides such as MCPA can also occur during crop spraying.

In the Danish Product Register PCOC is only registered as a substance, but was formerly found in one product at a concentration of around 1% in a survey carried out in 1985. With reference to information from industry it is concluded, that no exposure takes place through use of ordinary (non-herbicidal) consumer products.

One potential source of indirect exposure is the consumption of food treated with the herbicides, of which PCOC is a degradation product or an impurity, and drinking of water contaminated by the substance.

4.1.1.2 Occupational exposure

Two companies in the U.K. are high volume producers of PCOC, which is also used as an intermediate for further synthesis at the same sites of the herbicides MCPA, mecoprop, and MCPB. In addition one Dutch high volume producer has been identified, producing PCOC as a non-isolated part of a continuous process, which need not be reported under the Regulation. There are no data on occupational exposure available from this producer.
No occupational exposure limits for PCOC have been found but for related substances (cresols and chlorophenols) the values given below apply.

The occupational exposure limit (8-hour threshold limit value (TLV)) for cresols set by the UK and DK authorities (all isomers) is 22 mg/m$^3$ (HSE, 1994; AT, 1994). For chlorophenols (all isomers) the TLV in e.g. Denmark is 0.5 mg/m$^3$ (AT, 1994).

**Production**

At one of the production sites, plant operators were monitored at the workplace and tank farm operators were monitored whilst offloading PCOC to the road tanker on four and three occasions, respectively (Road tanker is used to move the substance within the area). According to the manufacturer less than 20 people are involved in these operations (pers. communication, 1997). For the plant operators the monitoring period lasted from 183 to 238 minutes. For the tank farm operators the monitoring period lasted from 15 to 101 minutes. Concentrations for plant operation and offloading to road tankers ranged from below detection limit to about 5 mg/m$^3$ (equivalent 8-hour TWA’s max. ca. 5 mg/m$^3$) (Marks, 1997b).

During cleaning operations, which were infrequent (twice per year) and where protective clothing and breathing apparatus was worn, a concentration of about 53.8 mg/m$^3$ was recorded (8 hr. TWA 1.2 mg/m$^3$). The monitoring was done at one occasion (A.H. Marks, 1997b). According to the manufacturer, less than 20 people are involved in this operation (pers. comm.). One of the main manufacturers has reported that the exposure actually only applies to one worker. (pers. comm., Marks, March 6 1998)

Beside the actual operator monitoring point location monitoring in working areas was performed showing TWA values of less than 5 mg/m$^3$ for all instances (e.g. control room, by reactor, by holding vessels, process scrubber, and whilst offloading PCOC to road tanker) except cleaning of the equipment where a concentration of 1,274.8 mg/m$^3$ was recorded (equivalent to 8 hr-TWA 18.6 mg/m$^3$) (Marks, 1997b).

For production of phenoxy herbicides, which was done at the same plant, monitoring data (operator and point location monitoring) was of a similar order of magnitude, less than 5 mg/m$^3$ at all occasions. Here cleaning of the equipment was not monitored (Marks, 1997b). PCOC is used in the molten state, which together with the corrosive nature of the substance, ensures that workers comply fully with personal protective equipment (PPE) requirements (pers. Comm., Marks, March 6, 1998).

There are no monitoring data on PCOC available from the other UK production site. According to the producer the occupational exposure to PCOC is regarded as being minimal, because all vessels and sample points are enclosed and maintained under extraction with air being discharged via caustic scrubbing columns. In addition all employees are provided with appropriate PPE is laundered and maintained by the company (Nufarm, 1997b).

According to the producers most of the manufacture and use of PCOC do not require operator intervention. However, there are exceptions e.g. maintenance and tanker loading and unloading. For these operations as well as for emergency situations appropriate PPE are provided including suit (PVC or full body cotton overalls), full-face mask, PVC gloves, boots (leather or PVC), safety helmet and glasses (Marks, 1997b; Nufarm, 1997b).

Assuming inhalation of 10 m$^3$ of air during an eight-hour work shift, for a 70 kg person, 5 mg/m$^3$ would correspond to a realistic worst-case dose for systemic toxicity of about 0.7 mg/kg/day. It can be noted that while this concentration is less than 0.25 of the TLV for cresols and thus meets
the UK regulatory standards, it is 10 times higher than the TLV for chlorophenols, which from a chemical-structural point of view are quite similar to PCOC.

The EASE estimation (Appendix D) of inhalation exposure during production and further processing of PCOC assuming use pattern is closed system and the pattern of control is full containment resulted in exposures of 0 to 0.1 ppm corresponding to 0 to 0.6 mg/m$^3$. This range is much lower than monitored data.

While some degree of dermal exposure may also occur, the EASE model predicts this as being of no consequence when compared with the inhalation route (Appendix D). Direct contact with the skin would only happen in the case of accidents, where it could result in systemic toxicity as well as severe burns.

In conclusion the known corrosive nature of PCOC together with its use in the molten form ensures that routine transfer and equipment cleaning and maintenance operations are performed with strict adherence to PPE requirements, resulting in minimal exposure to workers via both dermal and inhalation routes.

**Application**

In certain occupational settings such as municipal gardening, worst-case exposures may be higher. Using a standard model for plant protection product use (Lundeher, 1992) which also incorporates exposure during mixing and loading, a geometric mean exposure of 0.047 mg/kg/day is calculated for hand-held (knapsack) spraying of 1 ha assuming application of 2 kg/ha MCPA with a 1% content of PCOC and 100% absorption. The 90th percentile exposure using the same inputs results in a total of 0.35 mg/kg/day.

### 4.1.1.3 Consumer exposure

PCOC is not found in any ordinary consumer products. It can occur as an impurity or breakdown product in herbicides used for controlling weeds in lawns of private gardens. One such product available in the vegetable section of a Danish supermarket contains MCPA in concentrations of 5.20 g/l in a one-litre plastic bottle provided with a hand pump for aerosol generation. As this form of dispensation can lead to the highest exposures, a realistic worst case for combined inhalation and dermal exposure of 10% is assumed. If PCOC is present as an impurity at 0.5%, and a further 0.5% is generated by exposure of the aerosol to sunlight, a total exposure to PCOC of 5.2 mg/event, or 0.07 mg/kg/event for a 70 kg person could result.

It is difficult to assess the frequency with which such consumer exposure might occur, directions for use on the particular product only state that it can be used during the entire growth period, but is most effective during periods of rapid growth in May, June, July and August (Source, "Toxan" - Labelling information, Distribution: Bayer Denmark A/S, Gammelager 1, 2605 Brøndby). In addition to MCPA, one litre of this product is also stated to contain 1.50 g Dichloprop-p and 0.32 g Dicamba as active ingredients). Assuming a realistic worst case of five-

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6 During spraying, including mixing of pesticides, using sprayers on tractors the exposure is generally estimated to be around 0.00005% of the amount sprayed in a concentration of 15 g/ha using the best available technology. Using standard spraying equipment the exposure is 0.0002% of the amount sprayed (Lund & Kirknel, 1995)

Using a standard model for plant protection product use (Lundeher, 1992) which also incorporates exposure during mixing and loading, and assuming 2 kg MCPA per ha, with a 1% content of PCOC, a geometric mean exposure of 0.02 mg/kg body weight/day is derived, or for the 90th percentile, 0.28 mg/kg body weight/day for 20 ha of downward vehicle-mounted spraying.
time application per year the total yearly dose of PCOC would be \(5 \cdot 0.07 = 0.35\) mg/kg/year (= \(9.6 \cdot 10^{-4}\) mg/kg/day).

### 4.1.1.4 Indirect exposure via the environment

Exposure of the environment can take place during the production of PCOC itself, as well as from the production and use of phenoxy herbicides.

At the production site the potential exposure would be through wastewater and air effluent.

At sites of MCPA or other phenoxy herbicide applications, indirect exposure may occur, since PCOC is an impurity in the herbicide and has been identified as a degradation product of MCPA.

According to USES 1.0 calculations involving local indirect exposure due to use of herbicides the following daily doses can be expected:

<table>
<thead>
<tr>
<th>Intake</th>
<th>Daily Dose (mg/kg/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intake air</td>
<td>(1.63 \cdot 10^{-9})</td>
</tr>
<tr>
<td>Intake drinking water</td>
<td>(8.95 \cdot 10^{-8})</td>
</tr>
<tr>
<td>Intake fish</td>
<td>(4.04 \cdot 10^{-9})</td>
</tr>
<tr>
<td>Intake stem of plant</td>
<td>(7.97 \cdot 10^{-10})</td>
</tr>
<tr>
<td>Intake root of plant</td>
<td>(3.38 \cdot 10^{-12})</td>
</tr>
<tr>
<td>Intake meat</td>
<td>(5.49 \cdot 10^{-13})</td>
</tr>
<tr>
<td>Intake milk</td>
<td>(5.47 \cdot 10^{-13})</td>
</tr>
</tbody>
</table>

Amounting to a total human dose of \(9.60 \cdot 10^{-8}\) mg PCOC/kg/day.

The EUSES calculations (November 1997) for local indirect exposure resulting from production of PCOC are as follows:

<table>
<thead>
<tr>
<th>Site Description</th>
<th>Ready Biodegradability</th>
<th>Inherent Biodegradability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific site(^7)</td>
<td>(1.18 \cdot 10^{-4}) mg/kg/day</td>
<td>(2.52 \cdot 10^{-4}) mg/kg/day</td>
</tr>
<tr>
<td>Formulation site(^8)</td>
<td>(1.25 \cdot 10^{-4}) mg/kg/day</td>
<td>(4.30 \cdot 10^{-4}) mg/kg/day</td>
</tr>
</tbody>
</table>

We assume PCOC being readily biodegradable. However, knowing the substance may be a borderline case, the calculations for inherent biodegradability are included for comparison purposes only.

The EUSES calculations (November 1997) for regional indirect exposure assuming ready or inherent (worst case) biodegradability are given below. Again, inherent biodegradability has been included for comparison purposes only:

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\(^7\) Specific site incl. production, processing and formulation. The values are based on average monitoring data on emissions from the two main manufacturers

\(^8\) Formulation site is a generic site where it is assumed that 10% of the total PCOC production is formulated (worst case)
Daily human dose through:

<table>
<thead>
<tr>
<th>Intake</th>
<th>Ready biodegradability (mg/kg/day)</th>
<th>Inherent biodegradability (mg/kg/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intake air:</td>
<td>$1.60 \cdot 10^{-7}$</td>
<td>$2.17 \cdot 10^{-7}$</td>
</tr>
<tr>
<td>Intake drinking water:</td>
<td>$4.49 \cdot 10^{-6}$</td>
<td>$8.05 \cdot 10^{-6}$</td>
</tr>
<tr>
<td>Intake fish:</td>
<td>$7.74 \cdot 10^{-6}$</td>
<td>$1.39 \cdot 10^{-5}$</td>
</tr>
<tr>
<td>Intake from leaf crops:</td>
<td>$2.44 \cdot 10^{-7}$</td>
<td>$3.31 \cdot 10^{-7}$</td>
</tr>
<tr>
<td>Intake root of crops:</td>
<td>$2.56 \cdot 10^{-7}$</td>
<td>$3.01 \cdot 10^{-7}$</td>
</tr>
<tr>
<td>Intake meat:</td>
<td>$1.29 \cdot 10^{-9}$</td>
<td>$2.25 \cdot 10^{-7}$</td>
</tr>
<tr>
<td>Intake milk:</td>
<td>$7.60 \cdot 10^{-10}$</td>
<td>$1.33 \cdot 10^{-3}$</td>
</tr>
</tbody>
</table>

Regional total daily intake: $1.29 \cdot 10^{-5}$ mg/kg/day $2.28 \cdot 10^{-5}$ mg/kg/day

4.1.1.5 Combined exposure

Some parts of a population are exposed to PCOC both during work and during indirect exposure via the environment.

A person working at a production site for PCOC and/or phenoxy herbicides or a person spraying phenoxy herbicides on a field might apart from the occupational exposure also be exposed via the environment. However, the potential routes of exposure differ and as can be seen from 4.1.1.1, 4.1.1.2, and 4.1.1.3 the magnitude of the exposure varies greatly. In Table 4.1 the calculated exposure data are given.

Table 4.1 Calculated exposure data excl. agricultural spraying

<table>
<thead>
<tr>
<th>Exposure</th>
<th>mg PCOC/kg/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Occupational exposure during production</td>
<td>0.7</td>
</tr>
<tr>
<td>Spraying (municipal - hand spraying)</td>
<td>0.35</td>
</tr>
<tr>
<td>Consumer exposure</td>
<td>$9.5 \cdot 10^{-4}$</td>
</tr>
<tr>
<td>Indirect regional exposure via the environment</td>
<td>$1.3 \cdot 10^{-5}$</td>
</tr>
<tr>
<td>Local indirect exposure*</td>
<td>$1.2 \cdot 10^{-4}$</td>
</tr>
<tr>
<td>Combined exposure, total</td>
<td>1.05</td>
</tr>
</tbody>
</table>

*Local indirect exposure resulting from production, formulation or processing is estimated assuming ready biodegradability

4.1.2 Effects assessment: Hazard identification and Dose (concentration) - response (effect) assessment

All the PCOC studies below that were performed by Scantox, Denmark and Teknologisk Institut were conducted in accordance with the OECD guidelines for testing of chemicals and GLP.

The identity of the substance was as described in chapter 1 i.e. 97.09% 4-chloro-2-methylphenol, 1.21% 6-chloro-2-methylphenol, 0.92% 2-methylphenol, and 0.78% 2,4-dichloro-6-methylphenol. The study by the Institute of Toxicology in Denmark (Hansen, 1996) used a 97% pure Aldrich PCOC batch no. C5.520-8. The study by Hattula et al. (1979) used 100% pure PCOC.
4.1.2.1 Toxico-kinetics, metabolism and distribution

Very little is known about the toxico-kinetics, metabolism, distribution, and excretion of PCOC in humans and experimental animals. However, from the acute toxicity studies it can be inferred that PCOC can be taken up in the body through the gastrointestinal tract, the skin, and via inhalation. There is no information on the metabolism and excretion of PCOC.

The concentrations of PCOC in liver, kidney, spleen, and muscle were studied in an acute and a repeat dose study (Hattula et al., 1979). After 28 days of dosing by gavage with 100, 250, or 500 mg PCOC/kg, PCOC was found in the highest concentration 2.81 mg/kg in the spleen, and in the lowest concentration 0.27 mg/kg in muscle tissue in the high dose group. In the low-dose group only traces of PCOC were found.

PCOC was found in concentrations of 47-31 µg/g in the liver of rats receiving 2-3 g/l MCPA in the drinking water for three months (Hattula et al., 1977). A recent rat metabolism study with MCPA performed at Hazleton Lab. showed that PCOC was not a metabolite. It is therefore possible that the PCOC in the Hattula-study was a contaminant of MCPA (Jahanshahi J., 1995).

Acute toxicity

Animal data: Acute oral toxicity

In a guideline (401) study using five male and five female rats per group and dosing by gavage with the doses 1,728, 2,488, 3,583 and 5,160 mg/kg with oleum arachidis as vehicle, an LD50 of 3,195 mg PCOC/kg (range 2,698 – 3,834 mg/kg) was found.

In the 5,160 mg/kg group all animals died within one hour after dosing, in the 3,583 mg/kg group 5 deaths occurred up to 6 hours after dosing, in the 2,488 mg/kg group three deaths occurred within one day after dosing, and in the 1,728 mg/kg group no deaths occurred. Symptoms observed just after dosing at all dose levels were paresis and depressions. On the second day, ruffled fur, which lasted to day five in the 3,583 mg/kg group, was seen. Animals that died during the observation period showed bleeding in the mucous membrane of the stomach at autopsy. Animals sacrificed after the 14-day observation period showed no dose related macroscopic changes. However, two of the animals from the 2488 mg/kg group, sacrificed after the 14-day observation period, showed infiltrations between the oesophagus area of the ventricle and the diaphragm. In one animal from the high-dose group, infiltrations between the oesophagus area of the ventricle and the liver were seen (Scantox, 1982b).

Groups of ten male Wistar rats, 2-3 months of age, were given 1,000, 1,100, or 1,200 mg PCOC/kg with the substance dissolved in olive oil. The animals were all killed 24 hours after dosing. A LD50 of 1,190 mg/kg was derived (Hattula et al., 1979). At the histopathological examination the following observations were made: at 1,000 and 1,100 mg/kg inflammatory mononuclear infiltration was seen in many glomeruli in the kidney. Inflammatory infiltrations were also seen in other parts of the kidney mostly around distal tubules. At 1,200 mg/kg also histopathological alterations in the liver and spleen were seen. In the liver numerous pycnotic nuclei and hydropic degeneration of cytoplasm were observed. In the spleen the reaction centres were unusually large (Hattula et al., 1979).

Further studies on the acute oral toxicity of PCOC to rats include BASF (1978) and Hazleton (1977). These test reports have not been available, but their results (see Table 4.1) are in accordance with the results of the only guideline study available (Scantox, 1982b). It can be concluded that PCOC not only shows corrosive properties but also properties resulting in systemic effects, i.e. effects on liver and kidney.
In rats the oral LD$_{50}$ of PCOC is above 2,000 mg/kg in the most reliable study. In mice, Schrötter et al. (1977) report the oral LD$_{50}$ of PCOC as being 1,330 mg/kg, but few experimental details are provided.

In range finding studies of PCOC in aqueous gum tragacanth emulsion, mice died consistently at lower doses (4/4 at 1,200 mg/kg and 3/4 at 576 mg/kg) suggesting that the vehicle may play an important role in determining absorption following oral administration (Huntingdon, 1997).

**Animal data: acute inhalation toxicity**

Groups of five male and five female rats were exposed to an aerosol containing 0, 5.79, 8.33, 9.11, or 10% PCOC in 50% alcohol for 4 hours following OECD Guideline 403. All deaths during the study occurred during exposure or within the first hour after exposure. The deaths were distributed as follows between the groups: control 0 deaths, 5.79% 0 deaths, 8.33% two deaths, 9.11% four deaths, 10% 7 deaths. The LC$_{50}$ was calculated as 900 mg/m$^3$ (0.9 mg/l range 0.83 - 1.08 mg/l) (Scantox, 1983a). The alcohol aerosol was used as it was not possible to generate a dust aerosol, as the test substance clumped. The LC$_{50}$-value is based on the nominal concentration in the experiment. The symptoms observed during and after exposure were respiration difficulties, depressions, ruffled fur and bleeding from the nose. These symptoms occurred in a dose related manner. Petechiae of the lungs were also observed.

At macroscopic examination of the animals that died up to the first hour after dosing bleeding of the lungs and a thin, mucous, yellowish content of the small intestine were found.

Another study was performed by Hazleton Lab in 1977 is cited from BUA (1994): The original report is not available and the study was carried out before guidelines were in general use. By inhalation of 2,000 – 30,000 mg PCOC/m$^3$ (average particle size of 0.6 µm) for 4 hours no deaths occurred but swelling red noses and lips were seen. In one animal blood was found in the urine.

Animals sacrificed immediately after the exposure period or after 14 days of observation period showed no alterations in the lung or essential organs.

**Animal data: acute dermal toxicity**

Groups of five male and five female rats were dermally dosed with 1,667, 2,000, 2,400, or 2,880 mg PCOC in oleum arachidis in a guideline study (402). A LD$_{50}$ of 2240 mg/kg (range 2,023 – 2,484) was calculated from the observed deaths (Scantox, 1982c).

In the 2,880 mg/kg group 9 animals died within 6 hours after dosing, in the 2,400 mg/kg group six animals died within one day after dosing, in the 2,000 mg/kg group four animals died within one day after dosing, and in the 1,667 mg/kg group no animals died. At necropsy bleeding of the lungs, a mucous, red-yellow content of the jejunum, enlarged kidneys and blood or blood coagulum in the bladder plus bleeding of the bladder wall were observed.

During the first 24 hours after treatment blood was observed in the urine of all rats. From the day after treatment erythema and oedema at the application sites were seen. Paresis occurred in nearly all animals 1 to 6 hours after treatment. Depressions occurred up to 2 days after treatment, and ruffled fur up to 3 days after treatment. In animals sacrificed on day 14 weak bleeding of the intestine (jejunum) was observed in five of the rats (dose levels not stated).

In **Table 4.2** the acute toxicity data found for PCOC are given without any comments on quality of the studies.
Table 4.2  Data on acute toxicity of PCOC

<table>
<thead>
<tr>
<th>Species</th>
<th>Application</th>
<th>Dose</th>
<th>Effect</th>
<th>Literature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rat</td>
<td>oral</td>
<td>3,195 mg/kg</td>
<td>LD₅₀</td>
<td>Scantox, 1982b</td>
</tr>
<tr>
<td>Rat</td>
<td>oral</td>
<td>1,190 mg/kg</td>
<td>LD₅₀</td>
<td>Hattula et al., 1979</td>
</tr>
<tr>
<td>Rat</td>
<td>oral</td>
<td>2,650 mg/kg</td>
<td>LD₅₀</td>
<td>Hazleton Lab, 1977</td>
</tr>
<tr>
<td>Mouse</td>
<td>oral</td>
<td>2,700 mg/kg</td>
<td>LD₅₀</td>
<td>BASF AG, 1978 *</td>
</tr>
<tr>
<td>Rat</td>
<td>oral</td>
<td>1,330 mg/kg</td>
<td>LD₅₀</td>
<td>Schröter et al., 1977</td>
</tr>
<tr>
<td>Mouse</td>
<td>i.p.</td>
<td>794 mg/kg</td>
<td>LD₅₀</td>
<td>Hattula et al., 1979</td>
</tr>
<tr>
<td>Rat</td>
<td>i.p.</td>
<td>570 mg/kg</td>
<td>LD₅₀</td>
<td>BASF AG, 1978 *</td>
</tr>
<tr>
<td>Rat</td>
<td>inhal, 4h.</td>
<td>900 mg/m³</td>
<td>LC₅₀</td>
<td>Scantox, 1983a</td>
</tr>
<tr>
<td>Rat</td>
<td>inhal, 4h.</td>
<td>&gt;30,000 mg/m³</td>
<td>LC₅₀</td>
<td>Hazleton Lab., 1977</td>
</tr>
<tr>
<td>Rat</td>
<td>dermal</td>
<td>2,240 mg/kg</td>
<td>LD₅₀</td>
<td>Scantox, 1982c</td>
</tr>
<tr>
<td>Rat</td>
<td>dermal</td>
<td>&gt;5,000 mg/kg</td>
<td>LD₅₀</td>
<td>Hazleton Lab, 1977*</td>
</tr>
</tbody>
</table>

*Unpublished results sited in a BUA report (BUA, 1994)

In relation to acute oral, dermal and inhalation acute toxicity the Scantox Reports (1982b,c, 1983a) are found to be most reliable. For the acute oral toxicity the Hazleton and BASF studies support the oral LD₅₀ found by Scantox. Poor reporting of the Hattula study makes its interpretation difficult. No details are available which would allow further interpretation of the Hazleton inhalation study.

The overall conclusion for acute toxicity is:

\[
\text{LD}_{50} \text{oral, rat} = 2,650 – 3,195 \text{ mg/kg} \\
\text{LC}_{50} \text{inh, rat} = 0.9 \text{ mg/l (as an EtOH aerosol)} \\
\text{LD}_{50} \text{dermal, rat} = 2,240 \text{ mg/kg}
\]

4.1.2.2  Irritation and Corrosivity

Animal data: skin irritation

In a guideline (404) study 6 female rabbits were dermally exposed to 0.5 g PCOC in 0.1 ml oleum arachidis. A primarily irritation index of 8.0, the maximum value obtainable, was calculated. Immediately after removal of the test substance the skin was white as a sign of initial necrosis (Scantox, 1982d).

BUA (1994) reports a study of Hazleton labs (1977), where rabbits received 500 mg PCOC on the shaved back in a semi-occlusive bandage. It is not stated if a vehicle was applied, and what time of exposure was used. After 12 hours necroses were observed and after 24 hours pronounced erythema with light oedema was observed.

BUA (1994) reported a study of BASF where occlusive exposure to 80% of PCOC in water was carried out (species used and amount applied not mentioned). It was concluded that PCOC was very corrosive. After only one minute of exposure necrosis was found. After 20 minutes the necrosis was very pronounced, and after 8 days it had not disappeared. On day 8 after application the skin was still scarred.
In the rabbits eye BUA (1994) citing BASF reports 50 mg PCOC in an 80% aqueous solution as strongly corrosive. The eye turned red and after 1-hour oedema and opacity of the cornea was found. After 8 days the clinical observations were the same and a staphyloma was found.

In conclusion, some of the studies concerning corrosive effects of PCOC are cited from secondary references, but together with the results of the irritation test (Scantox, 1982d), they indicate that PCOC, according to EU criteria, may be classified as corrosive with R35: causes severe burns, in agreement with classification by the manufacturers.

At least one human fatality attributed to PCOC poisoning has been reported following exposure of the face and neck to a momentary blast of PCOC and steam during a workplace accident. It was not possible to estimate the dose or concentration involved (Pers. comm., HSE, UK 1996).

4.1.2.3 Sensitisation

In a Guinea Pig maximization test carried out according to OECD guidelines (Scantox, 1982e) PCOC caused no sensitisation. 40 female albino Guinea Pigs were used in the study. As the provocation test with 30% solution of PCOC caused erythema, a further provocation test with 10% and 20% of PCOC applied on the left and right flank, respectively, was carried out a week later. No clear differences between the control group and the test group were found at this occasion. Some animals of both groups were reacting with erythema (score 1-2). The reactions in the two groups were of the same magnitude. Macroscopically none of the reactions appeared to be of allergic nature.

The report by BUA (1994) mentions another negative sensitisation study. However, the study does not seem to have been reported properly.

4.1.2.4 Repeated dose toxicity

There are three available studies on repeated dose toxicity of PCOC.

In a guideline (407) study, groups of five male and five female rats were given 0, 50, 200, or 800 mg PCOC/kg in oleum arachidis by gavage for 28 days (Scantox, 1982a). During the last three days of dosing three rats from the 800 mg/kg group showed salivation after dosing, and on the last day of dosing three rats from the group had ruffled fur. Body weight gain and feed consumption did not differ between groups. In blood parameters the thromboplastin time and the number of leucocytes were statistically smaller in females from the 800 mg/kg group. In males from the same group the erythrocyte count was statistically significantly reduced. Serum alanine-aminotransferase (ALAT) was statistically significantly increased in males of the 800 mg/kg group, and marginally increased in females. In females from the 800 mg/kg group relative and absolute liver weights were significantly increased.

No histopathological changes were seen in any organ at 800 mg/kg. The changes of ALAT and liver weights in the 800 mg/kg group indicated mild toxicity to the liver. It was concluded in the test report that 800 mg/kg is a LOAEL, and that 200 mg/kg is a NOAEL.

Hattula et al. (1979) dosed groups of ten male Wistar rats with 0, 100, 250, or 500 mg PCOC/kg in olive oil for 28 days by injection (gavage). It is very difficult to interpret the results of this study, basically because of lack of tables and explanations to the few tables given. However, at 100 mg/kg all investigated organs were normal except for the small intestine, which had necrotic areas of the mucosa. The dose relationship of the other histopathological observations mentioned
is obscure. It is stated that blood analyses showed that leucocytes were decreased with larger doses. At 500 mg/kg a clear-cut leucopenia was found.

In a combined repeated dose/reproduction screening test carried out according to OECD draft guideline 422 (Hansen, 1996) groups of 10 male and 10 female rats per dose were given 0, 50, 200, or 600 mg PCOC/kg in soybean oil by gavage for two weeks prior to mating until day 20 of gestation i.e. dosing was for a total of 40-45 days.

Weight gain was slightly reduced, and water consumption increased in the highest dose groups. Males in the 600 mg/kg group showed a decrease in haemoglobin concentration (p<0.01). (A slight decrease in plasma creatine (p<0.05) in the middle dose group was considered to be without physiological significance.)

A dose-related decrease in the absolute and relative weight of the adrenals of female rats was seen (p<0.05 at 200mg/kg, p<0.01 at 600 mg/kg) but was unaccompanied by histopathological changes, and without obvious toxicological significance.

No effects were seen in other macroscopic and histological examinations of the organs. No behavioural changes were found by a functional observational battery, or in motor activity. It was concluded that the NOAEL was 200 mg/kg.

With regard to respiratory irritation and corrosivity after repeated dosing no data are available. However, due to the caustic properties of the substance it seems unlikely that an inhalation study would add any new information on the systemic toxicity. Further, it seems that the way the substance is handled and used in the existing productions do not lead to any respiratory problems.

At both production sites health surveillance programmes including examination of the respiratory function have been undertaken for several years. According to the medical reports submitted by the producers no significant increase in any specific symptoms such as sore throats, coughs and changes of lung function and no significant group changes of lung function have been observed (Marks, 1997b; Nufarm, 1997b).

4.1.2.5 Mutagenicity

Genetic toxicity in vitro

According to Ames et al., 1975, and/or OECD guideline 471 four direct plate Ames tests (Räsänen et al., 1977; Teknologisk Inst, 1982; Strobel & Grummt, 1987; BASF, 1988) and one pre-incubation Ames test (BASF, 1988) have been carried out to study the mutagenicity of PCOC in the dose range 1-500 µg/plate.

Ames direct plate test was performed with the Salmonella typhimurium strains TA1537, TA1535, TA100, and TA98 at 0, 1, 5, 10, 50, 100, and 500 µg/plate with and without metabolic activation. The identity of the substance was as described in chapter 1. There was clear general toxicity in all strains at 500 µg/plate, but none of the strains showed an increase in the number of revertants/plate (Teknologisk Institut, 1982).

Ames direct plate test was performed with the Salmonella typhimurium strains TA1537, TA1535, TA100, and TA98 at 0, 0.5, 5, 50, and 500 µg/plate with and without metabolic activation. None of the strains showed an increase in the number of revertants/plate (Räsänen et al. 1977).
Ames direct plate test was performed with the *Salmonella typhimurium* strains TA1537, TA1535, TA100, and TA98 at 0, 20, 100, 500, 2500, and 5000 µg/plate and at 0, 4, 20, 100, 500, and 1500 µg/plate with and without metabolic activation. There was clear general toxicity in all strains at and above 500 µg/plate, and none of the strains showed an increase in the number of revertants/plate (BASF, 1988).

An Ames direct plate test using the strains TA98, TA100, TA97 and TA104 at 10, 25, 50, 100, 250, 500, and 1000 µg/plate with and without metabolic activation showed a 4.4 fold dose related increase with TA97-S9 and a 5.4 fold dose related increase with TA97+S9. Only these results were significant. At the highest dose a toxic effect was found in all the strains (Strobel & Grummt, 1987). The report of these results in the literature leaves open some questions with regard to the interpretation of results. For this reason an additional test was performed.

In this new test, 97% PCOC (Aldrich lot no. 3302005) was dissolved in DMSO and tested according to the Salmonella/microsome standard plate assay in *S. typhimurium* strains TA-97 and TA-98 at doses of 500, 250, 100, 25, and 10 µg/plate with and without S9-mix (at 2 and 4 mg S9 protein/plate). No mutagenic effect was seen with or without metabolic activation in either strain. The experiments were repeated again with the same results. (Binderup, 1996).

In the Ames test with pre-incubation (BASF, 1988) *Salmonella typhimurium* strains TA1535, TA100, TA1537, and TA98 was used with and without metabolic activation in concentrations of 0, 4, 20, 100, 500, and 1,000 µg/plate and 0, 15, 30, 60, 125, and 250 µg/plate (two separated series). General toxicity occurred at dose levels of 125 µg/plate or higher. There was no increase in the number of revertants/plate.

**Genetic toxicity in vivo**

In a micronucleus assay performed according to the first version of OECD guideline 474 male and female mice were dosed by gavage with 1,600 mg PCOC/kg in 10 ml of peanut oil, corresponding to the maximum tolerable dose. Bone marrow cells were harvested at 24, 48, and 72 hours post dosing. A significant (p<0.0007) increase (4-6 times) in the frequency of micronuclei was observed in the dosed animals at all harvesting times (Scantox, 1982f). It was noted that there was no clear evidence of a time-course for these effects. The incidences of micronucleated cells in treated animals were not particularly high compared to published data for untreated mice, while the incidence in the control group was lower than what would usually be expected. It was not possible to re-examine concurrent control data to obtain information on background rates, as the records are no longer available.

A new mouse micronucleus assay was performed in 1997 according to current guidelines (EEC, 29 December 1992, Official Journal of the European Communities No. L358B: Methods for determination of toxicity, B12: Mutagenicity (Micronucleus test) p. 124), including the OECD guideline revision (OECD 1996) recommending use of aqueous suspending agents for poorly soluble substances. The test substance, 99.3% pure PCOC consisting of 50% of current production lots from each of the two U.K. producers was suspended in aqueous 0.5% gum tragacanth. A preliminary toxicity test indicated that in this vehicle, the maximum dose, which did not induce excessive lethality, was approximately 400 mg/kg. For the Micronucleus test, groups of 5 male and 5 female mice were dosed by gavage with 20 ml/kg suspensions of test substance corresponding to 100, 200 and 400 mg/kg body weight of PCOC, using the vehicle alone as the negative, and Mitomycin C as the positive control.

Severe lethargy was noted shortly after dosing at 400 mg/kg. One female in the high dose group died, and was replaced by another female from the concurrently treated satellite group. No
adverse clinical signs were observed for the positive or negative control groups during the duration of the test.

Bone marrow samples were examined (1,000 erythrocytes per smear) after 24 hours and 48 hours and did not show any substantial increase in the incidence of micronucleated immature erythrocytes or decrease in the proportion of immature erythrocytes. It was concluded that PCOC did not show any evidence of causing chromosome damage or bone marrow cell toxicity in this test. The positive control caused highly significant (P<0.001) increases in the number of micronucleated immature erythrocytes at both 24 and 48 hours. Results for PCOC treated and control animals were within the expected range for unaffected mice based on published information and laboratory control data (Huntingdon, 1997).

While cytotoxic effects were not seen in the bone marrow, there is little to suggest that PCOC would not be absorbed, or would break down prior to reaching this site. Clear evidence of leucopenia seen in the two repeat-dose studies is highly suggestive bone marrow effects. In vivo mutagenicity studies of the meta isomer of chlorocresol (4-Chloro-3-methyl phenol, Cas. No. 59-50-7) showed a similar pattern, with no change in the observed PCE/NCE ratio and no clastogenic activity (mouse micronucleus test, oral, 200 and 400 mg/kg, 24 hours: mouse micronucleus test, single i.p. injection of 125 mg/kg - 10 % mortality - investigations at 24, 48 and 72 hours post dosing, stat. significant response in cyclophosphamide control) (BUA, 1993 - U.S. EPA, 1997).

Conclusion on mutagenicity

PCOC was negative in 3 Ames tests, equivocal in one, and negative in repeat tests of the equivocal strain. An oral mouse micronucleus performed in 1982 according to the first OECD guidelines was positive. A repeat of this test in 1997 using modern guideline recommendations and possibly a more suitable test vehicle was clearly negative. Using the best available data PCOC cannot be considered a mutagen.

4.1.2.6 Carcinogenicity

For 4-chloro-o-cresol (PCOC) no studies in humans or animals are available.

Human data on phenoxy herbicide production

A cohort study of workers employed in manufacturing of phenoxy herbicides, primarily MCPA, in Denmark before 1982 was carried out. The study seems to support the Swedish observation of an increased risk of soft tissue sarcomas following exposure to phenoxy herbicides. The purpose of the study was to shed further light on the potential carcinogenic effect indicated by a Swedish case control study of the 2,4-dichlorophenol and 4-chloro-o-cresol based phenoxy herbicides unlikely to be contaminated with 2,3,7,8-tetrachlordibenzo-p-dioxin.

Cancer cases were identified by linkage with the National Cancer Register. Special attention was given to soft tissue sarcomas and malignant lymphomas. Five cases of soft tissue sarcomas were observed among male employees in contrast to 1.84 expected cases, RR=2.72, CI95 =0.88-6.34 (Lynge, 1985).

An update of the above mentioned cohort study (Lynge, 1993) adds data for the period 1983-87. Based on small numbers the study adds to the evidence for a possible association between phenoxy herbicide exposure and risk of soft tissue sarcomas. There are, however, a number of possible confounders in these studies, and the overall cancer incidence of workers employed in
manufacturing and packaging of phenoxy herbicides was the same as for the Danish population (66 observed v. 64.27 expected, SIR 1.0, 95% CI 0.8-1.3).

IARC (1987) concluded that the chlorophenoxy herbicides should be placed in group 2B because of limited evidence for carcinogenicity to humans and because no adequate published data were available on the carcinogenicity of MCPA to animals.

While PCOC is a breakdown product and possible contaminant of (impurity in) MCPA, implications of these finding for the effects of PCOC itself can remain only speculative.

4.1.2.7 Toxicity for reproduction

In a combined repeated dose/reproduction screening test carried out according to OECD draft guideline 422 (Hansen, 1996) groups of 10 male and 10 female rats were given 0, 50, 200, or 600 mg PCOC/kg in soybean oil by gavage for two weeks prior to mating and until day 20 of gestation. No toxic effects on any reproductive or developmental parameters were observed, resulting in a no effect level for these endpoints of 600 mg/kg.

In a recently conducted in vitro assay for estrogenic effects using human breast cancer cells (Körner et al., 1996; Körner et al., 1997), PCOC was found to express activity corresponding to $1 \times 10^{-6}$ that of 17-β-Estradiol. It is difficult to evaluate what possible influence this might have on reproductive parameters.

4.1.3 Risk characterisation

4.1.3.1 General aspects

Major effects of possible concern are corrosivity, acute inhalation toxicity and repeat dose toxicity. Direct exposure is possible for production workers, and indirect exposure for workers, consumers and the general population.

The human risk assessment according to the pesticide scenario is not conducted based on a decision at the EU Technical meeting on risk assessment of existing substances (TM III, Nov.1996) referring to this part of the risk assessment being conducted by DGVI working group on risk assessment of plant protection products.

4.1.3.2 Workers

Production facility workers

(See 4.1.1.2 for exposure levels).

Realistic worst-case exposure is likely to be of the order of 0.7 mg/kg/day according to information provided by one of the producers (A.H. Marks, 1997b).

* Herbicide application workers (see 4.1.1.1. for exposure levels)

The exposure PCOC as a 1% impurity in MCPA can be in the order 0.28 mg/kg/day (agricultural) or 0.35 mg/kg/day (municipal weed control). For the end-points irritation/corrosivity the concentration is below the level of concern. For repeat dose toxicity this should not present a major health problem, e.g. for repeat dose toxicity the margin of safety based on a NOAEL of 200 mg/kg/day is 200/0.35 = 571. The margin of safety for effects is in the order of 300-600, thus workplace exposure to PCOC does not seem to present a major risk.
Repeat dose toxicity is not likely to present a major health problem. The margin of safety based on a NOAEL of 200 mg/kg/day (slight effect on liver enzyme (ALAT), haemoglobin conc.) is 200/0.7 = 285.

Also the end-point irritation/corrosivity does not seem to cause any health concern. In situations with possible contact with the substance safety measures, such as wearing appropriate PPE, are prescribed in the existing productions. Further, health surveillance programmes including examination of the respiratory function have been undertaken for several years. According to the medical reports submitted by the producers no significant effects on the respiratory system have been observed.

It should be stressed that direct skin contact with PCOC can lead to burns and/or irritation, but that adequate warning of this effect is given by the manufacturers classification (R-35) and that the wearing of appropriate PPE is compulsory when exposure at the workplace is possible [according to the UK Control Of Substances Hazardous to Health regulation - referred to in (Marks, 1997a)].

Conclusion of the risk assessment for workers

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

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

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

4.1.3.3 Consumers

For this group exposure may be in the order of 0.07 mg/kg for each event corresponding to a daily dose of $9.6 \times 10^{-4}$ mg/kg/day (see 4.1.1.3 for further details). With a NOAEL for repeat dose toxicity of 200 mg/kg/day the margin of safety is at least 20,000 for each single event.

Conclusion of the risk assessment for consumers

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

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

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

4.1.3.4 Humans exposed indirectly via the environment

The exposure of man indirectly via the environment through herbicide use is likely to be $10 \cdot 10^{-8}$ mg/kg/day via human intake media. Regional exposure resulting from production of PCOC is estimated as being low ($1.3 \cdot 10^{-5}$ mg/kg/day), while local indirect exposure estimates of $1.2 \cdot 10^{-4}$ mg/kg/day does not give rise to immediate concern with regard to corrosivity or repeat dose toxicity.

Conclusions of the risk assessment for humans exposed indirectly via the environment
4.1.3.5 Combined exposure

On the basis of the conclusion made in 4.1.1.2 and 4.1.1.3 a consumer, who also works at a production site and sprays garden herbicides, will receive the highest dose of PCOC during work and during gardening activities of 1.05 mg/kg/day. The dose received indirectly via the environment is low compared to this, $1.2 \times 10^{-4}$ mg/kg/day, but would occur regularly. A margin of safety of 190 (200 mg/kg / 1.05 mg/kg) would not seem to present undue risk.

Conclusions of the risk assessment for man during combined exposure

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

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

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

4.2 HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

4.2.1 Exposure assessment

The substance PCOC gives no reason for concern in relation to the following physical-chemical properties. The tests performed all gave or were expected to give negative results.

4.2.2 Effects assessment: Hazard identification

4.2.2.1 Explosivity

Explosive properties have not been tested. No reports of explosive properties were found in the available literature, nor does the chemical structure contain any elements associated with explosivity.

4.2.2.2 Flammability

The substance does not burn according to methods used (EF 3.10 and EF 3.10 mod.), nor is it flammable in contact with water. (Quist Laboratory, 1983).
4.2.2.3 Oxidizing potential

The substance was classified as non oxidizing according to the test method from the working group PC II Annex V EEC/831/79, sixth amendment of Dir. 67/548/EEC. (Dantest, 1983).

4.2.3 Risk characterisation

It is not likely that any of the above mentioned adverse effects should occur under the conditions mentioned.
The documentation varies from original studies according to OECD test guidelines with GLP to literature references of varying quality. 4-Chloro-2-methylphenol (PCOC) is used in the industry as an intermediate in the synthesis of the phenoxy herbicides MCPA, MCPB and mecoprop (MCPP). From the industrial production, processing and formulation PCOC is emitted to air and wastewater. The produced pesticides contain PCOC as impurity (normally <1%, 0.5% estimated as realistic worst case). The use of the pesticides in the agriculture as herbicides results in exposure to soil of PCOC as an impurity and degradation product.

As MCPA is transformed to PCOC, and PCOC has a high vapour pressure, the atmosphere will receive a contribution from application of the above-mentioned pesticides. PCOC has a low to medium adsorption and may be considered mobile in some soils.

PCOC is according to an experiment primarily degradable by photolysis in clean water with a half-life of 4 days. However, a re-estimation of photolysis to typical EU surface water resulted in an estimated photolytic degradation half-life of 300-700 days and therefore photolysis is considered negligible. The available biodegradation data are somewhat conflicting but based on a judgement of the balance of evidence the “realistic worst case” aerobic biodegradation half-life of PCOC in soil is estimated to be 21 days, whereas no biodegradation has been found under anaerobic conditions. The aerobic biodegradation half-life in surface waters is also estimated to be 21 days. The estimated half-life in biological wastewater treatment plants is 0.7 hour resulting in an estimated removal of 88% which is in general accordance with simple mass balance estimations from one of the main manufacturers sites. The substance is therefore considered to be readily biodegradable (borderline).

PCOC has been found in water, soil, air and groundwater. In water, PCOC occurs mainly around emission sources; in air, near fields applied with MCPA or MCPP; and in soil and biota, after the application of the herbicides. The findings in groundwater are assumed to be the result of mobility and reduced degradation under anaerobic conditions.

The exposure assessment is primarily based on monitoring data from the two main manufacturing sites where all production and all processing of PCOC take place and where approximately 60% of the production volume is formulated. A worst-case environmental exposure scenario for a separate formulation site is included in the risk assessment.

The emissions to surface water from production sites are local and the risk assessment based on monitoring data (C_{STP + influent} and actual dilution in STPs) and TGD default environmental exposure assessment for formulation site where 10% of the production volume of phenoxy acids is formulated. Because only the STPs receiving wastewater from one of the production sites and the formulation sites are using sludge application to soil, the sludge application is considered local.

PCOC is very toxic to aquatic organisms. The acute toxicity to fish LC_{50} (96h) was observed to be about 2.3-6.6 mg/l. The EC_{50} (48h) to daphnids were 0.29-1.0 mg/l. The EC_{50} (96h) to algae was 8.2 mg/l and the EC_{10} (96h) was 0.89 mg/l. The long term toxic effects were observed in fish to have a NOEC(28d) of 0.5 mg/l and the Daphnia reproduction NOEC(21d) was 0.55 mg/l.

The PEC_{local water}/PNEC_{aquatic organism} relationship is <1. Model calculation using EUSES version 1.0 supports the assumption of no risks for adverse effects in the aquatic environment and for the microorganisms of STPs.
There are no data available on the terrestrial toxicity. The equilibrium partitioning method is applied as a conservative calculation, comparing $\text{PEC}_{\text{soil}}$, $\text{porewater}$ with $\text{PNEC}_{\text{aquatic, organisms}}$: $\frac{\text{PEC}_{\text{soil}}}{\text{PNEC}} < 1$.

PEC$_{\text{air}}$: There are no effect data present and no relation $\text{PEC}/\text{PNEC}$ can be calculated.

PCOC has a bioaccumulation potential based on $\log K_{\text{ow}}$ 3.09, but BCF found in fish was low ($\leq 30$). The risk characterisation of secondary poisoning is therefore not performed.

The substance is considered to be of no concern to aquatic organisms and microorganisms of STPs, and no further information on environmental release from production and formulation facilities is required.

No current evidence was found for the use of PCOC as such in products, although it may formerly have been employed as a disinfectant. Direct exposure is therefore likely to be restricted to those involved in the manufacture and handling of PCOC, and in conjunction with its use in the manufacture of phenoxy herbicides. Based on limited information, exposures in the range of 0.02 - 0.7 mg/kg/day are estimated for these activities.

The main exposure of human beings to PCOC is likely to be via production, or use of phenoxy herbicides, which may contain it as an impurity ($\leq 1\%$), or as a breakdown product following exposure of herbicides to sunlight, or to their metabolic transformation to the substance. It is difficult to quantify exposure occurring through transformation, but this is assumed to be less than 1%. During production, a realistic worst-case exposure of 0.7 mg/kg/day is indicated. In conjunction with agricultural application of herbicides, a worst-case estimate of exposure to PCOC of 0.28 mg/kg/day is obtained. Municipal gardeners may be exposed to higher levels with an estimate of 0.35 mg/kg/day suggested as a realistic worst case.

Similarly, some consumer exposure should also be expected, as the same herbicides can be used in lawn treatment and similar gardening activities. While no detailed information was found on such exposures, it may be amount to 0.07 mg/kg per event. Assuming a realistic worst case of five events per year, the total yearly dose of PCOC would be 0.35 mg/kg/year corresponding to $9.6 \cdot 10^{-4}$ mg/kg/day.

Indirect exposure via the environment resulting from partitioning into air/water/soil and biomagnification in food sources is low at a regional level, combined secondary exposure estimate being in the range of $1.40 \cdot 10^{-5}$ mg/kg/day of PCOC. Local indirect exposure estimates are about $1.2 \cdot 10^{-4}$ mg/kg/day.

The acute toxicity of PCOC (LD$_{50}$ oral rat 2650-3196 mg/kg, LD$_{50}$ dermal rat 2240 mg/kg, LC$_{50}$ inhal. rat 4h 0.9mg/l or $>30$ mg/l) does not give rise to immediate concern, particularly considering that the substance (crystalline needles) is unlikely to form aerosols or dusts, and that PPE is mandated during handling of the substance.

PCOC is corrosive in high concentrations, and has been assigned risk phrase R-35 by the manufacturers, which should provide adequate warning to those handling it in industrial settings. No consumer exposure is expected at concentrations, which could approach that required for corrosivity. No sensitisation was observed in a Guinea pig maximization test and no case studies indicating sensitisation of persons handling the substance were found.

There were no effects on reproduction according to OECD screening test 422 at doses of up to 600 mg/kg for a total of 40 days.
In 28-day repeat dose studies in rats, the best NOAEL appears to be 200 mg/kg, with a LOAEL of 800 mg/kg where salivation after dosing and ruffled fur was seen in some animals. At this dose, levels of serum alanine-aminotransferase were increased in males, and effects were seen on blood parameters (reduced thromboplastin times, reduction of leucocyte and erythrocyte counts). Liver weights in females were increased, but no histopathological changes were seen in this, or any other organs examined. Decreased adrenal weights were also seen in females at 200 mg/kg and above, but were unaccompanied by histopathological changes.

PCOC has not been investigated for carcinogenicity. Two older tests were positive for mutagenicity, one in vivo (mouse micronucleus test) and one in vitro in a single strain (TA97) of Salmonella in the Ames test (while showing no activity in other strains in a number of separate tests). Repeated testing with TA97 gave unequivocally negative results. A repeat of the micronucleus test according to current guidelines also gave clearly negative results. On the balance, it is not felt that there is evidence for PCOC being a mutagen.

The estimated human local indirect exposure of $1.2 \cdot 10^{-4}$ mg/kg/day is well below the repeat dose toxicity (NOAEL 200 mg/kg/day).

For the population with the highest potential exposure (production workers assuming inhalation exposure at 5 mg/m$^3$ for eight hours) a margin of safety of 285 (200 mg/kg/0.7 mg/kg/day) is obtained with regard to the repeat dose NOAEL. For agricultural workers engaged in spraying phenoxy herbicides the ratio is 200 mg/kg/0.28 mg/kg, or 714.

For municipal gardeners (0.35 mg/kg/day) a margin of safety of 571 is obtained. Consumers may be exposed to 0.07 mg/kg/day once, or a few times yearly. All other exposure scenarios result in much higher margins of safety.


Dantest (1983). Dantest study, prepared for the Danish-EPA.


Nufarm UK Ltd. (1997). Environmental release of 4-chloro-2-methylphenol (PCOC) during production and synthesis of phenoxy acid herbicides. **Confidential report**, Nufarm UK Ltd., Belvedere, UK. **Submitted to the CAs**.

Nufarm UK Ltd. (1997b). Further information on environmental release of 4-chloro-2-methylphenol (PCOC) as requested by the European Chemicals Bureau. **Confidential report**, Nufarm UK Ltd., Belvedere, UK, October 1997. **Submitted to the CAs**.


Qvist Lab. (1983). Test report from Qvist Laboratories, Denmark, prepared for the Danish-EPA.


<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>ADI</td>
<td>Acceptable Daily Intake</td>
</tr>
<tr>
<td>AF</td>
<td>Assessment Factor</td>
</tr>
<tr>
<td>ASTM</td>
<td>American Society for Testing and Materials</td>
</tr>
<tr>
<td>ATP</td>
<td>Adaptation to Technical Progress</td>
</tr>
<tr>
<td>AUC</td>
<td>Area Under The Curve</td>
</tr>
<tr>
<td>B</td>
<td>Bioaccumulation</td>
</tr>
<tr>
<td>BBA</td>
<td>Biologische Bundesanstalt für Land- und Forstwirtschaft</td>
</tr>
<tr>
<td>BCF</td>
<td>Bioconcentration Factor</td>
</tr>
<tr>
<td>BMC</td>
<td>Benchmark Concentration</td>
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<tr>
<td>BMD</td>
<td>Benchmark Dose</td>
</tr>
<tr>
<td>BMF</td>
<td>Biomagnification Factor</td>
</tr>
<tr>
<td>bw</td>
<td>body weight / Bw, b.w.</td>
</tr>
<tr>
<td>C</td>
<td>Corrosive (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)</td>
</tr>
<tr>
<td>CA</td>
<td>Chromosome Aberration</td>
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<td>CA</td>
<td>Competent Authority</td>
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<td>CAS</td>
<td>Chemical Abstract Services</td>
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<td>CEC</td>
<td>Commission of the European Communities</td>
</tr>
<tr>
<td>CEN</td>
<td>European Standards Organisation / European Committee for Normalisation</td>
</tr>
<tr>
<td>CMR</td>
<td>Carcinogenic, Mutagenic and toxic to Reproduction</td>
</tr>
<tr>
<td>CNS</td>
<td>Central Nervous System</td>
</tr>
<tr>
<td>COD</td>
<td>Chemical Oxygen Demand</td>
</tr>
<tr>
<td>CSTEE</td>
<td>Scientific Committee for Toxicity, Ecotoxicity and the Environment (DG SANCO)</td>
</tr>
<tr>
<td>CT&lt;sub&gt;50&lt;/sub&gt;</td>
<td>Clearance Time, elimination or depuration expressed as half-life</td>
</tr>
<tr>
<td>d.wt</td>
<td>dry weight / dw</td>
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<tr>
<td>dfi</td>
<td>daily food intake</td>
</tr>
<tr>
<td>DG</td>
<td>Directorate General</td>
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<tr>
<td>DIN</td>
<td>Deutsche Industrie Norm (German norm)</td>
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<tr>
<td>DNA</td>
<td>DeoxyriboNucleic Acid</td>
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<tr>
<td>DOC</td>
<td>Dissolved Organic Carbon</td>
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<tr>
<td>DT50</td>
<td>Degradation half-life or period required for 50 percent dissipation / degradation</td>
</tr>
<tr>
<td>DT90</td>
<td>Period required for 50 percent dissipation / degradation</td>
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<td>EASE</td>
<td>Estimation and Assessment of Substance Exposure Physico-chemical properties [Model]</td>
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<td>EbC50</td>
<td>Effect Concentration measured as 50% reduction in biomass growth in algae tests</td>
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<tr>
<td>Acronym</td>
<td>Definition</td>
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<tr>
<td>EC</td>
<td>European Communities</td>
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<tr>
<td>EC10</td>
<td>Effect Concentration measured as 10% effect</td>
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<td>median Effect Concentration</td>
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<tr>
<td>ECB</td>
<td>European Chemicals Bureau</td>
</tr>
<tr>
<td>ECETOC</td>
<td>European Centre for Ecotoxicology and Toxicology of Chemicals</td>
</tr>
<tr>
<td>ECVAM</td>
<td>European Centre for the Validation of Alternative Methods</td>
</tr>
<tr>
<td>EDC</td>
<td>Endocrine Disrupting Chemical</td>
</tr>
<tr>
<td>EEC</td>
<td>European Economic Communities</td>
</tr>
<tr>
<td>EINECS</td>
<td>European Inventory of Existing Commercial Chemical Substances</td>
</tr>
<tr>
<td>ELINCS</td>
<td>European List of New Chemical Substances</td>
</tr>
<tr>
<td>EN</td>
<td>European Norm</td>
</tr>
<tr>
<td>EPA</td>
<td>Environmental Protection Agency (USA)</td>
</tr>
<tr>
<td>ErC50</td>
<td>Effect Concentration measured as 50% reduction in growth rate in algae tests</td>
</tr>
<tr>
<td>ESD</td>
<td>Emission Scenario Document</td>
</tr>
<tr>
<td>EU</td>
<td>European Union</td>
</tr>
<tr>
<td>EUSES</td>
<td>European Union System for the Evaluation of Substances [software tool in support of the Technical Guidance Document on risk assessment]</td>
</tr>
<tr>
<td>F(+)</td>
<td>(Highly) flammable (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)</td>
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<tr>
<td>FAO</td>
<td>Food and Agriculture Organisation of the United Nations</td>
</tr>
<tr>
<td>FELS</td>
<td>Fish Early Life Stage</td>
</tr>
<tr>
<td>GLP</td>
<td>Good Laboratory Practice</td>
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<tr>
<td>HEDSET</td>
<td>EC/OECD Harmonised Electronic Data Set (for data collection of existing substances)</td>
</tr>
<tr>
<td>HELCOM</td>
<td>Helsinki Commission - Baltic Marine Environment Protection Commission</td>
</tr>
<tr>
<td>HPLC</td>
<td>High Pressure Liquid Chromatography</td>
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<tr>
<td>HPVC</td>
<td>High Production Volume Chemical (&gt; 1000 t/a)</td>
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<tr>
<td>IARC</td>
<td>International Agency for Research on Cancer</td>
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<tr>
<td>IC</td>
<td>Industrial Category</td>
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<tr>
<td>IC50</td>
<td>median Immobilisation Concentration or median Inhibitory Concentration</td>
</tr>
<tr>
<td>ILO</td>
<td>International Labour Organisation</td>
</tr>
<tr>
<td>IPCS</td>
<td>International Programme on Chemical Safety</td>
</tr>
<tr>
<td>ISO</td>
<td>International Organisation for Standardisation</td>
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<tr>
<td>IUCLID</td>
<td>International Uniform Chemical Information Database (existing substances)</td>
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<tr>
<td>IUPAC</td>
<td>International Union for Pure and Applied Chemistry</td>
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<tr>
<td>JEFCA</td>
<td>Joint FAO/WHO Expert Committee on Food Additives</td>
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<td>JMPR</td>
<td>Joint FAO/WHO Meeting on Pesticide Residues</td>
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<tr>
<td>Koc</td>
<td>organic carbon normalised distribution coefficient</td>
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<tr>
<td>Kow</td>
<td>octanol/water partition coefficient</td>
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<td>Abbreviation</td>
<td>Description</td>
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<tr>
<td>Kp</td>
<td>solids-water partition coefficient</td>
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<td>L(E)C50</td>
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<td>LAEL</td>
<td>Lowest Adverse Effect Level</td>
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<td>LD50</td>
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<td>Local Exhaust Ventilation</td>
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<td>Local Lymph Node Assay</td>
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<td>Lowest Observed Adverse Effect Level</td>
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<td>Maximum Acceptable Toxic Concentration</td>
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<td>Ministry of International Trade and Industry, Japan</td>
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<td>MOE</td>
<td>Margin of Exposure</td>
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<td>MOS</td>
<td>Margin of Safety</td>
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<tr>
<td>MW</td>
<td>Molecular Weight</td>
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<tr>
<td>N</td>
<td>Dangerous for the environment (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)</td>
</tr>
<tr>
<td>NAEL</td>
<td>No Adverse Effect Level</td>
</tr>
<tr>
<td>NOAEL</td>
<td>No Observed Adverse Effect Level</td>
</tr>
<tr>
<td>NOEL</td>
<td>No Observed Effect Level</td>
</tr>
<tr>
<td>NOEC</td>
<td>No Observed Effect Concentration</td>
</tr>
<tr>
<td>NTP</td>
<td>National Toxicology Program (USA)</td>
</tr>
<tr>
<td>O</td>
<td>Oxidizing (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)</td>
</tr>
<tr>
<td>OECD</td>
<td>Organisation for Economic Cooperation and Development</td>
</tr>
<tr>
<td>OEL</td>
<td>Occupational Exposure Limit</td>
</tr>
<tr>
<td>OJ</td>
<td>Official Journal</td>
</tr>
<tr>
<td>OSPAR</td>
<td>Oslo and Paris Convention for the protection of the marine environment of the Northeast Atlantic</td>
</tr>
<tr>
<td>P</td>
<td>Persistent</td>
</tr>
<tr>
<td>PBT</td>
<td>Persistent, Bioaccumulative and Toxic</td>
</tr>
<tr>
<td>PBPK</td>
<td>Physiologically Based PharmacoKinetic modelling</td>
</tr>
<tr>
<td>PBTK</td>
<td>Physiologically Based ToxicoKinetic modelling</td>
</tr>
<tr>
<td>PEC</td>
<td>Predicted Environmental Concentration</td>
</tr>
</tbody>
</table>
| pH           | logarithm (to the base 10) (of the hydrogen ion concentration \(\text{[H}^+\])}
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>pKa</td>
<td>logarithm (to the base 10) of the acid dissociation constant</td>
</tr>
<tr>
<td>pKb</td>
<td>logarithm (to the base 10) of the base dissociation constant</td>
</tr>
<tr>
<td>PNEC</td>
<td>Predicted No Effect Concentration</td>
</tr>
<tr>
<td>POP</td>
<td>Persistent Organic Pollutant</td>
</tr>
<tr>
<td>PPE</td>
<td>Personal Protective Equipment</td>
</tr>
<tr>
<td>QSAR</td>
<td>(Quantitative) Structure-Activity Relationship</td>
</tr>
<tr>
<td>R phrases</td>
<td>Risk phrases according to Annex III of Directive 67/548/EEC</td>
</tr>
<tr>
<td>RAR</td>
<td>Risk Assessment Report</td>
</tr>
<tr>
<td>RC</td>
<td>Risk Characterisation</td>
</tr>
<tr>
<td>RfC</td>
<td>Reference Concentration</td>
</tr>
<tr>
<td>RfD</td>
<td>Reference Dose</td>
</tr>
<tr>
<td>RNA</td>
<td>RiboNucleic Acid</td>
</tr>
<tr>
<td>RPE</td>
<td>Respiratory Protective Equipment</td>
</tr>
<tr>
<td>RWC</td>
<td>Reasonable Worst Case</td>
</tr>
<tr>
<td>S phrases</td>
<td>Safety phrases according to Annex III of Directive 67/548/EEC</td>
</tr>
<tr>
<td>SAR</td>
<td>Structure-Activity Relationships</td>
</tr>
<tr>
<td>SBR</td>
<td>Standardised birth ratio</td>
</tr>
<tr>
<td>SCE</td>
<td>Sister Chromatic Exchange</td>
</tr>
<tr>
<td>SDS</td>
<td>Safety Data Sheet</td>
</tr>
<tr>
<td>SETAC</td>
<td>Society of Environmental Toxicology And Chemistry</td>
</tr>
<tr>
<td>SNIF</td>
<td>Summary Notification Interchange Format (new substances)</td>
</tr>
<tr>
<td>SSD</td>
<td>Species Sensitivity Distribution</td>
</tr>
<tr>
<td>STP</td>
<td>Sewage Treatment Plant</td>
</tr>
<tr>
<td>T(+)</td>
<td>(Very) Toxic (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)</td>
</tr>
<tr>
<td>TDI</td>
<td>Tolerable Daily Intake</td>
</tr>
<tr>
<td>TG</td>
<td>Test Guideline</td>
</tr>
<tr>
<td>TGD</td>
<td>Technical Guidance Document (^1)</td>
</tr>
<tr>
<td>TNG</td>
<td>Technical Notes for Guidance (for Biocides)</td>
</tr>
<tr>
<td>TNO</td>
<td>The Netherlands Organisation for Applied Scientific Research</td>
</tr>
<tr>
<td>UC</td>
<td>Use Category</td>
</tr>
<tr>
<td>UDS</td>
<td>Unscheduled DNA Synthesis</td>
</tr>
<tr>
<td>UN</td>
<td>United Nations</td>
</tr>
<tr>
<td>UNEP</td>
<td>United Nations Environment Programme</td>
</tr>
<tr>
<td>US EPA</td>
<td>Environmental Protection Agency, USA</td>
</tr>
<tr>
<td>UV</td>
<td>Ultraviolet Region of Spectrum</td>
</tr>
<tr>
<td>UV-CVB</td>
<td>Unknown or Variable composition, Complex reaction products of Biological material</td>
</tr>
</tbody>
</table>
vB  very Bioaccumulative
vP  very Persistent
vPvB very Persistent and very Bioaccumulative
v/v  volume per volume ratio
w/w  weight per weight ratio
WHO World Health Organization
WWTP Waste Water Treatment Plant
Xn  Harmful (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
Xi  Irritant (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
Appendix A  Photolysis of PCOC in surface water

The effect of sunlight irradiation on aqueous solution of PCOC (100 µg/l = 0.7 mM) was studied. The PCOC-solution was sealed in a flask to prevent volatilisation. The exposure to "summer sunlight" (not further defined or specified) in California resulted in a stated photolytic half-life of 2.5 days (which from the presented graph rather seems to be 4 days) (Crosby & Bowers, 1985). It is below assumed that the experimental data (T½ = 4 days) were obtained using quartz flask, i.e. at the surface of pure water at 40° latitude in the summer.


Like hydrolysis, photolysis only results in primarily degradation (transformation) of substance. Furthermore, extrapolation of photolytic half-lives from one set of conditions to another is generally very uncertain. A substantial part of the uncertainty may be due to lack of information of the conditions under which the half-life was measured. On the other hand it is rather easy to define the conditions of the environment of the "standard EU region" (cf. TGD Sept.95). However the representativeness of such a "standard EU" environment for the whole EU may be questioned (e.g. how to define the "standard" water body as regards water depth and content of dissolved and suspended matter? The latitude range of the EU covers the latitudes from 35° to 70° giving a tremendous difference in the incoming sunlight intensity in the relevant wave length interval). Below, it is assumed that the standard EU environment is at the latitude 50°. As regard the content of dissolved and suspended matter and the depth of water in the EU "standard environment" cf. below.

Two types of extrapolation guidance have been considered:
- TGD (Sept.1995 ver.)
- TETRATECH (1983)

According to TGD (Sept.95):

The extrapolation methods recommended in TGD require use of computer models, which were not available.

According to TETRATECH:

Photolytic rate constant $K_{\text{photo}} = K_{\text{photo,0}} \cdot (I/I_0) \cdot 1/K \cdot Z$

where

$K_{\text{photo,0}} = $ Experimentally determined photolytic rate constant at surface of pure water

$I_0$: light intensity at the conditions under consideration

$I$: light intensity at the surface of pure water at which $K_{\text{photo,0}}$ was measured

$K$: light reduction (diffuse light attenuation coefficient) in water = $R/Z_{sd}$,

$Z_{sd}$: Secchi disc depth (m)

$R$: proportionality constant 1.44-1.7 for visible light (400nm to 800nm).
In the middle UV portion of the spectrum (i.e. near 312 nm) R = 9.15

Z: water depth (m)

The experimentally determined photolytic rate constant at the surface of pure water (summer, 40°C): T½ of 4 days, is calculated to be K_{\text{photo,CA}}: \ln(2)/4 (d^{-1}) = 0.17325 (d^{-1}), and Annual mean: I_{\text{EU}}: 460 langley/d (TETRATECH),

Winter mean: I_{\text{EU}}: 190 langley/d (TETRATECH)

Summer mean I_{\text{CA}}: 740 langley/d (TETRATECH)

In the table below extrapolations according to various Z_{sd} and Z of the "standard EU environment" for the whole year (upper four rows) and for the winter season (lower four rows, "realistic worst case" according to suggestion of draft TGD (sept.)) are presented.

<table>
<thead>
<tr>
<th>Z_{sd} m</th>
<th>K m^{-1}</th>
<th>Z</th>
<th>1/(K\cdot Z)</th>
<th>I_{\text{EU}} ly/d</th>
<th>K_{\text{photo,EU}} d^{-1}</th>
<th>T_{1/2photo,EU} days</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>9.15</td>
<td>1</td>
<td>0.1093</td>
<td>460</td>
<td>0.0118</td>
<td>58</td>
</tr>
<tr>
<td>0.5</td>
<td>18.3</td>
<td>1</td>
<td>0.0546</td>
<td>460</td>
<td>0.0059</td>
<td>117</td>
</tr>
<tr>
<td>0.3</td>
<td>30.5</td>
<td>1</td>
<td>0.0328</td>
<td>460</td>
<td>0.0035</td>
<td>198</td>
</tr>
<tr>
<td>0.6</td>
<td>15.3</td>
<td>3</td>
<td>0.0218</td>
<td>460</td>
<td>0.0023</td>
<td>301</td>
</tr>
<tr>
<td>1</td>
<td>9.15</td>
<td>1</td>
<td>0.1093</td>
<td>190</td>
<td>0.0049</td>
<td>141</td>
</tr>
<tr>
<td>0.5</td>
<td>18.3</td>
<td>1</td>
<td>0.0546</td>
<td>190</td>
<td>0.0024</td>
<td>289</td>
</tr>
<tr>
<td>0.3</td>
<td>30.5</td>
<td>1</td>
<td>0.0328</td>
<td>190</td>
<td>0.0015</td>
<td>475</td>
</tr>
<tr>
<td>0.6</td>
<td>15.3</td>
<td>3</td>
<td>0.0218</td>
<td>190</td>
<td>0.0009</td>
<td>715</td>
</tr>
</tbody>
</table>

Remark

The relevant dimensions of the "standard environment" employed in the regional model for calculation of PEC_{\text{water, regional}} for the EU is a water depth of 3 m and a concentration of suspended solids of 15 mg/l. The latter is equivalent with Z_{sd} = 0.6 m (pers. comm. O. Sortkær, Danish Environmental Research Inst.). Therefore it seems most reasonable to choose these values when extrapolating the experimental data on photolysis to photolytic degradation half-lives in EU surface waters. The estimated photolytic half-life in surface waters of the EU is therefore 301 days for the annual mean and 715 days for the winter season.

Based on the above estimations, the photolytic degradation of PCOC in surface waters of the EU is concluded to be negligible.
Appendix B  

**QSAR estimations on toxicity**

Comparison between QSAR predicted and experimental L(E)C$_{50}$-values for fish and daphnia using the preferred log K$_{ow}$ 3.09 and the log K$_{ow}$ 2.63 by Hansch et al. (1995).

The QSARs employed are those recommended in the TGD for polar narcotic acting substances. Recommended QSAR for polar narcotic acting substances on algae has not been agreed upon.

**Table B.1  Comparison between QSAR predicted and experimental L(E)C$_{50}$-values for fish and daphnia**

<table>
<thead>
<tr>
<th></th>
<th>QSAR L(E)C$_{50}$ (mg/l)</th>
<th>Experimental L(E)C$_{50}$ (mg/l)</th>
<th>Ratio QSAR/experimental</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Log K$_{ow}$ = 3.09</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fish</td>
<td>5</td>
<td>2.3</td>
<td>2.2</td>
</tr>
<tr>
<td>Daphnia</td>
<td>4</td>
<td>0.63 *</td>
<td>6.3</td>
</tr>
<tr>
<td><strong>Log K$_{ow}$ = 2.63</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fish</td>
<td>12</td>
<td>2.3</td>
<td>5.2</td>
</tr>
<tr>
<td>Daphnia</td>
<td>8</td>
<td>0.63 *</td>
<td>12.5</td>
</tr>
</tbody>
</table>

*Geometric mean of nominal acute toxicity by three independent authors.

The comparisons illustrate the reliability of the nominal experimental values may be invalid or that excess acute toxicity towards daphnids may occur. However, as the results are not used in the risk assessment, the validity is not further discussed.
Appendix C  TGD estimations on exposure

PEC\textsubscript{local, water} estimations

For illustrative purposes, estimations according to TGD are calculated below. The calculations of the predicted environmental concentrations are presented according to TGD, Appendix I, Tables A1.2, A2.1, A3.3 and B1.6, B2 and B3.2. Measured data from the two main production sites are included as average values to preserve confidentiality.

The calculation of PEC\textsubscript{local} for the aquatic compartment includes the calculation of the discharge concentration of STP to a water body, dilution effects and removal from the aqueous medium by adsorption to suspended matter.

The local emission during episode to the aquatic compartment, E\textsubscript{local, water}, is calculated by using the regional values in section 3.1.1.4 multiplied with the estimated fractions.

Fractions of total production volume used for local calculations:
- Production: 0.5
- Processing: 0.5
- Formulation: 0.5

"Days of emission" is according to TGD: 300 days/year. The calculation of the local concentration in untreated wastewater, C\textsubscript{local, influent}, is performed by the equation:

$$C_{\text{local, influent}} = \frac{E_{\text{local, water}} \times 10^6}{\text{EFFLUENT}_{\text{stp}}}$$

where the EFFLUENT\textsubscript{stp} is the capacity of the STP multiplied with the sewage flow per inhabitant (default 10000 \cdot 200 = 2 \cdot 10^6 l/d). For comparison the site specific EFFLUENT\textsubscript{stp} of 1.3 \cdot 10^8 l/d is included. Because this value is the lowest flow of the STPs receiving wastewater from the two known manufacturers, the resulting values are used in the further estimations.

| Table C.1  Concentration in wastewater before treatment in municipal STP |
|-----------------|-----------------|-----------------|-----------------|-----------------|
| Life cycle stage | Emission (t/y)   | Emission (kg/d) | C\textsubscript{site effluent}\textsuperscript{b} (mg/l) | C\textsubscript{STP influent} (mg/l) |
| Production      | 22.5            | 75.0            | 37.5            | 0.58            |
| Processing      | 3.75            | 12.5            | 6.3             | 0.10            |
| Formulation\textsuperscript{10} | 13.5            | 45.0            | 7.5             | 0.12            |
| Specific site   | 2.5\textsuperscript{a} | 7.8\textsuperscript{a} | 30\textsuperscript{b} | 0.004\textsuperscript{b} |

\textsuperscript{a} Monitoring data from an actual production site representing the average of two production plants where all three processes are included (cf. Confidential appendix, submitted to CAs)

\textsuperscript{b} Monitoring data, average value from two sites, 95% percentile

\textsuperscript{c} Dilution during entrance to STP with other wastewater, STP flow 130000 m\textsuperscript{3}/d

The concentration in the STP effluent is found by including the fraction of emission directed to water by STP:

\textsuperscript{10} The release estimations from the formulation performed outside the specific sites are calculated according to TGD using 10% of total production and a PCOC content of the formulation of 1% (worst case). The default local wastewater volume is according to TGD: 2000 m\textsuperscript{3}/d, and number of days of emission is according to TGD: 300.
C_{local, effluent} = C_{local, influent} \cdot F_{\text{stp, water}}

The EUSES model includes the model SimpleTreat 3.0 in the calculations and estimate 12% is directed to water (i.e.: 0.5% to air, 12.1% to water, 3.6% to sludge and 83.8% degraded, and a removal of 88%).

The local concentration in surface water is calculated according to the equation:

\[ C_{\text{local, water}} = C_{\text{local, effluent}} / (1 + K_{p(suspend)} \cdot \text{SUSP}_{\text{water}} \cdot 10^{-6}) \cdot D, \]

Where

\( K_{p(suspend)} = \) the solids-water partitioning coefficient of suspended matter = \( K_{oc} \cdot F_{ocsusp} = 401 \cdot 0.1 = 40.1 \text{ l/kg} \), and \( \text{SUSP}_{\text{water}} \) = the concentration of suspended matter in the river (default: 15 mg/l), and \( D = \) dilution factor (default 10). The dilution of EFFLUENT_{\text{STP}} to river based on river flows varies between 2.5 to 1700. But since unknown formulation sites are also included in the estimations the default value is used in the risk assessment.

The concentration at the regional scale (PEC_{regional, water}) is used as background concentration for the local scale. Therefore, these concentrations are summed for the predicted environmental concentration in surface water during episode and in the annual average:

\[ \text{PEC}_{\text{local, water}} = C_{\text{local, water}} + \text{PEC}_{\text{regional, water}} \]

\[ \text{PEC}_{\text{local, water,ann}} = C_{\text{local, water, ann}} + \text{PEC}_{\text{regional, water}} \]

**Table C.2** Predicted environmental local concentration in surface water during episode (PEC_{local, water}).

<table>
<thead>
<tr>
<th>Life cycle stage</th>
<th>( C_{\text{local, effluent}} ) (mg/l)</th>
<th>( C_{\text{local, water}} ) (mg/l)</th>
<th>( \text{PEC}_{\text{local, water}}^* ) (mg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Production</td>
<td>0.070</td>
<td>0.0070</td>
<td>0.0071</td>
</tr>
<tr>
<td>Processing</td>
<td>0.012</td>
<td>0.0012</td>
<td>0.0013</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.042</td>
<td>0.0042</td>
<td>0.0043</td>
</tr>
<tr>
<td>Site specific measurements</td>
<td>0.004</td>
<td>0.0036</td>
<td>0.0038</td>
</tr>
</tbody>
</table>

*Including \( \text{PEC}_{\text{regional, water}}^* \): 0.000169 mg/l

**Table C.3** The predicted annual average concentration in surface water (PEC_{local, water,ann}).

<table>
<thead>
<tr>
<th>Life cycle stage</th>
<th>( C_{\text{local, water}} ) (mg/l)</th>
<th>( C_{\text{local, water, ann}} ) (mg/l)</th>
<th>( \text{PEC}_{\text{local, water, ann}}^* ) (mg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Production</td>
<td>0.0070</td>
<td>0.0057</td>
<td>0.0059</td>
</tr>
<tr>
<td>Processing</td>
<td>0.0012</td>
<td>0.0010</td>
<td>0.0011</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.0042</td>
<td>0.0034</td>
<td>0.0036</td>
</tr>
<tr>
<td>Specific site **</td>
<td>0.0036</td>
<td>0.0036</td>
<td>0.0037</td>
</tr>
</tbody>
</table>

* Including \( \text{PEC}_{\text{regional, water}}^* \): 0.000169 mg/l

**Based on monitoring data**
Calculation of $\text{PEC}_{\text{local}}$ for sediment

The local concentration in sediments during emission episode is calculated according to TGD.

The concentration in the bulk sediment is derived from the corresponding water body concentration, assuming a thermodynamical partition equilibrium:

$$\text{PEC}_{\text{local, sed}} = \left( \frac{K_{\text{susp, water}}}{\text{RHO}_{\text{susp}}} \right) \cdot \text{PEC}_{\text{local, water}} \cdot 1000$$

$K_{\text{susp, water}}$ = the suspended matter partitioning coefficient = 10.9
$\text{RHO}_{\text{susp}}$ = bulk density of (wet) suspended matter = 1150 kg/m$^3$

<table>
<thead>
<tr>
<th>Life cycle stage</th>
<th>$\text{PEC}_{\text{local, water}}$ (mg/l)</th>
<th>$\text{PEC}_{\text{local, sed}}$ (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Production</td>
<td>0.0071</td>
<td>0.0676</td>
</tr>
<tr>
<td>Processing</td>
<td>0.0013</td>
<td>0.0126</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.0043</td>
<td>0.0412</td>
</tr>
<tr>
<td>Specific site*</td>
<td>0.0038</td>
<td>0.038</td>
</tr>
</tbody>
</table>

*Estimated, using monitoring data

Atmospheric exposure estimations

Exposure from production, processing and formulation

The concentration in air at a distance of 100 meters from the point source is estimated according to TGD. In the calculation for $\text{PEC}_{\text{local}}$ for air both emissions from a point source as well as the emission from a STP is taken into account. The maximum from the two concentrations (direct and via STP) is used as the $\text{PEC}_{\text{local}}$.

$E_{\text{local, air}}$ is the local direct emission rate to air during emission episode

$E_{\text{stp, air}}$ is the local indirect emission to air from STP during episode:

$$E_{\text{stp, air}} = F_{\text{stp, air}} \cdot E_{\text{local, water}}$$

$C_{\text{local, air}}$ is the local concentration in air during emission episode:

$$C_{\text{local, air}} = \text{max Emission (}E_{\text{local, air or E_{stp, air}}}\cdot C_{\text{std, air}},$$

where $C_{\text{std, air}}$ is the standard concentration in air at source strength of 1 kg/d = $2.78 \cdot 10^{-4}$ mg/m$^3$.

$C_{\text{local, air, ann}}$ is the annual average concentration in air, 100 m from point source:

$$C_{\text{local, air, ann}} = C_{\text{local, air}} \cdot 300/365$$

The annual average predicted environmental concentration in air, $\text{PEC}_{\text{local, air, ann}}$, is the local annual average added to the regional concentration in air.

$\text{PEC}_{\text{regional, air}}$: $1.18 \cdot 10^{-6}$ mg/m$^3$ (EUSES)
**Table C.5** PEC\textsubscript{local, air} calculations

<table>
<thead>
<tr>
<th></th>
<th>E\textsubscript{local, air} (kg/d)</th>
<th>E\textsubscript{stp, air} (kg/d)</th>
<th>C\textsubscript{local, air} (mg/m\textsuperscript{3})</th>
<th>C\textsubscript{local, air, ann} (mg/m\textsuperscript{3})</th>
<th>PEC\textsubscript{local, air, ann} (mg/m\textsuperscript{3})</th>
</tr>
</thead>
<tbody>
<tr>
<td>Production</td>
<td>0.075</td>
<td>0.25</td>
<td>0.00013</td>
<td>0.00011</td>
<td>0.00011</td>
</tr>
<tr>
<td>Processing</td>
<td>0.075</td>
<td>0.25</td>
<td>0.00007</td>
<td>0.00006</td>
<td>0.00006</td>
</tr>
<tr>
<td>Formulation</td>
<td>4.5</td>
<td>15.0</td>
<td>0.00417</td>
<td>0.00343</td>
<td>0.00343</td>
</tr>
<tr>
<td>Specific site*</td>
<td>0.57</td>
<td>0.047</td>
<td>0.00016</td>
<td>0.00013</td>
<td>0.00013</td>
</tr>
</tbody>
</table>

*Estimated using monitoring data

**Deposition from air**

The deposition from air includes the emissions from the two sources (direct and STP)

The annual average total deposition flux is defined as:

\[
\text{DEP}_{\text{total, ann}} = \text{DEP}_{\text{total}} \cdot \frac{300}{365}
\]

The total deposition from air during emission episode is calculated as:

\[
\text{DEP}_{\text{total}} = (E_{\text{local, air}} + E_{\text{stp, air}}) \cdot \left[ F_{\text{ass}} \cdot \text{DEP}_{\text{std, aer}} + (1 - F_{\text{ass}}) \cdot \text{DEP}_{\text{std, gas}} \right]
\]

where the fraction of PCOC associated with aerosol particles can be estimated on basis of the chemicals vapour pressure (TGD):

\[
F_{\text{ass}} = \frac{(\text{CON}_{\text{junge}} \cdot \text{SURF}_{\text{aer}})}{(\text{VP} + \text{CON}_{\text{junge}} \cdot \text{SURF}_{\text{aer}})}
\]

\[
= 1 \cdot 10^{-4} \left/ \left( 26.66 + 1 \cdot 10^{-4} \right) \right. = 3.75 \cdot 10^{-6}
\]

\text{DEP}_{\text{std, aer}}, the standard deposition flux of aerosol-bound compound at a source strength of 1 kg/d is default \(1 \cdot 10^{-2}\) mg/m\textsuperscript{2}/d

\text{DEP}_{\text{std, gas}}, the deposition flux of gaseous compounds as a function of Henry’s Law coefficient, at a source strength of 1 kg/d, is for PCOC \(4 \cdot 10^{-4}\) mg/m\textsuperscript{2}/d

**Table C.6** Deposition from the atmosphere calculations

<table>
<thead>
<tr>
<th></th>
<th>E\textsubscript{local, air} (kg/d)</th>
<th>E\textsubscript{stp, air} (kg/d)</th>
<th>DEP\textsubscript{total} (mg/m\textsuperscript{2}/d)</th>
<th>DEP\textsubscript{total, ann} (mg/m\textsuperscript{2}/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Production</td>
<td>0.25</td>
<td>0.482</td>
<td>0.00029</td>
<td>0.00024</td>
</tr>
<tr>
<td>Processing</td>
<td>0.25</td>
<td>0.075</td>
<td>0.00013</td>
<td>0.00011</td>
</tr>
<tr>
<td>Formulation</td>
<td>15.0</td>
<td>0.289</td>
<td>0.00612</td>
<td>0.00503</td>
</tr>
<tr>
<td>Site specific*</td>
<td>0.57</td>
<td>0.047</td>
<td>0.00025</td>
<td>0.00020</td>
</tr>
</tbody>
</table>

*Estimated from monitoring data

**PEC\textsubscript{local, soil} estimations**

The estimated concentration of PCOC in soil may be a results of atmospheric deposition and sludge application.

**Deposition from air**

The contribution from atmospheric deposition, \(D_{\text{air}}\) is derived by converting the total deposition flux \(\text{DEP}_{\text{total, ann}}\) (calculated in the previous appendix) by the equation:
\[ D_{\text{air}} = \frac{\text{DEP}_{\text{total, ann}}}{(\text{DEPTH}_{\text{soil}} \cdot \text{RHO}_{\text{soil}})} \]

where \( \text{RHO}_{\text{soil}} \) is the bulk density of (wet) soil: 1700 kg/m\(^3\)

### Table C.7  Deposition from the atmosphere per kg of soil

<table>
<thead>
<tr>
<th></th>
<th>( \text{DEP}_{\text{total, ann}} ) (mg/m(^2)/d)</th>
<th>( D_{\text{air}} ) 0.2 m soil (mg/kg/d)</th>
<th>( D_{\text{air}} ) 0.1 m soil (mg/kg/d)</th>
<th>( D_{\text{air}} ) 0.05 m soil (mg/kg/d)</th>
<th>( C_{\text{dep, soil}}(0) ) 0.2 m soil (mg/kg)</th>
<th>( C_{\text{dep, soil}}(0) ) 0.1 m soil (mg/kg)</th>
<th>( C_{\text{dep, soil}}(0) ) 0.05 m soil (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Production</td>
<td>0.00024</td>
<td>7.08 \cdot 10^{-7}</td>
<td>1.42 \cdot 10^6</td>
<td>2.83 \cdot 10^6</td>
<td>2.12 \cdot 10^5</td>
<td>4.20 \cdot 10^4</td>
<td>8.23 \cdot 10^6</td>
</tr>
<tr>
<td>Processing</td>
<td>0.00011</td>
<td>3.19 \cdot 10^{-7}</td>
<td>6.39 \cdot 10^7</td>
<td>1.28 \cdot 10^6</td>
<td>9.58 \cdot 10^5</td>
<td>1.90 \cdot 10^5</td>
<td>3.71 \cdot 10^5</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.00503</td>
<td>1.48 \cdot 10^5</td>
<td>2.96 \cdot 10^6</td>
<td>5.91 \cdot 10^6</td>
<td>4.43 \cdot 10^4</td>
<td>8.77 \cdot 10^4</td>
<td>1.72 \cdot 10^5</td>
</tr>
<tr>
<td>Site specific</td>
<td>0.00020</td>
<td>5.88 \cdot 10^{-7}</td>
<td>1.18 \cdot 10^6</td>
<td>2.35 \cdot 10^6</td>
<td>1.76 \cdot 10^5</td>
<td>3.49 \cdot 10^4</td>
<td>6.84 \cdot 10^5</td>
</tr>
</tbody>
</table>

The initial concentration after 10 years of continuous deposition was calculated as:

\[ C_{\text{dep, soil}}(0) = (D_{\text{air}}/k) - (D_{\text{air}}/k) \cdot e^{-365 \cdot 10^{-6} \cdot k} \]

where \( k \) is the first order rate constant for removal from top soil (calculated below).

### Sludge application

The concentration in soil will be high just after sludge application and reduced in time due to removal processes (degradation, volatilisation, leaching, etc.). Therefore, the concentration is averaged over a certain time period for different endpoints:

### Table C.8  Concentration in soil

<table>
<thead>
<tr>
<th></th>
<th>Depth of soil (m)</th>
<th>Averaging time (days)</th>
<th>Sludge application (kg dw/m(^2)/year)</th>
<th>Endpoint</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \text{PEC}_{\text{local, soil}} )</td>
<td>0.20</td>
<td>30</td>
<td>0.5</td>
<td>terrestrial ecosystem</td>
</tr>
<tr>
<td>( \text{PEC}_{\text{local, agr. soil}} )</td>
<td>0.20</td>
<td>180</td>
<td>0.5</td>
<td>crops</td>
</tr>
<tr>
<td>( \text{PEC}_{\text{local, grassland}} )</td>
<td>0.10</td>
<td>180</td>
<td>0.1</td>
<td>grass for cattle</td>
</tr>
</tbody>
</table>

The concentration in dry sewage sludge, \( C_{\text{sludge}} \), is calculated from the emission rate to water, the fraction of emission sorbed to sludge and the rate of sewage sludge production:

\[ C_{\text{sludge}} = \frac{(F_{\text{stp,sludge}} \cdot E_{\text{local, water}} \cdot 10^6)}{\text{SLUDGERATE}} \]

Where SLUDGERATE is the rate of sewage sludge production (default 710 kg/d, local STP).

\( F_{\text{stp,sludge}} \) is 3.59%. The estimated values consider the site specific data known from the manufacturers in using STP flow of 130000 m\(^3\)/d and 700000 personequivalents.
Table C.9  Predicted environmental local concentration in sludge (PEC_{local\_sludge})

<table>
<thead>
<tr>
<th>Life cycle stage</th>
<th>( E_{\text{local, water}} ) (kg/d)</th>
<th>( C_{\text{sludge}} ) (mg/kg dw)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Production</td>
<td>75.0</td>
<td>52.5</td>
</tr>
<tr>
<td>Processing</td>
<td>12.5</td>
<td>8.7</td>
</tr>
<tr>
<td>Formulation</td>
<td>45.0</td>
<td>31.5</td>
</tr>
<tr>
<td>Site specific*</td>
<td>7.8</td>
<td>0.002</td>
</tr>
</tbody>
</table>

*Estimated, using monitoring data

The concentration in soil just after the first year of sludge application is calculated by:

\[
C_{\text{sludge, soil } 1(0)} = \frac{C_{\text{sludge}} \cdot \text{APPL}_{\text{sludge}}}{(\text{DEPTH}_{\text{soil}} \cdot \text{RHO}_{\text{soil}})}
\]

where dry sludge application rate, \( \text{APPL}_{\text{sludge}} \), the soil depth, \( \text{DEPTH}_{\text{soil}} \) and the bulk density of soil is presented in the table above.

Table C.10  Concentration in soil just after sludge application

<table>
<thead>
<tr>
<th></th>
<th>( C_{\text{sludge, local, agr. soil}} ) (mg/kg)</th>
<th>( C_{\text{sludge, local, grassland}} ) (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Production</td>
<td>0.077</td>
<td>0.031</td>
</tr>
<tr>
<td>Processing</td>
<td>0.013</td>
<td>0.005</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.045</td>
<td>0.018</td>
</tr>
<tr>
<td>Site specific**</td>
<td>0.000003</td>
<td>0.000001</td>
</tr>
</tbody>
</table>

*Estimated, using monitoring data

The concentration in soil after sludge application for 10 consecutive years is assumed to be a realistic worst case. The initial concentration after 10 applications of sludge including the fraction that remains from previous year(s) is calculated by:

\[
C_{\text{sludge, soil } 10(0)} = C_{\text{sludge, soil } 1(0)} \cdot (1 + \sum \text{Face}^n)
\]

where the fraction accumulated in one year, \( \text{Face} = e^{-365 \cdot k} = 5.16 \cdot 10^{-6} \) for 0.2 m soil depth and \( 4.54 \cdot 10^{-6} \) for 0.1 m soil depth.

The initial concentration in soil after 10 years of sludge application is estimated to be the same as after first year. There is no indication of accumulation in top soil.

Removal rates

The first order constant for removal from top soil, \( k \), is derived by adding the biodegradation \( (k_{\text{bio, soil}}) \), volatilisation \( (k_{\text{volat}}) \) and leaching rate constant \( (k_{\text{leach}}) \) calculated for PCOC according to TGD.
Table C.11  Removal rate constants

<table>
<thead>
<tr>
<th>Soil depth</th>
<th>$K_{bio\text{soil}}$ $(d^{-1})$</th>
<th>$k_{volat}$ $(d^{-1})$</th>
<th>$k_{leach}$ $(d^{-1})$</th>
<th>$k$ $(d^{-1})$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.2 m</td>
<td>0.033007</td>
<td>0.000156</td>
<td>0.000197</td>
<td>0.033353</td>
</tr>
<tr>
<td>0.1 m</td>
<td>0.033007</td>
<td>0.000312</td>
<td>0.000393</td>
<td>0.033705</td>
</tr>
<tr>
<td>0.05 m</td>
<td>0.033007</td>
<td>0.000624</td>
<td>0.000785</td>
<td>0.034416</td>
</tr>
</tbody>
</table>

**Local concentration in soil**

The sum of both the concentration due to deposition and sludge application as the initial concentration in year 10 is calculated by the equation:

$$C_{soil\text{10}(0)} = C_{dep,\text{soil\text{10}(0)}} + C_{sludge, \text{soil\text{10}(0)}}$$

Table C.12  Initial concentration in soil after 10 years of deposition and sludge application

<table>
<thead>
<tr>
<th></th>
<th>$C_{local,\text{agriculture\text{soil\text{10}(0)}}}$ $(mg/kg)$</th>
<th>$C_{local,\text{grassland\text{soil\text{10}(0)}}}$ $(mg/kg)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Production</td>
<td>0.077</td>
<td>0.031</td>
</tr>
<tr>
<td>Processing</td>
<td>0.013</td>
<td>0.005</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.045</td>
<td>0.018</td>
</tr>
<tr>
<td>Site specific*</td>
<td>0.000003</td>
<td>0.000001</td>
</tr>
</tbody>
</table>

*Estimated, using monitoring data

The initial concentration is used to calculate the average concentration over a certain time period by:

$$C_{local,\text{soil}} = \left( \frac{D_{air}}{k} \right) + \frac{1}{(k \cdot T)} \cdot [(C_{soil\text{(0)}}) - (\frac{D_{air}}{k}) \cdot (1 - e^{-k \cdot T})]$$

For natural soil is deposition from air included only as no sludge application is assumed.

$$PEC_{\text{local, soil}} = C_{\text{local, soil}} + PEC_{\text{regional, natural soil}}$$

$$PEC_{\text{regional, natural soil}}: 8.5 \cdot 10^{-7} \text{ mg/kg ww.}$$

$PEC_{\text{local, soil}}$ is set equal to $C_{\text{local, soil}}$ because the regional contribution is estimated to be very much lower than the estimated values except for the specific site estimated from $C_{\text{sludge monitoring data}}$ (cf. Table C.13).

Table C.13  Estimated local concentration in soil

<table>
<thead>
<tr>
<th></th>
<th>PEC local, soil $(mg/kg)$</th>
<th>PEC local, agr. soil $(mg/kg)$</th>
<th>PEC local, grassland $(mg/kg)$</th>
<th>PEC local, natural soil $(mg/kg)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Production</td>
<td>0.049</td>
<td>0.0128</td>
<td>0.0058</td>
<td>0.00003</td>
</tr>
<tr>
<td>Processing</td>
<td>0.008</td>
<td>0.0021</td>
<td>0.0009</td>
<td>0.00001</td>
</tr>
<tr>
<td>Formulation</td>
<td>0.030</td>
<td>0.0061</td>
<td>0.0039</td>
<td>0.00336</td>
</tr>
<tr>
<td>Specific site*</td>
<td>0.000002</td>
<td>0.0000014</td>
<td>0.000011</td>
<td>0.00000088</td>
</tr>
</tbody>
</table>

*Estimated, using monitoring data
**Concentration in pore water**

The concentration in porewater is calculated by the equation:

$$\text{PEC}_{\text{local, soil, porew}} = \frac{\text{PEC}_{\text{local, soil}} \cdot \rho_{\text{soil}}}{(K_{\text{soil-water}} \cdot 1000)}$$

where $K_{\text{soil-water}}$ is the soil-water partition coefficient calculated to be 12.2

<table>
<thead>
<tr>
<th>Table C.14 Local concentration in soil porewater</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PEC</strong>_{local, soil, porewater} (mg/l)</td>
</tr>
<tr>
<td>Production</td>
</tr>
<tr>
<td>Processing</td>
</tr>
<tr>
<td>Formulation</td>
</tr>
<tr>
<td>Site specific*</td>
</tr>
</tbody>
</table>

*Estimated using monitoring data

**Concentration in groundwater**

The concentration in groundwater calculated for indirect exposure to humans through drinking water is initially assessed by the concentration in porewater in agricultural soil for a worst case estimation according to TGD.

$$\text{PEC}_{\text{local, grw}} = \frac{\text{PEC}_{\text{local, agr. soil, porew}}}{(K_{\text{soil-water}} \cdot 1000)}$$

<table>
<thead>
<tr>
<th>Table C.15 Local concentration in groundwater</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PEC</strong>_{local, grw} (mg/l)</td>
</tr>
<tr>
<td>Production</td>
</tr>
<tr>
<td>Processing</td>
</tr>
<tr>
<td>Formulation</td>
</tr>
<tr>
<td>Site specific</td>
</tr>
</tbody>
</table>
Appendix D  Estimation of inhalation and dermal exposure EASE-model

Results from Ease model
This file was created by the EASE system
EASE for Windows version: 2.0

Standard data (inhalation)
Name of the substance: 4-chloro-2-methylphenol
The temperature of the process: 150°C
The physical state of the substance: Liquid
The exposure type: gas/vapour/liquid aerosol
The status vp is measured at a different temperature
The measurement-temperature is 70
The vp value of the substance is 0.16 KPa at 70°C
The calculated vp is 4.023 Kpa
The volatility of the substance is moderate
The ability-airborne-vapour of the substance is moderate

Scenario: Production (or processing) of PCOC
Aerosol-formed is false
The use-pattern is closed system
Significant-breaching is false
The pattern of control is full containment
The predicted gas/vapour/liquid aerosol exposure to 4-chloro-2-methylphenol is 0-0.1 ppm

Standard data (dermal)
Name of the substance: 4-chloro-2-methylphenol
The temperature of the process: 150°C
The physical state of the substance: Liquid
The exposure type is dermal

Scenario: Production (or processing) of PCOC
The use-pattern is closed system
Significant-breaching is false
The pattern of control is not direct handling

Conclusion
The predicted dermal exposure to 4-chloro-2-methylphenol is very low.
The report provides the comprehensive risk assessment of the substance 4-Choro-o-cresol. It has been prepared by Denmark 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 man and the environment, laid down in Commission Regulation (EC) No. 1488/94.

The evaluation considers the emissions and the resulting exposure to the environment and the human populations 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. For human health the scenarios for occupational exposure, consumer exposure and humans exposed via the environment have been examined and the possible risks have been identified.

The risk assessment for 4-chloro-o-cresol concludes that there is at present no concern for the environment or for human health. There is at present no need for further information and/or testing or for risk reduction measures beyond those that are being applied already.
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