European Chemicals Bureau

Existing Substances

European Union Risk Assessment Report

CAS No: 79-10-7
EINECS No: 201-177-9

acrylic acid

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ACRYLIC ACID

CAS No: 79-10-7

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RISK ASSESSMENT
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ACRYLIC ACID

CAS No: 79-10-7
EINECS No: 201-177-9

RISK ASSESSMENT

Final Report, 2002

Germany

The risk assessment of acrylic acid (AA) has been prepared by Germany on behalf of the European Union.

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Final report: 2002

(The last full literature survey was carried out in 1995 - targeted searches (for example on grouting and PBPK model) were carried out subsequently, and information found through scanning certain sources has also been included).
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) 793/931 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) 1488/942, which is supported by a technical guidance document3. 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. The Risk Assessment Report is also presented to the Organisation for Economic Co-operation and Development as a contribution to the Chapter 19, Agenda 21 goals for evaluating chemicals, agreed at the United Nations Conference on Environment and Development, held in Rio de Janeiro in 1992.

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 Mc Sweeney
Director-General
DG Joint Research Centre

Catherine Day
Director-General
DG Environment

1 O.J. No L 084, 05/04/199 p.0001 – 0075
2 O.J. No L 161, 29/06/1994 p. 0003 – 0011
OVERALL RESULTS OF THE RISK ASSESSMENT

CAS-No.: 79-10-7
EINECS-No.: 201-177-9
IUPAC name: 2-propenoic acid
Synonym: acrylic acid

Environment

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

Acrylic acid (AA) presents, based on the present data, a risk to the environment around point sources. A potential risk to municipal wastewater treatment plants is identified for the downstream use scenarios of super absorber polymers (SAP) production (based on default calculation and highest site-specific PECwwtp) and wet polymerisation (based on default calculation and known sites L, Q).

Since the PNECmicroorganisms is derived from single species tests with ciliated protozoa, there is a need for further data reflecting the integrity of the native ciliate population in sewage sludge as a whole. However, since risk reduction measures are necessary to remove concern for surface water (see below), these measures will also cover the protection of municipal wastewater treatment plants, and additional testing is not required.

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

This conclusion applies to effects on sediment, atmosphere, soil, and secondary poisoning. Conclusion (ii) applies also to the aquatic compartment regarding all production sites, the processing scenario (dry polymerisation), and the relevant use scenarios (leather finishing, textile finishing, formulation of paints and application of water treatment agents).

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

Acrylic acid (AA) presents, based on the present data, a risk to the environment around point sources.

A potential risk to the local aquatic environment is identified from wet polymerisation processes including wet production of SAP (super absorber polymers) by downstream users of monomeric AA (based on default calculations and known sites N, O, Q).

Although an improvement of the data (i.e. effluent measurements and/or site specific data on flow rates) may in principle be possible, it is judged to be unlikely that sufficiently complete representative monitoring data from the downstream users can be obtained with reasonable expenditure of time and money. For certain known SAP production sites and wet polymerisation sites, regular effluent concentrations up to 100 mg/l AA and significantly more have been reported. These data indicate that high effluent concentrations cannot be excluded, even if certain types of process engineering are applied. On the other hand, application of wastewater reutilization / recycling systems is known to result in zero emissions to the hydrosphere at a
number of downstream user sites, processing about 50% of AA used externally for SAP production and about 12% of AA used externally in wet polymerisation processes. For sites applying this kind of technique, no further risk reduction measures are deemed necessary. Measures applied for limiting the risk to the local aquatic environment are presumed to be also protective for municipal wastewater treatment plants.

During the use of a grouting agent containing magnesium diacrylate high concentrations of AA are released via the drainage water. The exposure assessment was based on measured effluent concentrations at a tunnel construction site. A quantitative extrapolation to other construction sites seems difficult, but similar conditions might be anticipated. Measures appropriate to local circumstances should be applied.

**Human health**

**Human health (toxicity)**

**Workers**

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

This conclusion is reached because of:

- concerns for respiratory tract irritation and corrosivity as a consequence of single inhalation exposure arising from production and processing, production of adhesives containing the substance and use of adhesives containing the substance (industrial area and skilled trade),

- concerns for local effects as a consequence of repeated inhalation exposure arising from production and use of adhesives containing the substance,

- concerns for general systemic toxicity as a consequence of repeated inhalation exposure arising from production and use of adhesives containing the substance.

**Consumers**

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

**Humans exposed via the environment**

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

**Human health (risks from physico-chemical properties)**

**Conclusion (ii)** There is at present no need for further information or testing or risk reduction measures beyond those which are being applied already.
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1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTIFICATION OF THE SUBSTANCE

CAS-No.: 79-10-7
EINECS-No.: 201-177-9
IUPAC name: 2-Propenoic acid
Synonyms: Acrylic acid
Molecular weight: 72.06 g/mol
Molecular formula: C₃H₄O₂
Structural formula:

1.2 PURITY/IMPURITIES, ADDITIVES

Purity: 99.7% w/w
Impurity: < 0.05% w/w water
< 0.05% w/w propionic acid
< 0.2% w/w acetic acid
< 0.5% w/w dimers of acrylic acid
Additives: < 0.02% w/w hydroquinone monomethylether

For commercial acrylic acid (AA) 0.02% hydroquinone monomethylether (MEHQ) is added as stabilizer to avoid a spontaneous polymerisation (Bauer, 1991).
1.3 PHYSICO-CHEMICAL PROPERTIES

Table 1.1 Physico-chemical properties of acrylic acid

<table>
<thead>
<tr>
<th>Properties</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical state</td>
<td>liquid at 20°C</td>
<td>Merck Index (1996)</td>
</tr>
<tr>
<td>Melting point</td>
<td>14°C</td>
<td>Merck Index (1996)</td>
</tr>
<tr>
<td>Boiling point</td>
<td>141°C at 1,013 hPa</td>
<td>Merck Index (1996)</td>
</tr>
<tr>
<td>Density</td>
<td>1.0621 g/cm$^3$ at 20°C</td>
<td>Merck Index (1996)</td>
</tr>
<tr>
<td>Vapour pressure</td>
<td>3.8 hPa at 20°C - (dynamic method)</td>
<td>BASF AG (1994a)</td>
</tr>
<tr>
<td>Surface tension</td>
<td>59.6 mN/m c=1g/l - (ring method)</td>
<td>Hüls AG (1995)</td>
</tr>
<tr>
<td>Water solubility</td>
<td>miscible in all ratios</td>
<td>Merck Index (1996)</td>
</tr>
<tr>
<td>Dissociation constant</td>
<td>pK$_a$ = 4.25</td>
<td>Weast (1989)</td>
</tr>
<tr>
<td>Partition coefficient</td>
<td>log P$_{ow}$ 0.46 at 25°C - (shake flask method)</td>
<td>BASF AG (1988)</td>
</tr>
<tr>
<td>Flash point</td>
<td>48-55°C</td>
<td>CHEMSAFE</td>
</tr>
<tr>
<td>Auto flammability</td>
<td>395°C - DIN 51794</td>
<td>CHEMSAFE</td>
</tr>
<tr>
<td>Flammability</td>
<td>flammable</td>
<td>Test A.12 not conducted because of structural reasons</td>
</tr>
<tr>
<td>Explosive properties</td>
<td>not explosive</td>
<td>no test because of structural reasons</td>
</tr>
<tr>
<td>Oxidizing properties</td>
<td>no oxidizing properties</td>
<td>no test because of structural reasons</td>
</tr>
</tbody>
</table>

1) Under normal conditions acrylic acid is a clear, colourless liquid with a pungent smell

1.4 CLASSIFICATION

Classification and labelling according to the 28$^{th}$ ATP of Directive 67/548/EEC$^4$:

Classification: R10 Flammable
Xn; R20/21/22 Harmful by inhalation, in contact with skin and if swallowed
C; R35 Corrosive; Causes severe burns
N; R50 Dangerous for the environment; very toxic to aquatic organisms

Note D

Labelling: C; N
R: 10-20/21/22-35-50
S: (1/2-)26-36/37/39-45-61

Concentration limits

\[ C \geq 25\%: \quad C; \ R20/21/22-35 \]
\[ 10\% \leq C < 25\%: \quad C; \ R35 \]
\[ 5\% \leq C < 10\%: \quad C; \ R34 \]
\[ 1\% \leq C < 5\%: \quad Xi; \ R \ 36/37/38 \]

2 GENERAL INFORMATION ON EXPOSURE

2.1 PRODUCTION

2.1.1 Production processes
Acrylic acid (AA) is produced commercially by catalytic oxidation of propylene in two steps via acrolein or by a modification of the Reppe process from acetylene. In addition, AA can be prepared by hydrolysis of acrylonitrile (ECETOC, 1995).

2.1.2 Production capacity
As an important intermediate for polymer industry AA is produced in large quantities within the EU. Data from 6 producers and 3 importers are included in the IUCLID-database. The maximum cumulative production capacity from the indicated ranges amounts to 1,610,000 t/a. Taking into account the actual production capacities also provided by all companies a total production capacity of 810,000 t/a is calculated. This corresponds well to SRI’s 1996 estimate of 815,000 tonnes annual production capacity (SRI, 1996). The highest actual production capacity at a single site is 330,000 t/a. At most, one large production site is located in a single region. A few isolated import figures have been communicated from different sites for different years, ranging up to 8,800 t per site from outside the EU. According to information from one producer 15,000 t/a are exported outside the EU.

Since only limited information on actual production and import volumes were provided by industry, a total amount of 830,000 t/a AA consumption (810,000 t/a production capacity plus 20,000 t/a total net import) is taken as a basis for the calculations.

The market trend appears quite dynamic during the last decade. Fox et al. (1990) estimated the Western European production of acrylic acid in 1987 at about 342,000 tonnes. For 1994, the worldwide consumption of AA is estimated at 2.4 million tonnes. The worldwide production capacity is expected to expand by another 500,000 t/a within 4 years (Rohe, 1995), that equals ca. 5% growth per year. In Germany a new production site is under construction, the operating company announcing for 1999 an initial production capacity of 80,000 t/a crude AA, and as commercial products 60,000 t/a glacial, i.e. purified AA and 60,000 t/a acrylate esters. An option for quick doubling of the capacity is reported (Europa Chemie 14/98, pp. 4-6: “Ausbau zum Vorzeigestandort”).

2.2 USES
Acrylic acid serves as an industrial intermediate product, i.e. it is either processed directly into a polyacrylate or polymerised via the intermediate stage of an acrylate ester. Furthermore, acrylic acid is used as an ingredient and occurs as residual monomer in consumer products like adhesives, paints, binding agents and printing inks.

According to the producers about half of the 830,000 t/a crude AA is processed to purified (glacial) AA, which is further processed both on-site (captive use) and by external downstream users. The other half of crude AA is transformed into various acrylate esters at the production
sites. As for glacial AA, these acrylic esters serve as commercial products, which are further processed both on-site and by external downstream users.

The segmentation of the user industry has been recalculated independently by the producers yielding the new data given in Table 2.1. Calculations have been made on the basis of general accessible market information like TECNON:

<table>
<thead>
<tr>
<th>Product / use</th>
<th>% in this application</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dispersions</td>
<td>42</td>
</tr>
<tr>
<td>Superabsorber polymers</td>
<td>25</td>
</tr>
<tr>
<td>Textiles / leather</td>
<td>10</td>
</tr>
<tr>
<td>Acrylic fibres</td>
<td>4</td>
</tr>
<tr>
<td>Others</td>
<td>19</td>
</tr>
</tbody>
</table>

Among the polyacrylates (homo- and copolymers of AA), superabsorber polymers (SAP) are the most expansive use. In an assessment for 1993, the need for acrylic acid for the manufacture of superabsorbents and detergents in Western Europe was estimated to be 50,000 t/a each (Fox et al., 1990). It is estimated that its part in the global consumption will rise from 15 to 20% (Rohe, 1995). The actual EU figure of 25% represents an amount of about 210,000 t/a used for SAP production.

Further applications of the polyacrylates are co-builders in phosphate-free washing agents, and as flocculating agents for treatment of drinking water and wastewater.

Acrylic acid is also used as a pre-stage in the manufacture of coatings for printed circuit boards, so-called photoresists (Winkelmann et al., 1989; Reichert, 1993; Hoechst, 1994). AA may also be released as a decomposition product, when these photoresists are partially depolymerised by means of UV-light in the electroplating industry.

Recently, release of monomeric AA has been reported from the use of acrylate-based grouting agents at tunnel construction sites.

In the consumer field acrylic acid is used as an ingredient in adhesives. Moreover, acrylic acid occurs as residual monomer, e.g. SAP-containing products like sanitary towels, pantyliners and nappy pants. In addition, residual acrylic acid monomer may be contained in materials used for food packaging and in other commodities covered by specific legislation.

A summary of the content of AA in different products is presented in the Danish Product Register from June 1996 (no production of AA in Denmark), as shown in Table 2.2. The most frequent product types are paints, lacquers and varnishes, binding agents and printing inks.

In the Norwegian Product Register from 1994, 76 products containing a total quantity of 19 tonnes AA are registered. The most frequent product types are raw materials and additives (11 t/a), varnishes (4 t/a) and binders for paint, glue and castings (2 t/a).

According to the Swedish Product Register, 13 out of 56 products containing acrylic acid are used by consumers.
Table 2.2  AA-content - ranges in products and amount sold in Denmark  
(Danish Product Register, June 1996)

<table>
<thead>
<tr>
<th>Content of AA in the product</th>
<th>Number of products</th>
<th>Quantity of AA sold in Denmark [t/a]</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-1%</td>
<td>700</td>
<td>384</td>
</tr>
<tr>
<td>1-10%</td>
<td>75</td>
<td>4</td>
</tr>
<tr>
<td>10-80%</td>
<td>27</td>
<td>50</td>
</tr>
<tr>
<td>80-100%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>not determined</td>
<td>22</td>
<td></td>
</tr>
</tbody>
</table>

Table 2.3 gives an overview about the main, industrial and use categories according to the TGD (EC, 1996). Due to uncertainties about the amounts of AA assignable to these categories no quantitative information or percentages can be given.

Table 2.3  Main, industrial and use categories according to the Technical Guidance Document (EC, 1996)

<table>
<thead>
<tr>
<th>Main category (MC)</th>
<th>Industrial category (IC)</th>
<th>Use category (UC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>isolated intermediate (1b, 1c)</td>
<td>chemical industry (3)</td>
<td>intermediate (33)</td>
</tr>
<tr>
<td>matrix-inclusion (2), non dispersive use (3)</td>
<td>polymers industry (11)</td>
<td>intermediate (33)</td>
</tr>
<tr>
<td>non dispersive use (3)</td>
<td>paints, lacquers and varnishes industry (14)</td>
<td>intermediate (33)</td>
</tr>
<tr>
<td>wide dispersive use (4)</td>
<td>personal / domestic (5), public domain (6)</td>
<td>adsorbents, absorbents (1)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>adhesives, binding agents (2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>cleaning/washing additives (9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>flotation agents (23)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>others (0)</td>
</tr>
</tbody>
</table>

**Acrylate esters**

Yielded via esterification from about 415,000 t/a AA (50% of the total EU consumption amount), acrylate esters are important monomers for the production of homo- and copolymers, which are used in the paint and adhesives industry, as well as for finishing of paper, textiles and leather; mainly as dispersions but also in solution. 99% of the acrylate esters are n-butyl, ethyl, methyl and 2-ethylhexyl acrylates, butylacrylate predominating quantitatively. Through polymerisation of the esters, transparent UV-resistant acrylic resins are produced, either thermoplastic or duroplastic by inclusion of functional groups like hydroxyl or carboxyl groups. These are employed as binding agents in paints and adhesives as well as coatings, thickeners and dispersion agents. With co-monomers containing vulcanising groups, acrylate rubbers are produced through polymerisation of acrylate esters, especially ethyl- and butyl acrylate (Rohe, 1995).

Fox et al. (1990) present the following consumption breakdown for acrylate esters in Western Europe in 1988, with a projection for 1993:
### Table 2.4  Consumption breakdown for acrylic esters

<table>
<thead>
<tr>
<th>Product</th>
<th>Consumption [t]</th>
<th>1988</th>
<th>1993</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emulsion polymers</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Latex Surface Coatings</td>
<td></td>
<td>92,000</td>
<td>110,000</td>
</tr>
<tr>
<td>Paper Coatings</td>
<td></td>
<td>20,000</td>
<td>23,000</td>
</tr>
<tr>
<td>Adhesives and Sealants</td>
<td></td>
<td>35,000</td>
<td>44,000</td>
</tr>
<tr>
<td>Textiles and Non-wovens</td>
<td></td>
<td>40,000</td>
<td>40,000</td>
</tr>
<tr>
<td>Leather</td>
<td></td>
<td>5,000</td>
<td>5,000</td>
</tr>
<tr>
<td>Polishes</td>
<td></td>
<td>4,000</td>
<td>4,000</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td></td>
<td>10,000</td>
<td>10,000</td>
</tr>
<tr>
<td>Non-emulsion Applications</td>
<td></td>
<td>92,000 (31%)</td>
<td>101,000 (30%)</td>
</tr>
<tr>
<td>Solvent-Based Surface Coatings</td>
<td></td>
<td>36,000</td>
<td>40,000</td>
</tr>
<tr>
<td>Acrylic Fibers</td>
<td></td>
<td>32,000</td>
<td>32,000</td>
</tr>
<tr>
<td>Impact Modifiers</td>
<td></td>
<td>6,000</td>
<td>7,000</td>
</tr>
<tr>
<td>Copolymers and Miscellaneous</td>
<td></td>
<td>18,000</td>
<td>22,000</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>298,000</td>
<td>337,000</td>
</tr>
</tbody>
</table>
3 ENVIRONMENT

3.1 ENVIRONMENTAL EXPOSURE

3.1.1 Environmental releases

Releases of AA into the environment are to be expected during production and processing mainly via wastewater and lesser amounts via exhaust gases. Regarding the formulation step, relevant releases may possibly occur during formulation of polymer dispersions.

Further releases are to be expected through residual monomeric AA-contents in the final products. Residual monomeric concentrations determined by several companies in various polymer products ranged from 2.2 to 2,000 ppm. Per tonne AA used, a maximum residue of 4,000 g AA monomer was measured in newly manufactured products. For acrylate esters a residual monomeric AA content of 25 ppm has been reported. Due to the large amounts of AA being processed and related to the specific polymer end-uses the residual amounts of AA have to be taken into account for release estimation.

From the use of grouting agents containing magnesium diacrylate, releases of AA to the hydrosphere occur via drainage water.

Direct releases to agricultural or natural soil are not expected from the current use pattern.

3.1.2 Environmental fate

3.1.2.1 Degradation

Hydrolysis

In sterile water, acrylic acid has proved to be completely stable to hydrolysis at all tested pHs (3, 7, 11) over 28 days of investigation (Shah, 1990).

Photooxidation

When released into the atmosphere, acrylic acid reacts with photochemically produced hydroxyl radicals primarily by addition to the double bond, and with atmospheric ozone, resulting in estimated half-lifes of 39.6 hours and 6.5 days, respectively, assuming a hydroxyl radical concentration of 500,000 molecules/cm$^3$ and an ozone concentration of $7 \times 10^{11}$ molecules/cm$^3$.

Because the UV absorption band extends to 320 nm (Sadtler, 1960), direct sunlight photolysis of acrylic acid is possible, but no experimental data are available.

Biodegradation

Based on the results of three guideline tests acrylic acid can be considered as readily biodegradable:

1. Douglas (1991) found that degradation achieved 81% of the theoretical oxygen demand (ThOD) after 28 days when acrylic acid was tested in a Closed-Bottle-Test (OECD 301 D).
2. In another investigation (Modified MITI Test (I); OECD 301 C) after 14 days the oxygen demand amounted to 68% of ThOD (CITI, 1992).

3. The result of a DOC-Die-Away-Test (OECD 301 A) showed 95% decrease of DOC within 9 days (Hüls, 1995a).

Furthermore, results from other tests are available which show high biodegradation rates. In an investigation conducted by Pahren and Bloodgood (1961) the biodegradation of acrylic acid measured as CO$_2$ evolution was in one case 70% after 19 days and in another case 71% after 42 days (percentage of theoretical CO$_2$ evolution, substance concentration 10 mg/l). In a Zahn-Wellens Test 100% of the substance (200 mg/l DOC) was degraded after 5 days (BASF AG, 1993).

A test on biodegradation in soil, performed according to a US EPA guideline is also available (Hawkins et al., 1992). From the test design, the test can be rated as a simulation test. The test conditions and main results are given in Table 3.1.

<table>
<thead>
<tr>
<th>Test conditions</th>
<th>(14^C)-labelling of the substance</th>
</tr>
</thead>
<tbody>
<tr>
<td>aerobic conditions</td>
<td>content of acrylic acid in soil: 100 mg/kg dw</td>
</tr>
<tr>
<td>duration: 28 days</td>
<td>incubation in darkness at 25°C</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Main results</th>
<th>72.9% mineralisation within 3 days</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>81.1% mineralisation after 28 days</td>
</tr>
<tr>
<td>DT$_{50}$ for primary degradation</td>
<td>&lt;1 day (estimated)</td>
</tr>
<tr>
<td>10% of radioactivity non-extractable after 28 days</td>
<td></td>
</tr>
</tbody>
</table>

From the soil test it can be concluded that acrylic acid is readily biodegradable in this soil type. The applicability of the comparatively short DT$_{50}$ < 3 d to other soil types and environmentally more relevant lower temperatures is not clear. However, the rate constant calculated according to the Technical Guidance Document (TGD) (Ch. 3, Table 6) $\text{k}_{\text{bio, soil}} = 0.023$ d$^{-1}$ corresponding to a DT$_{50}$ of ca. 30 d appears to underestimate biodegradation. As a possibly reasonable approach, the rate constant for ready biodegradability in surface water $\text{k}_{\text{bio, soil}} = 0.047$ d$^{-1}$ corresponding to DT$_{50}$ $\approx$ 15 d is used, also taking into account the low adsorption rates of AA (see below).

Acrylic acid is also susceptible to degradation by anaerobic microbes. In a screening study using 10% sludge from a secondary digester as an inoculum, acrylic acid was judged to be degradable with $> 75\%$ of theoretical methane being produced within 8 weeks of incubation (Shelton and Tiedje, 1984). The result does not reveal environmental conditions as the study was conducted at 35°C.

Results from other biodegradation simulation tests are not available for the different compartments and have to be estimated according to the TGD based on the above-described screening tests and the partitioning behaviour of AA:
### Table 3.2  Biodegradation rate constants for different compartments

<table>
<thead>
<tr>
<th>Compartment / medium</th>
<th>Biodegradation rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Activated sludge (WWTP)</td>
<td>$k_{\text{WWTP}} = 1 \text{ h}^{-1}$</td>
</tr>
<tr>
<td>Surface water</td>
<td>$k_{\text{deg_water}} = 0.047 \text{ d}^{-1}$, (Table 5, TGD)</td>
</tr>
<tr>
<td>Sediment</td>
<td>$k_{\text{bio_sed}} = 0.0023 \text{ d}^{-1}$</td>
</tr>
<tr>
<td>Soil</td>
<td>$k_{\text{bio_soil}} = 0.047 \text{ d}^{-1}$ (See text)</td>
</tr>
</tbody>
</table>

#### 3.1.2.2 Distribution

On the basis of a vapour pressure of 380 Pa and a water solubility of 1 kg/l (AA is miscible with water in all ratios) a Henry's law constant $H = 0.027 \text{ Pa} \cdot \text{m}^3/\text{mol}$ at 25°C was calculated, which suggests that AA is essentially non volatile. Due to its pKa value of 4.25 the dissociated anionic form of AA is dominating under normal environmental conditions (pH range 5.5-9). Therefore it has to be expected that the real partition of the total AA is determined only by its anionic form with a Henry's law constant being much lower than calculated above.

Adsorption and desorption of AA were examined on five different soils (an aquatic sandy loam sediment, a loamy sand, a clay loam and two loam soils). The Kp values for the adsorption phase ranged from 0.28 to 0.63 l/kg. The Kp values for the three desorption phases were scattered more widely with values ranging from 0.38 to 3.85 l/kg (Archer and Horvath, 1991). All adsorption and desorption results proved to be independent from the organic matter contents. It can be assumed that the adsorption behaviour of the anionic form of AA depends primarily on the inorganic fraction of the different soils. Therefore, and due to missing partition coefficients for different types of solid matter, it was preferred to use a uniform value of 1 l/kg for all partition coefficients (Kp soil, Kp sed, Kp susp, Kp sludge), which is very close to the value of 1.285 as exactly calculated from the given absorption and desorption ranges.

Using the fugacity model of Mackay (EQC model, level 1), the theoretical distribution of AA at equilibrium can be estimated:

#### Table 3.3  Equilibrium distribution according to fugacity model of Mackay (EQC model 1.0, level 1)

<table>
<thead>
<tr>
<th>Compartment</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>5.2</td>
</tr>
<tr>
<td>Water</td>
<td>94.7</td>
</tr>
<tr>
<td>Soil</td>
<td>0.1</td>
</tr>
</tbody>
</table>

Taking into account that the dissociated anionic form of AA is normally dominating (pKa 4.25), more than 99% of total AA has to be expected in the water compartment. In any case, the hydrosphere is the preferred target compartment for distribution.
Elimination in WWTPs

Based on log $H = -1.57$ and log $P_{ow} = 0.46$, as well as the biodegradation rate of 1 h$^{-1}$ in WWTP, the elimination through biodegradation and distribution can be estimated with the model SIMPLETREAT 3.0 (Version 1997):

<table>
<thead>
<tr>
<th>Biodegradation (%)</th>
<th>87.3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Release to air (%)</td>
<td>0</td>
</tr>
<tr>
<td>Release to water (%)</td>
<td>12.7</td>
</tr>
<tr>
<td>Adsorption to sewage sludge (%)</td>
<td>0</td>
</tr>
<tr>
<td>Total elimination from water (%)</td>
<td>87.3</td>
</tr>
</tbody>
</table>

This distribution would not change, if due to the dominance of the anionic form of AA lower and more realistic values for log $H$ and log $P_{ow}$ are assumed.

3.1.2.3 Accumulation

No experimental results on bioaccumulation are available. The measured log $P_{ow}$ of 0.46 does not indicate a potential for bioaccumulation though. Based on this value a BCF of $0.49 \text{l} \cdot \text{kg}^{-1}$ can be estimated for fish according to the TGD (Chapter 4, Table 6). This calculation implies that the undissociated form of AA is dominating. For the anionic form a much lower BCF has to be expected.

The experimentally determined Kp values for soils in the range of 0.28 to 3.85 l/kg and the ready biodegradability also indicate no potential for geoaccumulation. Acrylic acid is highly mobile in soil and the amount which is not degraded may leach with seepage to the groundwater.

3.1.3 Aquatic compartment

Specific information from the production sites covering on-site processing is taken into account for the exposure assessment. Generic exposure scenarios for external processing and use are based on market information provided recently (1999) by the AA producers.

3.1.3.1 Estimation of $C_{local\,water}$ / Generic approach: production and processing

In the TGD, a generic (i.e. non site-specific) exposure scenario for the release of intermediates into surface water during production and processing is proposed (Emission Scenario Document IC-3).

The highest single production capacity of AA equals 330,000 t/a. Since this capacity is high enough to represent a realistic worst case, this amount is taken for default calculation, considering production and processing of AA at one site. However, as for the actual situation more specific data are available for the largest production site (see below), the generic scenario has not been carried through to risk characterisation. A $C_{local\,water}$ of 270 $\mu$g/l is estimated (default emission factors: 0.3% for production and 0.7% for processing; duration of emissions:
300 d/a; 87.3% elimination rate in WWTP according to SIMPLETEAT; default river flow rate according to ESD: 60 m$^3$/s; cf. Appendix A2 for calculation).

### 3.1.3.2 Estimation of $C_{\text{local}_{\text{water}}}$ / Site-specific approach: production and processing

Using the available specific data for several production sites, more precise PEC estimations can be performed for the hydrosphere. The results are presented in **Table 3.5**.

**Table 3.5** Basic data and results of local release estimations into the hydrosphere

<table>
<thead>
<tr>
<th>Site</th>
<th>Flow of receiving water</th>
<th>Release to WWTP [t/a]</th>
<th>Release to hydrosphere [t/a]</th>
<th>$C_{\text{local}_{\text{water}}}$ [µg/l]</th>
<th>Specific data used for exposure estimation</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>site specific</td>
<td>97</td>
<td>12.3</td>
<td>0.64</td>
<td>estimated annual emission based on measurements of WWTP effluent*; specific WWTP flow</td>
</tr>
<tr>
<td>B</td>
<td>site specific</td>
<td>1.28</td>
<td>0.162</td>
<td>0.16</td>
<td>estimated annual emission based on measurements of WWTP effluent; specific WWTP flow</td>
</tr>
<tr>
<td>C</td>
<td>site specific</td>
<td>0.08</td>
<td>0.01</td>
<td>0.77</td>
<td>calculated on the basis of effluent monitoring (n=16, two separate 8-d periods, max = 6.6 µg/l, min = 3.6 µg/l – one single measurement 1998: 10 µg/l, $C_{\text{local}}$ based on average: 4.8 µg/l); specific WWTP flow, specific dilution</td>
</tr>
<tr>
<td>D</td>
<td>site specific</td>
<td>4.21$^{(\dagger)}$</td>
<td>0.53$^{(\dagger)}$</td>
<td>6.6$^{(\dagger)}$</td>
<td>measured concentrations in WWTP influent (n = 281 24 h composite samples in 1998; max = 261 mg/l, 15 samples &gt; 50 mg/l, 49 samples &gt; 15 mg/l, 138 samples &lt; 0.1 mg/l, $C_{\text{local}}$ based on 90%ile: 29 mg/l); specific dilution factor</td>
</tr>
<tr>
<td>E</td>
<td>-</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>no release to controlled or surface waters, no wastewater generating process applied</td>
</tr>
<tr>
<td>F</td>
<td>-</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>no production since 1995; wastewater from production was incinerated before</td>
</tr>
<tr>
<td>G</td>
<td>-</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>no AA containing wastewater during regular operation, continuous process; possible releases to hydrosphere during cleaning operations only</td>
</tr>
<tr>
<td>H</td>
<td>-</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>no import since 1994</td>
</tr>
<tr>
<td>I</td>
<td>site specific</td>
<td>0.173</td>
<td>0.022</td>
<td>0.043</td>
<td>estimated annual emission based on measurements in WWTP effluent**; specific WWTP flow</td>
</tr>
<tr>
<td>**</td>
<td>Total</td>
<td>99</td>
<td>12.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Calculation is based on the detection limit of 100 µg/l, which was not exceeded in any case of 30 measurements. Further measurements applying another analytical method with lower detection limit have to be regarded as not representative (only 3 samples, average ca. 30 µg/l)

** Site D: AA manufacture ceased by October 1999 (confirmed by operating company in March 2000); releases not included in sum for estimation of regional exposure

*** Measurements of WWTP effluent were performed during a production period over one week (six samples, 24h sampling period each, sampling for 3 sec every 3 min); the detection limit was not exceeded and is the basis of calculation...
3.1.3.3 Estimation of C\textsubscript{local}\textsubscript{water} / Generic and site-specific approach: processing by non producers/importers

For the EU, quantities of AA processed at production sites (i.e. captive use) compared to quantities processed at external sites, either of the same company or sold to other companies (non-producer / intermediary), have been communicated by industry.\(^5\)

The production capacity of AA in the EU amounts to 810,000 t/a. A total of 830,000 t/a is taken as EU consumption figure (cf. Section 2.1.2). As mentioned above, half of this amount, i.e. 415,000 t/a, is esterified directly at the production sites. Further, a significant part of the remaining half is processed directly at the production sites, too. All releases due to processing of these quantities are covered by the site-specific emissions of the production sites (see Section 3.1.3.2, Table 3.5). Therefore, the estimation of releases due to processing at external downstream user sites can be limited to the remaining tonnage. The following main areas of use have been identified:

- production of super absorber polymers (SAP, wet and dry processes),
- wet polymerisation, namely dispersions, water-treatment agents, detergents, UV-curing agents, others,
- dry polymerisation.

All the main external processing types mentioned above comprise wet and dry polymerisation techniques. According to a comprehensive set of specific confidential information, provided by a significant portion of AA downstream users, an amount of 10,000 t/a processing volume at one generic single site is assumed as a realistic worst case.

Applying the respective default parameters according to the TGD (Table A3.10: emission factor 0.01, Table B3.9: emission episode 300 d, WWTP flow 2,000 m³/d, elimination rate 87.3%, dilution in surface water 1:10), gives:

\[
C_{\text{local\textsubscript{water}}} = 2.12 \text{ mg/l}
\]

(the respective calculation is presented in Appendix A3).

From dry polymerisation no relevant releases with wastewater into the aquatic compartment have to be assumed (emission factor = 0 according to Table A3.10 of the TGD).

Based on additional information provided by external processing companies it was possible to refine the generic scenario and distinguish between production of SAP (wet) and wet polymerisation. The estimates are based on a comprehensive set of specific confidential information provided by downstream users.

Production of super absorber polymers (SAP)

Site-specific volumes of AA explicitly used for SAP production have been provided for almost 90% of AA used for external SAP-production. For almost 50% of the processing volume zero emission to the hydrosphere has been stated, justified by concise descriptions of employed wastewater reutilization / recycling systems. Specific emission data (measured effluent

\(^5\) Important parts of submitted information have been provided on a strictly confidential basis, since sensitive marketing interests are concerned. Therefore, a number of calculation results are presented in a way, which is intended to prevent recalculation of use tonnages, especially of sensitive downstream use branches. The underlying input data and calculations are confidentially filed by the responsible authorities.
concentrations, specific flow rates of wastewater site effluent, receiving WWTP, receiving river) provided for a further portion of AA tonnage used, enabling estimation of $C_{\text{local\_water}}$, resulted in values up to:

$$C_{\text{local\_water}} = 0.97 \ \mu g/l$$

Based on the known remaining annual AA tonnage not covered by the above-mentioned specific release data, and applying the respective default parameters according to the TGD, a

$$C_{\text{local\_water}} = 339 \ \mu g/l$$

is calculated.

**Wet polymerisation**

The volumes of AA applied for wet polymerisation have been provided for thirteen European sites, covering about 30% of AA used for external wet polymerisation:

<table>
<thead>
<tr>
<th>Number of plants</th>
<th>1</th>
<th>3</th>
<th>7</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Annual use of AA [t]</td>
<td>&lt; 500</td>
<td>500-1,000</td>
<td>1,000-5,000</td>
<td>5,000-10,000</td>
</tr>
</tbody>
</table>

Three further sites provided site-specific emission data without revealing their processing tonnages. For seven sites (covering ca. 12% of the tonnage), zero release to the hydrosphere has been stated, justified by a concise description of employed wastewater reutilization / recycling systems.

For eight sites processing > 500 t/a, a calculation of the $C_{\text{local\_water}}$ is performed based on site-specific tonnages or on measured AA effluent concentrations, incorporating site-specific information on wastewater treatment and dilution as far as available and default release factors if necessary. The underlying site-specific data are confidential. The results are compiled in Table 3.6.

![Table 3.6](image)

The remaining ca. 70% of AA used for external wet polymerisation are not covered by the site-specific calculations presented above. A default calculation, applying the respective parameters according to the TGD (fraction of main source, emission episode, emission factor, standard
WWTP flow, elimination rate of 87.3%, dilution in surface water), results in a \( C_{\text{local}} \) significantly exceeding 100 µg/l. With regard to the specific \( C_{\text{local}} \) values given in Table 3.6 above, considering the different degrees of site-specific information incorporated, it can be concluded that the indicated generic \( C_{\text{local}} \) represents a realistic worst case for this scenario “external wet polymerisation”.

### 3.1.3.4 Estimation of \( C_{\text{local}} \) / Generic approach: use of polymers

According to Table 2.1, AA based polymers are mainly used in dispersions, superabsorbents and for treatments of leather and textiles. Additionally, application of water treatment agents has been identified as a relevant use. During these uses, releases of AA are possible due to residual monomeric AA present in the respective products.

Measurements of the residual AA contents in polymer dispersions have been communicated by two AA producers and EPDLA (European Polymer Dispersion and Latex Association, 1998), giving the following results:

<table>
<thead>
<tr>
<th>Product Type</th>
<th>AA Content (ppm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Special polymeric dispersion</td>
<td>60</td>
</tr>
<tr>
<td>AA related polymeric dispersions</td>
<td>3-20</td>
</tr>
<tr>
<td></td>
<td>100-1000</td>
</tr>
<tr>
<td></td>
<td>1,000-2,500</td>
</tr>
<tr>
<td>Polymer latex</td>
<td>2.2</td>
</tr>
<tr>
<td>Desizing agents for textile finishing industry</td>
<td>110, 350, 370</td>
</tr>
<tr>
<td>Water treatment agents</td>
<td>200, 240, 400, 540, 960, 2,000</td>
</tr>
</tbody>
</table>

One producer stated residual monomeric AA contents in freshly manufactured products ranging from 260 to 4,000 grams per tonne, related to the initial amount of AA used. The results averaged 1,580 g/t. Compared to the above given range of residual monomeric contents in various products, the latter figure is independent from different portions of AA incorporated into different products. Therefore, this figure equals rather the quantity of monomer remaining unreacted after polymerisation of 1 tonne AA, the exact value primarily depending on applied polymerisation process engineering. This database is preferred for calculation of scenarios, where sufficiently specific residual monomeric AA contents are not known for the used products.

The application areas leather production, textile finishing, formulation of paints and application of water treatment agents are considered relevant for the assessment of local aquatic exposure scenarios. Private use of paints as well as use of superabsorber polymers (mainly in nappy pants, pantyliners and other consumer articles of hygiene) was not considered relevant for local assessment due to wide dispersion of these applications.

### Leather finishing

During leather production, various finishing processes are presumably the main application area of AA containing products. Grain leather is generally surface treated by the addition of lacquer and/or resin containing colorants and other additives in either aqueous or organic solvent systems. Leather products typically need several coatings, which contain pigments or metal-complex dyes in dispersion matrices. Emission Scenario Document ESD IC-7 is applied, based on the following assumptions (see Appendix A4 for calculation):

- Percentage of dispersion applied to leather: 5%.
- Maximum residual monomeric AA content of product: 4,000 g/t (actually related to initial AA amount),
gives 0.05 t · 4,000 g/t = 200 g/t residual AA content. According to the ESD, 15 tonnes of leather are processed daily at one site. The calculations presented in Appendix A4 result in:

\[ C_{\text{local}} = 0.95 \mu g/l \]

Due to insufficient information, two overly conservative assumptions are incorporated in this estimate: (1) the amount of residual AA is based on the assumption, that the applied dispersion contains 100% polyacrylate; (2) the calculation is based on 95% fixation degree, which appears to be an underestimation.

**Textile finishing**

In textile finishing, various processes are presumed to involve application of treatment agents containing AA based polymers. As an example, desizing agents are listed among the products, for which residual monomeric AA contents were communicated by industry (see above). Emission Scenario Document ESD IC-13 is applied, based on the following assumptions (see Appendix A5 for calculation):

- percentage of dispersion applied to textiles: 5%
- maximum residual monomeric AA content of product: 4000 g/t (actually related to initial AA amount)

\[ C_{\text{local}} = 0.19 \mu g/l \]

Due to insufficient information, two overly conservative assumptions are incorporated in this estimate: (1) the amount of residual AA is based on the assumption, that the applied dispersion contains 100% polyacrylate; (2) the calculation is based on 95% fixation degree, which appears to be an underestimation.

**Paint formulation**

The release of monomeric AA is possible during formulation of paints. In the Emission Scenario Document for paints, lacquers and varnishes industry (ESD IC-14, TGD, Chapter 7) no data are available on the release of monomeric substances during formulation of polymeric suspensions. The emissions are therefore estimated with the emission tables (Tables A2.1 and B2.3) presented in Appendix I of Chapter 3 of the TGD.

According to industry, about 15,000 t/a of AA are directly polymerised and used for manufacturing paints by downstream users. This equals a processing volume of 300,000 t/a dispersion solids according to EPDLa statement of 5% AA as typical use level in dispersion solids. Applying 0.4 as fraction of main source according to TGD Table B2.3, a use tonnage of 6,000 t/a AA (120,000 t/a dispersion solids) at one site would be assumed as a worst case.

Referring to additional information from industry, about 400 plants are formulating paints in Europe. Taking into account considerable ranges of plant size and degree of AA use, and considering the data on used AA tonnages as submitted by various downstream users (cf. Table 3.6), the processing volume of AA at one single site is assumed to be 2,000 t/a (equalling a processing volume of 40,000 t/a dispersion solids at this site) as a realistic worst case.
Considering a maximum monomeric AA content of 4,000 g/t, related to the initial AA tonnage, an amount of 2,000 t/a \( \cdot 0.004 = 8 \) t/a monomeric AA is assumed for this scenario.

Applying release factor 0.003 (Table A2.1), 300 d processing (Table B2.3) and 2,000 m\(^3\)/d WWTP flow, the local concentration is estimated to (see Appendix A6 for calculation):

\[
C_{\text{local, water}} = 0.51 \ \mu \text{g/l}
\]

**Application of water treatment agents**

Consideration of acrylic polymers in water treatment agents is based on the following information (Schumann 1997; TEGEWA 1999): sodium acrylate is used as co-monomer in anionic polyacrylamides, which serve as coagulation aids in water treatment. Areas of application are raw, drinking and wastewater treatment and mineral processing. Use in the area of drinking water processing is controlled by other fora. About 50% of the total tonnage are used for mineral processing, 60% of this being used in closed circuits, whereby the water is reused after removal of the mineral solids. The amount used in municipal wastewater treatment is of minor quantitative importance compared to cationic products.

A generic estimation of \( C_{\text{local, water}} \) is based on a specific application rate ranging around 1 mg/l. Taking into account residual monomeric AA contents communicated for water treatment agents (200 ... 2,000 mg/kg), expected concentrations of residual AA in treated water range to at most 2 \( \mu \)g/l. Assuming default dilution 1:10,

\[
C_{\text{local, water}} = 0.2 \ \mu \text{g/l}
\]

This value implies the additional worst-case assumption, that all residual monomeric AA is released to water and no portion of AA is biodegraded, neither incorporated in the sludge and thus removed from the water phase.

### 3.1.3.5 Measured levels

No monitoring data for the aquatic environment are available.

AA measurements had been performed in the drainage water from a tunnel construction site in Norway. Grouting agents are used to reduce water leakage in constructions like tunnels and building parts which are exposed to high inward pressure of groundwater or in sewer systems. During the application of a new grouting agent containing magnesium diacrylate, high concentrations of AA have been found in the drainage water of the Norwegian tunnel.

During application of the product (about 10 t per injection, total amount of 57 t) AA concentrations up to 5.6 mg/l were detected in the drainage water. After injection was terminated, the concentrations decreased rapidly (within one week) to a level between 10 and 100 \( \mu \)g/l.

The drainage water was collected in a treatment plant and the effluent concentrations were analysed. In general, AA concentrations did not exceed 100 \( \mu \)g/l, but when the influent concentrations were above 1,000 \( \mu \)g/l, AA elimination was not significant and during the injection periods (about 2 weeks per injection) effluent concentrations between 2,000 and 5,600 \( \mu \)g/l were measured.

These values represent a site-specific situation and extrapolation to other tunnel constructing sites may not be appropriate due to varying draining conditions.
From the measured effluent concentrations the Clocal\textsubscript{water} in the receiving surface water can be calculated. The effluent is transported via the river \textit{Alna} into the \textit{Oslofjord}. As the \textit{Alna} is partially covered by casing, it may not represent an ecosystem to be protected. The concentrations in the \textit{Oslofjord} can be estimated using a default dilution factor of 10 for dilution in the \textit{Alna} and an additional factor of 10 for dilution in the \textit{Oslofjord}.

According to the available reports on the tunnel construction site (Aquateam, 1999), the calculation can also be performed on the basis of specific information on dilution. The annual average flow of the \textit{Alna} of 75 m\textsuperscript{3}/min is divided by a factor of 3 to estimate a low flow of 25 m\textsuperscript{3}/min according to the TGD. The average flow of drainage water during the construction period (nearly one year) was 2,550 l/min. Dilution factors for the \textit{Alna} in the \textit{Oslofjord} are available for different distances from the mouth of the \textit{Alna}.

Regarding a realistic worst-case situation the measured effluent concentration of 5.6 mg/l is used for the calculations of the Clocal\textsubscript{water}:

<table>
<thead>
<tr>
<th>Distance (m)</th>
<th>100</th>
<th>200</th>
<th>300</th>
<th>400</th>
<th>500</th>
<th>Default</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dilution factor</td>
<td>2</td>
<td>6</td>
<td>9</td>
<td>13</td>
<td>18</td>
<td>10</td>
</tr>
<tr>
<td>Clocal\textsubscript{water} (µg/l)</td>
<td>280</td>
<td>93</td>
<td>62</td>
<td>43</td>
<td>31</td>
<td>56</td>
</tr>
</tbody>
</table>

### 3.1.3.6 Sediment

As neither monitoring data on concentrations of AA in sediment nor experimental results with benthic organisms are available and there is no evidence for relevant adsorption of AA onto sediment, there is no need to perform a quantitative risk assessment for this compartment. In addition, as AA dissociates in water and no correlation has been found between adsorption and organic carbon content for this substance the standard estimation method proposed in the TGD would not be applicable.

### 3.1.4 Atmosphere

#### 3.1.4.1 Estimation of Clocal\textsubscript{air} / Generic approach: production and processing

No use category document for the release of intermediates into the atmosphere during production and processing is available at the moment. The emissions are therefore estimated with the emission tables presented in Chapter 3, Appendix I of the TGD (1996). A production and processing volume of 330,000 t/a is used for the generic approach corresponding to the highest amount produced at one single site. It is assumed that production and on-site processing can be assigned to main category Ib (isolated intermediates). The vapour pressure at 20°C being in the range of 100 to 1,000 Pa, release fractions of 0.0001 during production and of 0.00001 during on-site processing are proposed. External processing is considered in Section 3.1.4.3.

For the generic scenario, a resulting release of 36.3 t/a is calculated. Applying the OPS-model the local concentration and total annual deposition rate are calculated to (Fmainsource = 1, Temission = 300 d/a, Fstp\textsubscript{air} = 0, Fass\textsubscript{aer} = 2.6 \times 10^{-7}; see Appendix A7 for calculation):

\[
\text{Clocal}_{\text{air}} = 34 \, \text{µg/m}^3
\]

\[
\text{DEP}_{\text{total,ann}} = 40 \, \text{µg} \cdot \text{m}^{-2} \cdot \text{d}^{-1}
\]
3.1.4.2 Estimation of C\text{local}_{\text{air}} / Site-specific approach: production and processing

In comparison to the generic approach a site-specific calculation using emission data submitted by industry leads to considerably lower values:

Table 3.7 Site-specific emissions to air, C\text{local}_{\text{air}} and DEP\text{total}_{\text{ann}}

<table>
<thead>
<tr>
<th>Site</th>
<th>Emission to air [t/a]</th>
<th>C\text{local}_{\text{air}} [µg/m}^3]</th>
<th>DEP\text{total}_{\text{ann}} [µg/m}^2/d]</th>
<th>Specific data used for calculation</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>8</td>
<td>6</td>
<td>9</td>
<td>specific release to air; no emission to air from WWTP</td>
</tr>
<tr>
<td>B</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>neglected due to incineration and flue-gas washing</td>
</tr>
<tr>
<td>C</td>
<td>4.8</td>
<td>4</td>
<td>5</td>
<td>specific release to air; no emission to air from WWTP</td>
</tr>
<tr>
<td>D</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>specific release to air; no emission to air from WWTP</td>
</tr>
<tr>
<td>E</td>
<td>0.5</td>
<td>0.5</td>
<td>0.4</td>
<td>default release to air (emission factor for processing: 0.0001); no emission to air from WWTP</td>
</tr>
<tr>
<td>F</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>no production or import since 1995</td>
</tr>
<tr>
<td>G</td>
<td>1.7</td>
<td>1</td>
<td>2</td>
<td>specific release to air; no emission to air from WWTP</td>
</tr>
<tr>
<td>H</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>no production or import since 1994</td>
</tr>
<tr>
<td>I</td>
<td>&lt; 0.025</td>
<td>&lt; 0.02</td>
<td>&lt; 0.03</td>
<td>specific release to air; no emission to air from WWTP</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

3.1.4.3 Estimation of C\text{local}_{\text{air}}/ Generic approach: processing by non-producers/importers

Specific information on emissions to atmosphere is known only for a few downstream user sites stating zero emission to atmosphere. For one big processing site specific emission data to air have been communicated. The resulting emission factor is $7.3 \times 10^{-7}$. It should be noted, that this specific site applies very efficient air clean-up measures while the corresponding releases to hydrosphere are considerably higher (emission factor $1.2 \times 10^{-3}$ based on measured effluent concentrations). Additionally, emission data have been submitted for a number of individual processing sites located on-site at a large AA production site. Resulting emission factors to air range between $1 \times 10^{-7}$ and $3.4 \times 10^{-4}$. However, there were no data made available to the authors of this report, which could serve as a basis for reliable emission factors being representative for all types of downstream user sites.

All the main processing types applied externally (SAP production, wet polymerisation and dry polymerisation) comprise wet and dry polymerisation techniques. For AA, both wet and dry techniques are allocated to the same default emission factor of 0.01 (TGD, Table A3.10). However, for a generic calculation of downstream user emissions to atmosphere it appears reasonable to lower this factor to 0.001 with regard to the emission factors reported above. This approach covers the worst-case assumption, that the highest specific overall emissions out of all known sites would be directed entirely to air.

According to the comprehensive set of specific confidential information, provided by a significant portion of AA downstream users with regard to releases to the hydrosphere (cf.
Section 3.1.3.3), an amount of 10,000 t/a processing volume at one single site is assumed as realistic worst case.

Applying the respective default parameters according to the TGD (Table B3.9: emission episode 300 d - see Appendix A8 for calculation), gives:

\[
\begin{align*}
\text{C}_{\text{local\_air}} &= 9.3 \, \mu g/m^3 \\
\text{DEP}_{\text{total\_ann}} &= 11 \, \mu g \cdot m^{-2} \cdot d^{-1}
\end{align*}
\]

The end-use of polymer products (e.g. formulation and use of leather and textile finishing agents and paints) is considered to be not relevant for environmental exposure to monomeric AA on a local scale.

### 3.1.5 Terrestrial compartment

The release of AA to soil is expected to occur through atmospheric deposition after local release to the atmosphere at the production and processing site (see Section 3.1.4). The input through sludge application on agricultural soil is considered to be negligible, as AA does not partition to a significant extent to sewage sludge in the WWTP (see Section 3.1.1).

Direct releases to soil are possible due to the use of superabsorbents for soil amelioration. However, this application is assumed to be restricted to special areas like landfills or specific urban soils and is therefore considered as not relevant for natural soil.

**Generic approach**

With the generic deposition rate calculated in Section 3.1.4.1 \((\text{DEP}_{\text{total\_ann}} = 40 \, \mu g \cdot m^{-2} \cdot d^{-1})\), equilibrium soil concentrations in direct vicinity to the generic production / processing plant can be calculated according to the TGD (see Appendix A9). The result concerning the ecosystem is:

\[
\begin{align*}
\text{C}_{\text{local\_soil}} &= 2.4 \, \mu g/kg \\
\text{C}_{\text{local\_soil-porewater}} &= 2.4 \, \mu g/l
\end{align*}
\]

(other non-default input data: \(K_{\text{soil\_water}} = 1.7, \, K_{\text{air\_water}} = 1.1 \cdot 10^{-5}\))

The generic deposition rate calculated in Section 3.1.4.3 for external processing sites is significantly lower \((\text{DEP}_{\text{total\_ann}} = 11 \, \mu g \cdot m^{-2} \cdot d^{-1})\). Therefore, the \(\text{C}_{\text{local\_soil}}\) resulting for production and processing sites covers all downstream user scenarios.

**Site-specific approach**

The local concentrations in soil calculated with site specific deposition rates for the known production and processing sites A to I (cf. Table 3.7) are significantly lower, for most sites below 0.5 \(\mu g/kg\), and range to a maximum of 1.1 \(\mu g/kg\). No specific deposition rates are known for the external processing sites of downstream user companies.
3.1.6  Secondary poisoning

As AA does not present indications of a bioaccumulation potential, a quantitative risk assessment for secondary poisoning is not required.

3.1.7  Regional concentrations

For determination of a regional background concentration all releases, from both point and diffuse sources, are considered. 20% of the total exposure quantity from point sources is taken into account for the defined regional EU standard model (densely populated area of 200·200 km with 20 million inhabitants), since AA is produced and processed in large tonnage at local sites. Therefore, the EUSES 1.0 default value for the fraction related to the region of 0.1 has been set to 0.2 for a more realistic regional scenario. This assumption is confirmed by the specific information recently provided by downstream users.

From diffuse sources, the default of 10% is considered for the standard region. The rest (80% from point sources and 90% from diffuse sources) of the total exposure quantity is taken into account for the continental model.

No direct release into the soil was identified. Diffuse release only occurs as a result of dispersal processes. Release is therefore to be expected as a result of deposition from the air (see Section 3.1.4).

Since not all of the previously mentioned releases arising from use of the substance enter the hydrosphere directly, but instead primarily via the wastewater which is possibly purified in municipal wastewater treatment plants, a 70% connection to wastewater treatment plants, in which 87.3% of the substance is eliminated, is assumed for this scenario. The remaining 30% of the water is discharged directly into the hydrosphere.

Point releases

In Table 3.5 the total annual release of 12.5 t/a AA to hydrosphere is allocated to all production and processing sites. The annual amount released to atmosphere totals 18 t/a (cf. Table 3.7). Point releases due to external processing and use are calculated for some generic exposure scenarios and for a number of specific sites as described in Sections 3.1.3.3 and 3.1.4.3. In order to keep the sensitive marketing figures confidential, overall sums are calculated for all releases to hydrosphere and atmosphere, which have been estimated for:

- SAP production based on site-specific information
- SAP production based on default calculation
- wet polymerisation based on site-specific information
- wet polymerisation based on default calculation
- dry polymerisation based on site-specific information
- dry polymerisation based on default calculation

The resulting releases are totalled up to 113 t to hydrosphere and 190 t to atmosphere.

Releases of residual monomeric AA during handling, use and disposal of AA based polymeric products are considered in an overall estimate as diffuse releases (see below). Consequently, the use scenarios considered in Section 3.1.3.4 (leather and textile finishing, formulation of paints, water treatment agents) have been calculated only for local exposure assessment for illustrative purposes.
Diffuse releases

For diffuse releases a conservative estimation is carried out, using the EU consumption amount of 830,000 t/a. A portion of about 38% of this (315,000 t/a) is polymerised to various AA-based polymeric products. Freshly manufactured products contain 260 ... 4,000 g/t monomeric AA, related to the initial amount of AA used (cf. Section 3.1.3.4). The average value of 1,580 g/t is used for calculation. Due to additional reaction steps, acrylate esters contain only about a hundredth of the residual monomeric AA reported above.

During use and disposal of the products the residual monomer can be washed out or evaporate. For the resulting monomer amount of 513 t/a (including 15 t/a residual monomeric AA from acrylate esters) it is assumed that 80% of the monomer (410 t/a) is released during formulation, use and disposal and that 20% remains in products which are incinerated.

As the aquatic compartment is the target compartment of AA, it is assumed that 70% of the releases (287 t/a) occur into the hydrosphere and 30% (123 t/a) into the atmosphere.

With a connection rate of 70% to WWTPs, 86 t/a are calculated to be released directly and 201 t/a into WWTPs of which 26 t/a (12.7%) are released with the effluent to surface water.

As mentioned at the beginning of this Section 3.1.7, for production, processing and formulation 20% of the point-source releases are assumed to occur into a region whereas according to the TGD from the diffuse releases only 10% are considered for the region.

The individual environmental releases are summarised in Tables 3.8 and 3.9:

Table 3.8  Summary of environmental releases

<table>
<thead>
<tr>
<th>Life cycle stage</th>
<th>Distribution to regional / continental model [%/%]</th>
<th>Release into hydrosphere [t/a]</th>
<th>Release into atmosphere [t/a]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Production and on-site processing of AA</td>
<td>20/80</td>
<td>Direct 0</td>
<td>WWTP 99</td>
</tr>
<tr>
<td>External processing by downstream users</td>
<td>20/80</td>
<td>Direct 0</td>
<td>WWTP 891</td>
</tr>
<tr>
<td>Diffuse releases during handling, use and disposal of polymeric products</td>
<td>10/90</td>
<td>Direct 86</td>
<td>WWTP 201</td>
</tr>
</tbody>
</table>

Table 3.9  Environmental releases for calculation of continental and regional model

<table>
<thead>
<tr>
<th></th>
<th>Continental model [t/a]</th>
<th>Regional model [t/a]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>0.8 · (18+190)t/a + 0.9 · 123 t/a = 277 t/a</td>
<td>0.2 · (18+190)t/a + 0.1 · 123 t/a = 54 t/a</td>
</tr>
<tr>
<td>Soil</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Water direct</td>
<td>0.9 · 86 t/a = 77.4 t/a</td>
<td>0.1 · 86 t/a = 8.6 t/a</td>
</tr>
<tr>
<td>into WWTPs</td>
<td>0.8 · (99+891)t/a + 0.9 · 201 t/a = 973 t/a</td>
<td>0.2 · (99+891)t/a + 0.1 · 201 t/a = 218 t/a</td>
</tr>
</tbody>
</table>
In Appendix A10 the input and output figures of a SimpleBox 2.0 calculation adapted to the TGD and EUSES 1.00 are presented. The results of this calculation are consistent with EUSES. The resulting regional concentrations are:

- \( \text{PEC}_{\text{regional}}^{\text{aquatic}} = 0.40 \ \mu\text{g/l} \)
- \( \text{PEC}_{\text{regional}}^{\text{air}} = 2 \ \text{ng/m}^3 \)
- \( \text{PEC}_{\text{regional}}^{\text{agr.-soil}} = 0.02 \ \mu\text{g/kg (wwt)} \)
- \( \text{PEC}_{\text{regional}}^{\text{agr.-soil_porew}} = 0.02 \ \mu\text{g/l} \)
- \( \text{PEC}_{\text{regional}}^{\text{natural-soil}} = 0.07 \ \mu\text{g/kg (wwt)} \)

The calculated regional and continental distributions depend on vapour pressure, log Pow and Henry’s law constant which are only valid for the undissociated form of AA. Since no other models are available to calculate the distribution for anionic AA, the values above are taken as an approximation.
CHAPTER 3. ENVIRONMENT

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

3.2.1 Aquatic compartment

3.2.1.1 Available effect data

In the following, the most relevant results from acute and long-term toxicity tests with aquatic organisms are presented. Other results are available, but they are not valid as pH effects cannot be excluded (see IUCLID).

Vertebrates

*Leuciscus idus*

(48-h LC$_{50}$) 315 mg/l

(Juhnke and Lüdemann, 1978)

*Brachydanio rerio*

(96-h LC$_{50}$) 222 mg/l

(Hüls, 1995b)

*Oncorhynchus mykiss*

(96-h LC$_{50}$) 27 mg/l

(Bowman, 1990)

Invertebrates

*Daphnia magna*

(24-h EC$_{50}$) 765 mg/l

(Bringmann and Kühn, 1982)

*Daphnia magna*

(48-h EC$_{50}$) 95 mg/l

(Burgess, 1989)

*Daphnia magna*

(48-h EC$_{50}$) 47 mg/l

(Hüls, 1995c)

*Daphnia magna*

(21-d NOEC) 7 mg/l

(Hüls, 1995d)

Plants

*Microcystis aeruginosa* (blue-green algae)

(8-d TGK) 0.15 mg/l

(Bringmann and Kühn, 1977; 1978a)

*Scenedesmus quadricauda*

(8-d TGK) 18 mg/l

(Bringmann and Kühn, 1977; 1978a)
The TGKs or “toxic threshold concentrations” were determined at 3% effect compared to the controls and can therefore be considered as NOECs.

**Selenastrum capricornutum**
- 96-h EC$_{50}$: 0.17 mg/l
- (effect: biomass; nominal concentrations;
  NOEC could not be determined, <0.13 mg/l)
  (Forbis, 1989)

**Scenedesmus subspicatus**
- 72-h E$_{b}$C$_{50}$: 0.04 mg/l
- 72-h E$_{b}$C$_{10}$: 0.01 mg/l
- 72-h NOEC: 0.008 mg/l
- 72-h E$_{c}$C$_{50}$: 0.13 mg/l
- 72-h E$_{c}$C$_{10}$: 0.03 mg/l
- 72-h NOEC: 0.016 mg/l
  (BASF, 1994b)

**Scenedesmus subspicatus**
- 72-h E$_{b}$C$_{50}$: 0.06 mg/l
- 72-h E$_{b}$C$_{10}$: 0.01 mg/l
- 72-h NOEC: <0.01 mg/l
- 72-h E$_{c}$C$_{50}$: 0.205 mg/l
- 72-h E$_{c}$C$_{10}$: 0.031 mg/l
- 72-h NOEC: 0.025 mg/l
  (Hüls, 1995e)

**Protozoa**

**Entosiphon sulcatum**
- 72-h TGK: 20 mg/l

**Uronema parduczi**
- 20-h TGK: 11 mg/l

**Chilomonas paramecium**
- 48-h TGK: 0.9 mg/l
  (effect: biomass; nominal concentrations),
  (Bringmann and Kühn, 1978a; 1980)

The TGKs or “toxic threshold concentrations” were determined at 5% effect compared to the controls and can therefore be considered as NOECs.

**Bacteria**

**Pseudomonas putida**
- 16-h TGK: 41 mg/l
  (effect: biomass),
  (Bringmann and Kühn, 1977)

The TGK or “toxic threshold concentration” was determined at 3% effect compared to the controls and can therefore be considered as a NOEC.

**activated sludge**
- 30-min EC$_{20}$: 900 mg/l
- 30-min NOEC: 100 mg/l
  (effect: respiration inhibition; nominal concentration)
  (BASF, 1993)
### 3.2.1.2 Determination of PNEC_{aquatic}

The above-cited test results with acrylic acid are only those which show a clear substance related toxicity and no pH effects. Acute and long-term tests reveal that algae are the most sensitive organisms. As their EC\textsubscript{50} and NOEC values are more than two orders of magnitude lower than those for species of other trophic levels it is obvious that acrylic acid shows a specific toxicity to algae although for marine algae a high natural AA content is reported. (The result of one algae test showing a NOEC of 18 mg/l might be caused by recovery over a longer test period of eight days.)

For growth rate reduction, the lowest EC\textsubscript{10} value derived in two tests (BASF, 1994b; Hüls, 1995e) was 0.03 mg/l for \textit{Scenedesmus subspicatus}. The respective values based on biomass reduction are \( \leq 0.01 \) mg/l. Striking correspondence is noted regarding most resulting EC values of these two independently conducted tests.

A recent study investigated the influence of growth pattern on effective concentrations (EC) of cell number, biomass integral and growth rate in the alga growth inhibition test (AGIT), systematically evaluating 38 existing AGITs and investigating several test scenarios by simulated AGITs (Ratte, 1998). In summary, it was concluded that preference of EC\textsubscript{biomass} or EC\textsubscript{growth rate} has to be based on a case-by-case decision, depending on various experimental conditions. For the majority of test scenarios, growth rate was found to provide the most reliable estimate of “true” toxicity, featuring the advantages of

- less susceptibility to experimental disturbances,
- independence of test duration,
- better statistics, and
- immediate ecological relevance.

Neither of the \textit{Scenedesmus} test reports gives specific cause to prefer estimates based on biomass. Since a NOEC value depends on individual test design (intervals of test concentrations, number of replicates, variability of treatments and control), it is preferred to use available EC\textsubscript{10} values for PNEC derivation, provided that a smooth concentration-response curve is obtained as in the present tests. Keeping in mind the above-mentioned arguments for using effect estimates based on growth rate, the PNEC\textsubscript{aqua} is derived from the E\textsubscript{µ}C\textsubscript{10} values. This conclusion is confirmed in a critical re-evaluation of the relevant AGITs by a distinguished expert (Nyholm, 1999).

Although long-term NOECs/EC\textsubscript{10}-values are available from only two trophic levels, an assessment factor of 10 can be chosen because of the comparatively high toxicity of AA to algae. The acute EC\textsubscript{50} values for fish are in the same range as those for daphnids and with high probability a NOEC for fish will not be lower than that of algae.

Therefore: \[ PNEC_{aqua} = \frac{30 \text{ µg/l}}{10} = 3 \text{ µg/l}. \]

### 3.2.1.3 Determination of PNEC\textsubscript{microorganisms}

There are three test results available which can be used for the derivation of this PNEC. According to the different endpoints and sensitivities of the test systems different assessment factors (AF) have to be applied:
As a worst-case assumption a PNEC\textsubscript{microorganisms} of 0.9 mg/l has to be used in the risk characterisation for municipal wastewater treatment plants.

For industrial on-site plants, a PNEC\textsubscript{microorganisms} of 10 mg/l is proposed.

### 3.2.2 Atmosphere

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

### 3.2.3 Terrestrial compartment

To assess the effects of acrylic acid on terrestrial organisms, only a test on the respiration inhibition of natural soil microflora is available (Hossack et al., 1992). During 28 days of exposure, 100 mg/kg dry weight had no effects, while 1,000 mg/kg completely blocked the respiration rate. An EC\textsubscript{50} was not determined. A light sandy loam soil with an organic carbon content of 0.3% was used.

**Determination of PNEC\textsubscript{soil}**

Based on the above cited soil respiration test a derivation of a PNEC is possible. With an assessment factor of 1,000 a PNEC\textsubscript{soil} of 0.1 mg/kg would result. A conversion to standard soil is not necessary as the organic content of the soil is not critical for the adsorption behaviour of AA. On the other hand, according to the TGD, Chapter 3.6.2.1, the aquatic PNEC can be used for the risk characterisation of the soil ecosystem.

With a PNEC\textsubscript{water} = 3 µg/l and a K\textsubscript{soil-water} = 1.7 m³/m³, the PNEC\textsubscript{soil} amounts to 3 µg/kg. As this is the lower value it will be taken for comparison with the PEC\textsubscript{soil}.

### 3.2.4 Secondary poisoning

As AA does not present indications of a bioaccumulation potential, an effect assessment for secondary poisoning is not required.
3.3 RISK CHARACTERISATION

3.3.1 Aquatic compartment

3.3.1.1 Wastewater treatment plants

There is a need for further information and/or testing.

Because of the significant differences in responsibilities, functional control measures and data quality the possible risk to microorganisms is evaluated separately for municipal and industrial wastewater treatment plants (WWTP).

All WWTP at production and processing sites are considered as industrial plants (except site D, based on specific information). The highest effluent concentrations for industrial plants are reported for the external processing site O, which is equipped with an on-site WWTP:

\[
\text{PEC}_{\text{microorganisms}} = 1 \text{ mg/l} \quad \text{(industrial plant site O, measured conc.)}
\]

With a \(\text{PNEC}_{\text{microorganisms}}\) of 10 mg/l for industrial plants, \(\text{PEC}/\text{PNEC} \ll 1\) for all respective industrial sites.

The following effluent concentrations were calculated for the standard treatment plants of 2,000 m\(^3\)/d which might be considered as municipal plants, and for specific municipal plants, if respective data have been available:

### Table 3.10 Calculation of effluents concentrations for wastewater treatment plants

<table>
<thead>
<tr>
<th>PEC(_{\text{microorganisms}}) [(\mu\text{g/l})]</th>
<th>Scenario</th>
</tr>
</thead>
<tbody>
<tr>
<td>246 (^1)</td>
<td>production site D, specific</td>
</tr>
<tr>
<td>3,390</td>
<td>external SAP production, wet process, default</td>
</tr>
<tr>
<td>990</td>
<td>external SAP production, wet process, highest specific</td>
</tr>
<tr>
<td>(\gg 1,000)</td>
<td>external wet polymerisation, default</td>
</tr>
<tr>
<td>0.04</td>
<td>external wet polymerisation, site K</td>
</tr>
<tr>
<td>19,050 (^2)</td>
<td>external wet polymerisation, site L</td>
</tr>
<tr>
<td>283</td>
<td>external wet polymerisation, site M</td>
</tr>
<tr>
<td>76</td>
<td>external wet polymerisation, site N</td>
</tr>
<tr>
<td>12,700 (^2)</td>
<td>external wet polymerisation, site Q</td>
</tr>
<tr>
<td>21,200</td>
<td>external wet polymerisation, generic site</td>
</tr>
<tr>
<td>9.5</td>
<td>leather finishing</td>
</tr>
<tr>
<td>1.9</td>
<td>textile finishing</td>
</tr>
<tr>
<td>5.1</td>
<td>formulation of paints</td>
</tr>
<tr>
<td>2</td>
<td>application of wastewater treatment agents</td>
</tr>
</tbody>
</table>

\(^1\) based on measured discharge concentration (90\%ile), further dilution on entry into WWTP, elimination according to SIMPLETREAT

\(^2\) based on measured wastewater concentration, elimination in WWTP considered according to SIMPLETREAT - the respective specific sites communicated the statement "no biological WWTP"; nevertheless, these values are considered as realistic worst cases for a treatment plant
With a PNEC\textsubscript{microorganisms} of 900 µg/l for municipal plants, PEC/PNEC \textgreater\textless 1 for the downstream use scenarios of SAP production (default calculation and highest site-specific PEC figures) and wet polymerisation (default calculation and known sites L, Q).

An improvement of exposure data may in principle be possible e.g. by performing effluent measurements. However, PEC\textsubscript{microorganisms} for the known processing sites L and Q are based on measured AA effluent concentrations (ranges 40 … 100 mg/l and 50 … 150 mg/l). Likewise, the PEC\textsubscript{microorganisms} for a large known SAP producing site is based on measured effluent concentrations (ranges 40 … 100 mg/l and 50 … 150 mg/l). These site-specific data indicate, that high effluent concentrations cannot be excluded. At the same time, gathering of sufficiently complete information for all downstream user sites applying wet polymerisation techniques seems not achievable with reasonable expenditure of time and money.

Since the PNEC\textsubscript{microorganisms} is derived from single species tests with ciliated protozoa, there is a need for further data reflecting the integrity of the native ciliate population in sewage sludge as a whole. However, regarding the conclusion for surface water (see below), it is accepted to postpone this testing need, since risk reduction measures necessary to remove concern for surface water will also cover the protection of wastewater treatment plants.

3.3.1.2 Surface water

In Table 3.11 the comparison between PEC\textsubscript{local} and PNEC\textsubscript{aqua} for all relevant exposure scenarios are presented (PNEC = 3 µg/l). As described in Section 3.1.7, the PEC\textsubscript{regional} is calculated to be 0.4 µg/l.

Regional model

The release assessment for the regional model was carried out on the basis of several calculated default emissions. Although received specific emission data have lowered the PEC\textsubscript{regional}, it appears as impossible to gain specific and actual emission data for the whole area of downstream use with reasonable expenditure of time and money.

Local assessments

There is a need for further testing / gathering of exposure information or for limiting the risk.

As for several exposure scenarios PEC/PNEC \textgreater\textless 1, a risk for the aquatic compartment has to be deduced for the present data. Table 3.11 gives an overview on all scenarios considered.
### Table 3.11 Clocal, PEClocal and PEC/PNEC ratios for hydrosphere

<table>
<thead>
<tr>
<th>Scenario</th>
<th>C(\text{local} ) [µg/l]</th>
<th>PEC(\text{local} ) [µg/l]</th>
<th>PEC/PNEC</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Production / processing at site</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>0.64</td>
<td>1.1</td>
<td>0.4</td>
</tr>
<tr>
<td>B</td>
<td>0.16</td>
<td>0.6</td>
<td>0.2</td>
</tr>
<tr>
<td>C</td>
<td>0.77</td>
<td>1.2</td>
<td>0.4</td>
</tr>
<tr>
<td>D</td>
<td>6.6</td>
<td>7.0</td>
<td>2.3*</td>
</tr>
<tr>
<td>E</td>
<td>0</td>
<td>0.4</td>
<td>0.1</td>
</tr>
<tr>
<td>F</td>
<td>0</td>
<td>0.4</td>
<td>0.1</td>
</tr>
<tr>
<td>G</td>
<td>0</td>
<td>0.4</td>
<td>0.1</td>
</tr>
<tr>
<td>H</td>
<td>0</td>
<td>0.4</td>
<td>0.1</td>
</tr>
<tr>
<td>I</td>
<td>0.04</td>
<td>0.5</td>
<td>0.2</td>
</tr>
<tr>
<td><strong>External processing</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAP production (wet)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>default</td>
<td>339</td>
<td>339</td>
<td>113</td>
</tr>
<tr>
<td>highest specific</td>
<td>0.97</td>
<td>1.4</td>
<td>0.5</td>
</tr>
<tr>
<td>Polymerisation (wet)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>unknown sites default</td>
<td>&gt;&gt; 100</td>
<td>&gt;&gt; 100</td>
<td>&gt;&gt; 33</td>
</tr>
<tr>
<td>generic site</td>
<td>2,120</td>
<td>2,120</td>
<td>707</td>
</tr>
<tr>
<td><strong>Known external processing sites</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>K</td>
<td>0.004</td>
<td>0.4</td>
<td>0.1</td>
</tr>
<tr>
<td>L</td>
<td>0.18</td>
<td>0.6</td>
<td>0.2</td>
</tr>
<tr>
<td>M</td>
<td>1.25</td>
<td>1.7</td>
<td>0.8</td>
</tr>
<tr>
<td>N</td>
<td>8.7</td>
<td>9.1</td>
<td>3.0</td>
</tr>
<tr>
<td>O</td>
<td>12.7</td>
<td>13.1</td>
<td>4.4</td>
</tr>
<tr>
<td>P</td>
<td>0.2</td>
<td>0.6</td>
<td>0.2</td>
</tr>
<tr>
<td>Q</td>
<td>10,000</td>
<td>10,000</td>
<td>3,333</td>
</tr>
<tr>
<td>R</td>
<td>0.22</td>
<td>0.62</td>
<td>0.2</td>
</tr>
<tr>
<td><strong>Use in leather finishing (default)</strong></td>
<td>0.95</td>
<td>1.5</td>
<td>0.5</td>
</tr>
<tr>
<td><strong>Use in textile finishing (default)</strong></td>
<td>0.19</td>
<td>0.6</td>
<td>0.2</td>
</tr>
<tr>
<td><strong>Use in paint formulation (default)</strong></td>
<td>0.51</td>
<td>0.9</td>
<td>0.3</td>
</tr>
<tr>
<td><strong>Use of grouting agent (default)</strong></td>
<td>56</td>
<td>56</td>
<td>19</td>
</tr>
<tr>
<td>100 m from the mouth of river</td>
<td>280</td>
<td>280</td>
<td>93</td>
</tr>
</tbody>
</table>

* Site D: manufacture of AA ceased by October 1999 (confirmed by operating company in March 2000)

**Producers / importers**

At site D, the respective \(C_{\text{local}}\) exceeds the PNEC by a factor of more than two. The calculation is based on measurements of AA concentrations in WWTP influent. Specific updated dilution factors have been provided. For a refined estimate of site-specific releases to hydrosphere, detailed results of discharge monitoring and specific flow rates have been submitted. 20% out of the 281 24-hour composite samples showed AA concentrations resulting in PEC/PNEC ratios exceeding one. Nonetheless, further risk reduction measures are not necessary for this site because AA manufacture was ceased by October 1999 (confirmed by operating company in March 2000).

**External processing, use of polymers**

For several known downstream users where AA is applied for wet polymerisation processes, for the default scenarios of wet polymerisation and wet SAP production covering tonnages without
specific release data, and for the use of a grouting agent, PEC/PNEC ratios above one are calculated and a risk for the aquatic compartment has to be deduced on the basis of the present data. Risk reduction measures are recommended.

As an assessment factor of 10 is used for PNEC derivation, it is not likely to remove the concern by further testing.

For the wet polymerisation scenarios an improvement of exposure data may in principle be possible e.g. by performing effluent measurements. However, it is questionable whether sufficiently complete representative monitoring data from all downstream users can be obtained with reasonable expenditure of time and money. Moreover, regarding the dynamic year-to-year variations of used AA amounts, being typical for this market, the goal of sufficiently comprehensive collection of up-to-date monitoring data appears not appropriate. As additional background for the present risk characterisation, the following points should be noted:

- Site-specific information accounts for ca. 90% of AA externally used in SAP production and for ca. 30% of AA externally used in wet polymerisation processes. Only a limited part of this information is sufficient for derivation of emission factors, the calculated figures ranging between $2 \cdot 10^{-6}$ and $1.2 \cdot 10^{-3}$ (TGD default $1 \cdot 10^{-2}$).

- For both SAP production sites and wet polymerisation sites, regular effluent concentrations up to 100 mg/l AA and significantly more have been reported. At two of these sites, comparatively low $C_{\text{local}}$ figures result only because of high dilution factors (magnitude $10^6$). However, these data indicate that high effluent concentrations cannot be excluded, even if certain types of process engineering are applied.

- On the other hand, application of wastewater reutilization / recycling systems is known to result in zero emissions to the hydrosphere at a number of downstream user sites which are processing about 50% of AA used externally for SAP production and about 12% of AA used externally in wet polymerisation processes.

During the use of a grouting agent containing magnesium diacrylate high concentrations of AA are released via drainage water. The exposure assessment is based on measured concentrations of drainage water at a tunnel construction site. A quantitative extrapolation to other construction sites seems difficult, but similar conditions might be anticipated. Measures appropriate to local circumstances should be applied.

The use of water based emulsion polymers, containing residual AA, covers a wide range of applications, different products and used technologies making it nearly impossible to calculate a resulting PEC for each application. Therefore it has to be noted that most of the polymer applications could not be assessed specifically in Section 3.1.3.4 due to the lack of representative information for emulsion acrylates.

### 3.3.1.3 Sediment

Neither monitoring data on concentrations of AA in sediment nor experimental results with benthic organisms are available. As there is no evidence for relevant adsorption of AA onto sediment, there is no need for performing a quantitative risk assessment for this compartment. From the current manufacturing and use of AA no risk for the sediment compartment is expected.
3.3.2 Atmosphere

Due to the physical properties of AA and the Mackay distribution (see Section 3.1.1) the atmosphere is not regarded as a target of distribution. Emissions of AA into the atmosphere occur only on a local scale (concentrations in a distance of 100 m to the point source are between <0.02 and 9.3 µg/m³). Furthermore AA reveals a short half-life for atmospheric photooxidation of 39.6 h (oxidation by hydroxyl radicals) or 6.5 d (oxidation by ozone). With these data the regional PEC for the atmosphere was calculated to 2 ng/m³. Because of these features a risk to the atmosphere by abiotic or biotic effects is not to be expected and the performance of a plant fumigation test is not considered as high priority.

3.3.3 Terrestrial compartment

A generic exposure scenario representing a worst-case situation in the vicinity of a production and processing site was used for the calculation of the concentration in the soil porewater due to atmospheric deposition. This scenario covers also the refined generic estimation for exposure of soil in vicinity of a downstream user site applying wet or dry polymerisation processes, including SAP production.

The $C_{\text{local, soil-porewater}}$ was 2.4 µg/l in the vicinity of such a site and on a regional scale a concentration of 0.02 µg/l was estimated.

An indicative risk assessment can be performed on the basis of the aquatic PNEC resulting in a PNEC$_{\text{soil}}$ of 3 µg/l (soil pore water, cf. Section 3.2.3). Considering this PNEC as a quite conservative figure, the resulting PEC/PNEC ratio of 0.8 can be regarded as maximum for the generic worst-case scenario production and processing. For the site-specific scenarios for production and processing as well as for the generic scenario for external processing PEC/PNEC ratios between < 0.16 and 0.36 are resulting.

For the aquatic compartment, algae were the most sensitive species tested. Extrapolation of the toxicity to terrestrial plants is difficult due to physiological and exposure route differences. Thus, performance of a terrestrial plant test would be desirable. However, as the PNEC derived from aquatic tests is more than 30 times lower than the PNEC derived from the available soil microorganism test and in addition the exposure of the terrestrial compartment is only a local problem, additional testing of higher plants is not considered as high priority.

Therefore, no risk for the soil compartment is expected from the current manufacturing and use of AA.

3.3.4 Secondary poisoning

As AA does not present indications of a bioaccumulation potential, a risk characterisation for secondary poisoning is not required.
4 HUM AN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1 General discussion

In the Swedish product register, 13 out of 56 products containing acrylic acid are used by consumers.

In the Federal Republic of Germany, products containing acrylic acid are also used by consumers. Thus, the consumer may be exposed to acrylic acid. This may occur via the inhalation, dermal or oral route.

For workers the inhalation and dermal exposure routes are the most likely.

Acrylic acid is primarily used as a chemical intermediate which is further processed to acrylic esters, homopolymers and copolymers. For some of the processed products e.g. water treatment materials and sizing preparations manufactured from acrylic acid the content of the residual monomer is known (less than 900 ppm). Further information on the monomer content in other products such as paints is not available. By comparison with the concentration of monomeric methacrylic acid in paints (up to 700 ppm) it may be concluded, that the content of acrylic acid lies in the same range. Adhesives may contain up to 10% acrylic acid.

Acrylic acid may arise as a decomposition product during the production of printed circuit boards and during the removal of paints using gas flames.

The substance may also be released during the use of grouting agents.

4.1.1.2 Occupational exposure

The following occupational exposure limits are established for acrylic acid (ILO, 1994):

UK, CH, S, US (NIOSH/OSHA) 30 mg/m³ (10 ml/m³)
B 29 mg/m³ (10 ml/m³)
AUS, US(ACGIH), NL*, DK**, F 5.9 mg/m³ (2 ml/m³)

and the following short-term exposure limits are established for acrylic acid (ILO, 1994):

UK 60 mg/m³ (20 ml/m³)
S 45 mg/m³ (15 ml/m³)
F 30 mg/m³ (10 ml/m³)

* De Nationale MAC lijst (Ministerie van Sociale Zaken en Werkgelegenheid, 1996)
** Grænseværdier for stoffer og materialer (Arbejdstilsynet, 1996)
For occupational exposure, exposures during the
- production and further processing of acrylic acid,
- manufacture of adhesives,
- use of adhesives containing acrylic acid,
- decomposition of photoresist materials,
- gas flame removal of paints
is considered.

Acrylic acid may be released from grouting agents used to reduce water leakage in constructions like tunnels and building parts which are exposed to high inward pressure of groundwater or in sewer systems. During application AA concentrations up to 5.6 mg/l were detected in the drainage water. For the highly automated rehabilitation method in use (injection procedure) exposure may occur during tasks like cleaning of the equipment. The exposure level depends on the dissociation of the acrylate compound within the grouting agent (< 10% w/w) during the grouting works. Since the producer confirmed that the used acrylate dissolves only slightly to acrylic acid at the given conditions, within the framework of this exposure assessment, it is assumed that this scenario is of minor relevance.

4.1.1.2.1 Occupational exposure during production and further processing in the large-scale chemical industry

Production and further processing as a chemical intermediate

Acrylic acid is primarily used as a chemical intermediate which is further processed to acrylic esters, homopolymers and copolymers. Production and further processing are performed in closed systems which may be breached during sampling, transfer, filling, cleaning, maintenance, and repair works. According to information provided by a manufacturer acrylic acid is distributed in railway tank wagons or trucks and is transferred into storage tanks and reactors via closed pipes (gas displacement device).

Products manufactured from acrylic acid (e.g. sizing preparations, water treatment materials) contain less than 900 ppm acrylic acid monomer. For adhesives it is known that the monomer is present at concentrations of 1-10%. Further information of monomer content in other products e.g. paints is not available. By comparison with the concentration of monomeric methacrylic acid in paints (up to 700 ppm) it may be derived, that the content of acrylic acid lies in the same range.

Pure acrylic acid and preparations containing ≥ 5% AA are labelled as corrosive.

Workplace measurements

Results of workplace measurements provided by the manufacturers are presented in the following table, and are classified according to 8-hour TWA and short-term measurements.
Table 4.1  Acrylic acid exposures at workplaces during production and further processing

<table>
<thead>
<tr>
<th>Job category / activities</th>
<th>Year of measurement</th>
<th>Number of samples</th>
<th>Range of measurement data [mg/m³]</th>
<th>Geometric mean [mg/m³]</th>
<th>90th-percentile [mg/m³]</th>
<th>Duration and frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>8-h TWA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Manufacture (not specified in detail)</td>
<td>1987 – 1997</td>
<td>83</td>
<td>0.06 - 7.8 ¹)</td>
<td>0.51</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Manufacture, distillation</td>
<td>1993</td>
<td>3</td>
<td>0.33 - 0.67 ²)</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Manufacture, polymerisation, charging, piping into vessels</td>
<td>1994 – 1996</td>
<td>4/16</td>
<td>0.13 - 2.59/0.01 - 0.46</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Manufacture / further processing</td>
<td>1990 – 1996</td>
<td>245</td>
<td>&lt; 15</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Esterification (not specified in detail)</td>
<td>1986 – 1994</td>
<td>37</td>
<td>0.03 - 6.0</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Polymerisation (not specified in detail)</td>
<td>1986 – 1994</td>
<td>18</td>
<td>0.03 - 6.0</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Filling</td>
<td>1986 – 1994</td>
<td>10</td>
<td>0.03 - 6.0</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Pilot plant / laboratory (not specified in detail)</td>
<td>1986 – 1994</td>
<td>36</td>
<td>0.03 - 6.0</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Short-term values</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Manufacture</td>
<td>1987 – 1997</td>
<td>36</td>
<td>0.06 - 29.4 ¹)</td>
<td>0.7</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Filling drums</td>
<td>1987 – 1997</td>
<td>12</td>
<td>0.078 - 34.8 ¹)</td>
<td>5.5</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Loading/unloading tank trucks / railway tank wagons</td>
<td>1987 – 1997</td>
<td>84</td>
<td>0.03 - 187.2 ¹)</td>
<td>1.38</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Charging acrylic acid</td>
<td>1987 – 1997</td>
<td>121</td>
<td>0.3 - 44.4 ¹)</td>
<td>1.65</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Laboratory, development work, quality control</td>
<td>1987 – 1997</td>
<td>51</td>
<td>0.06 - 11.43 ¹)</td>
<td>0.63</td>
<td>---</td>
<td>---</td>
</tr>
</tbody>
</table>

¹) analytical method using HPLC or GC not described in detail
²) derivatisation to ethyl ester, detection with GC-FID

The applied analytical method comprises HPLC with UV detection after adsorption of the substance to a special scavenger (XAD-8) and desorbing with a methanol/water mixture (OSHA No. 28). The detection limit of the method is 0.042 mg/m³ (0.002 ml/m³). Two manufacturers used different analytical methods (see Table 4.1).

The measurement results are regarded as valid, although the results are based on workplaces and activities which, in part, are described in general terms only. In addition, further detailed information on the duration and frequency of exposure as well as on the collective of the exposed workers is missing.

The shift average values (8-h TWA) are measured between 0.03 and < 15 mg/m³ (0.01-5 ml/m³) with a 90th percentile at 3 mg/m³ (1 ml/m³) for different tasks, short-term values between 0.03-187.2 mg/m³ (0.01-62.4 ml/m³) without calculation of the 90th percentile. Taken into account the geometric mean values between 0.63-5.5 mg/m³ (0.2-1.8 ml/m³), the maximum of 187.2 mg/m³ (62.4 ml/m³) is assumed as an outlier.
Because on the basis of the available data it is not possible to calculate the 90\textsuperscript{th} percentile (reasonable worst case), the next available result of 44.4 mg/m\textsuperscript{3} (14.8 ml/m\textsuperscript{3}) is taken as representing a 90\textsuperscript{th} percentile.

Manufacture of adhesives

Acrylic acid is used as an additive for the production of one- and two-package (anaerobic and radiation-hardened) polymerisation adhesives. In the manufacture of these special-purpose adhesives for high-quality bonding of metal, acrylic acid monomer is added (concentration: 1-10\%) to improve adhesion. Adhesives are manufactured either quasi continuously or batchwise in both closed and partially open systems (lidded mixer). For the production of solvent-based adhesives it is known, that only high volume preparations are produced quasi continuously. Therefore batchwise production is to be assumed within the chemical industry, whereby partially open systems operate in conjunction with local ventilation equipment.

Within the chemical industry exposure is possible during sampling and analysis, filling and drumming, as well as during cleaning, maintenance and repair work. Because neither workplace measurements nor detailed information on the duration and frequency of exposure are available, exposure assessment is performed applying the EASE model (see Section 4.1.1.2.4) assuming daily exposure for two hours (manufacturing of formulations).

Dermal exposure during manufacture and further processing in the large-scale chemical industry

On account of the corrosive effect of pure acrylic acid as well as of preparations (labelled as corrosive at a content of \(\geq 5\%\)) and taking into consideration the highly accepted use of suitable protective equipment within the large-scale chemical industry, it can be assumed that, as a rule, daily repeated skin contact is avoided to a large extent by using suitable personal protective equipment (gloves and eye protection). During activities like drumming, filling, cleaning and maintenance potential exposure is assumed only by single contacts. The corresponding exposure level is assessed by the EASE model.

Daily repeated dermal exposure is assessed to be low.

4.1.1.2.2 Occupational exposure in the further processing industries, outside the chemical industry

Manufacture of adhesives

Further processing of acrylic acid to special-purpose adhesives may not be limited to the large-scale chemical industry but occurs in the industrial area, too, as well as in small and medium-sized chemical companies. Batchwise production is also assumed (see Section 4.1.1.2.1).

In these areas it cannot be excluded, that the substance is handled in open systems during certain tasks, e.g. metering and filling activities, and that suitable technical measures (LEV, local exhaust ventilation) are not used (Voullaire and Kliemt, 1995).

Inhalation exposure is possible during sampling and analysis, filling and drumming, as well as during cleaning, maintenance, and repair work. Because neither workplace measurements nor detailed information on the duration and frequency of exposure is available, exposure assessment is performed applying the EASE model (see Section 4.1.1.2.4) assuming daily exposure for two hours (manufacturing of formulations).
The use of personal protective equipment (gloves and eye protection) is assumed during certain tasks (e.g. filling, drumming) considering the corrosive effect of the pure acrylic acid and the produced preparations (content of AA ≥ 5%). Furthermore at a site at which corrosive products are handled it is assumed, that the workers will protect themselves even if they handle preparations with irritant effects. The daily dermal exposure for these scenarios is assessed to be low. During activities like filling, transfer, cleaning, maintenance and repair work, potential exposure is assumed only by single contacts.

**Use of adhesives in the further processing industry**

In the field of engineering, device and tool construction industries, one- and two-package adhesives with up to 10% acrylic acid are used to bond metals either anaerobically or radiation-hardened during assembly. Automatic or semi-automatic bonding machines are employed within continuous production processes (production lines). It is assumed that low amounts of the adhesives are applied to bond small areas. If radiation-hardened adhesives are used the bonded workpiece is hardened by UV light within closed systems. The components, which are still warm, are in some cases stored in open systems.

Inhalation and dermal exposure is possible during charging and bonding work (semi-automatic machines), during cleaning, maintenance and repair work, and during work in the vicinity of openly-stored components which are still warm. It is to be assumed that not every production plant is equipped with suitable technical ventilation equipment and it cannot be excluded that PPE (gloves) is not worn (Voullaire and Kliemt, 1995), if non-corrosive adhesives are handled.

On account of the corrosive effect of adhesives (content of AA ≥ 5%), it can be assumed, as a rule, that daily repeated immediate skin contact is avoided to a large extent by using suitable personal protective equipment (gloves and eye protection). In this case daily dermal exposure is assessed to be low. During activities like filling, cleaning and maintenance potential exposure is assumed only by single contacts. The corresponding exposure level is assessed applying the EASE model (see Section 4.1.1.2.4).

In the case of handling adhesives not labelled as corrosive, frequent immediate skin contact has to be taken into consideration. Generally workers avoid immediate skin contact with adhesives that can be removed only with difficulties (Kliemt, 1995). The corresponding adhesives could be removed more easily because they harden only slowly, and thus have the opportunity to penetrate the skin. These adhesives are removed later with the aid of skin cleaning agents which are also employed after contact with paints. The corresponding exposure level is assessed applying the EASE model (see Section 4.1.1.2.4).

Neither workplace measurements nor information on the duration and frequency of exposure or on the collective of the exposed group are available.

**Decomposition of photoresist materials during the production of integrated circuits**

Acrylic acid may be released as a decomposition product during the production of integrated circuits or in the galvanic industry, when photoresist materials (e.g. methyl methacrylate) are partially depolymerized by means of UV light. The subsequent development with dissolving the depolymerized material is performed in closed systems (Reichert, 1993), so that it is to be assumed that inhalation exposure to acrylic acid can be neglected. No information could be obtained whether the UV light treatment occurs in closed systems or if the workplaces are equipped with local exhaust ventilation. However, it is assumed that during UV light treatment
only a part of the formed acrylic acid is evaporated, and, unless other information is provided, that inhalation exposure is low.

Because acrylic acid is released during thermal processes, normally no immediate skin contact occurs. The dermal exposure level caused by touching of acrylic acid contaminated surfaces (indirect exposure) is regarded as being low.

4.1.1.2.3 Occupational exposure in the skilled trade sector

Use of adhesives

Adhesives which contain acrylic acid are used for metal repair e.g. workpieces. During repair works, which may involve rather small areas, workers may be subjected to inhalation and dermal exposure. It may be assumed that exhaust ventilation systems are absent, and that suitable personal protective equipment is not worn if adhesives not labelled as corrosive are handled. For further description of dermal exposure during the handling of corrosive adhesives and adhesives not labelled as corrosive see Section 4.1.1.2.2, “use of adhesives in the further processing industry”.

Neither workplace measurements nor information on the duration and frequency of exposure or on the collective of the exposed group are available.

It is to be assumed, that the adhesives are not handled daily and that the duration is much shorter than the shift length.

Gas flame removal of paints

Acrylic acid may be released as a thermal decomposition product when paints are removed from steel or concrete surfaces by means of gas flames.

Occupational exposure measurements (n=6) during the removal of old paints from windows with gas flame or with a hot-air blower under realistic workplace conditions show no release of acrylic acid above the detection limits of 0.01-0.06 ppm (Työterveyslaitos, written communication from 08.12.1998).

In laboratory experiments acrylic acid was detected as a decomposition product during gas flame removal of 8 out of 10 steel protective paints, used in the Finnish shipyards (Henricks-Eckerman et al., 1990). The emissions of acrylic acid were determined between 0.2-1 mg/m$^3$ (n = 8).

The air concentration of free acrylic acid during normal working processes involving welding, flame cutting or straightening of painted steel sheets is estimated to be approximately three orders of magnitude lower (Työterveyslaitos, written communication from 08.12.1998).

Because acrylic acid is released during thermal processes, normally no immediate skin contact occurs. According to a rough estimation of dermal exposure caused by touching acrylic acid contaminated surfaces (indirect exposure), the exposure level is regarded as being low.
4.1.1.2.4 Estimation of the exposure according to the EASE model

The estimation of the level of inhalation and dermal exposure performed on accordance with the EASE model (TGD) produces the following results:

Inhalation exposure

a) Exposure to acrylic acid vapour during the manufacture and further processing of acrylic acid in the large-scale chemical industry:

Input parameters: T = 20°C
closed system
significant breaching
LEV present

Estimated exposure: 1.5-9 mg/m³ (0.5-3 ml/m³)

b) Use of adhesives containing acrylic acid, with local exhaust ventilation:

Input parameters: T = 20°C
non dispersive use
direct handling
LEV present

Estimated exposure: 1.5-9 mg/m³ (0.5-3 ml/m³)

c) Exposure to acrylic acid vapour when adhesives on the basis of acrylic acid are produced or adhesives containing acrylic acid are used, without suitable local exhaust ventilation:

Input parameters: T = 20°C
non dispersive use
direct handling
dilution ventilation

Estimated exposure: 30-150 mg/m³ (10-50 ml/m³)

The partial vapour pressure of acrylic acid cannot be considered, because the composition of the adhesives is not known. Therefore it is assumed for the above estimation that the partial vapour pressure is in the same EASE category as the vapour pressure of pure acrylic acid.

The EASE model cannot be applied to estimate the exposure to acrylic acid as a decomposition product (e.g. gas flame removal of paints).

Dermal exposure

a) Potential dermal exposure during production and further processing of acrylic acid in the chemical industry and in the industrial sector as well as during the use of adhesives (labelled as corrosive at a content of ≥ 5%):

Input parameters: T = 20°C
non dispersive use
direct handling
incidental

Estimated exposure: 0-0.1 mg/cm²/day
Use of adhesives: Considering a content of 10% AA in adhesives, dermal exposure amounts to 0-0.01 mg/cm²/day

b) Dermal exposure via immediate contact with adhesives (not labelled as corrosive) which are used:

| Input parameters: | T = 20°C                      |
|                  | non dispersive use            |
|                  | direct handling              |
|                  | intermittent                 |

Estimated exposure: 0.1-1 mg/cm²/day

Use of adhesives: Considering a content of 5% acrylic acid in adhesives, dermal exposure amounts to: 0.005-0.05 mg/cm²/day

4.1.1.2.5 Integrated Assessment

General

Acrylic acid is primarily used as a chemical intermediate which is further processed to acrylic esters, homopolymers and copolymers. For some of the processed products e.g. water treatment materials and sizing preparations manufactured from acrylic acid the content of the residual monomer is known (less than 900 ppm). Further information on the monomer content in other products e.g. paints is not available. By comparison with the concentration of monomeric methacrylic acid in paints (up to 700 ppm) it may be concluded, that the content of acrylic acid lies in the same range. Anaerobic and radiation-hardened polymerisation adhesives may contain up to 10% acrylic acid.

Production and further processing in the large-scale chemical industry

Production and further processing as a chemical intermediate

The submitted measurement results are regarded as valid although the results are based on workplaces and activities which, in part, are described in general terms only. In addition further detailed information on the duration and frequency of exposure as well as on the collective of the exposed group is missing. The undifferentiated presentation of the measurement results makes it impossible to state exposure levels typical for particular workplaces.

The measurements in the range of 0.03-7.8 mg/m³ (0.01-3.9 ml/m³) agree well with the inhalation exposure level of 1.5-9 mg/m³ (0.5-3 ml/m³) which is estimated in application of the EASE model. For the assessment of the risks of inhalation exposure to acrylic acid during manufacture and further processing in the chemical industry, the 90th percentile of one measurement collective of 3 mg/m³ (1 ml/m³) is to be assumed, whereby the short-term exposure may be even higher during certain activities: concentrations as high as 187.2 mg/m³ (62.4 ml/m³) have been measured when tankers or railway tank wagons are loaded and unloaded. For the assessment of the risk 44.4 mg/m³ (14.8 ml/m³) should be used as a reasonable worst case (see Section 4.1.1.2.1).
Manufacture of special-purpose adhesives in the large-scale chemical industry

Within the chemical industry inhalation exposure is possible during sampling and analysis, filling and drumming, as well as during cleaning, maintenance, and repair work. Because neither workplace measurements nor detailed information on the duration and frequency of exposure are available, exposure assessment is performed applying the EASE model (see Section 4.1.1.2.4) assuming daily exposure for two hours (manufacturing of formulations). For the assessment of the risks of inhalation exposure to acrylic acid during manufacture of adhesives exposures of 0.375-2.25 mg/m$^3$ (0.125-0.75 ml/m$^3$, with LEV, 2 h) estimated according to the EASE model are to be assumed.

Dermal exposure

On account of the corrosivity of pure acrylic acid and adhesives (labelled as corrosive at a content of $\geq 5\%$ AA) immediate skin contact is only assumed by single contacts, because, in general, suitable personal protection equipment (gloves and eye protection) are worn to avoid the contact to a large extent. The estimation of a potential exposure by single contacts according to the EASE model is about 0-0.1 mg/cm$^2$/day; on account of an exposed skin area of about 420 cm$^2$ an exposure level of 0-42 mg/person/day would result. Taken into consideration that the use of gloves has a high acceptance within the chemical industry, daily dermal exposure is assessed to be low, even if non-corrosive adhesives are handled.

Occupational exposure in the further processing industry, outside the large-scale chemical industry

Manufacture of special-purpose adhesives

Further processing of acrylic acid to one- and two-package polymerisation adhesives may not be limited to the large-scale chemical industry but occurs in the industrial area, too, as well as in small and medium-sized chemical companies. In these areas it cannot be excluded, that acrylic acid is handled also in open systems during certain tasks, e.g. metering and filling activities, and that no suitable technical measures (LEV, local exhaust ventilation) are used, when preparations not labelled as corrosive are handled.

Since no workplace measurements are available, the EASE model is applied to estimate inhalation exposure. In the case of workplaces provided with suitable local exhaust ventilation systems, the inhalation exposure is calculated to 1.5-9 mg/m$^3$ (0.5-3 ml/m$^3$). If it is assumed that no local exhaust ventilation system is present, the level of inhalation exposure amounts to 30-150 mg/m$^3$ (10-50 ml/m$^3$). Taking into consideration a daily duration of exposure of 2 hours, exposures of 0.375-2.25 mg/m$^3$ (0.125-0.75 ml/m$^3$, with LEV) or 7.5-37.5 mg/m$^3$ (2.5-12.5 ml/m$^3$, without LEV) result.

The use of personal protective equipment (gloves and eye protection) is assumed during certain tasks (e.g. filling, drumming) considering the corrosive effect of the pure acrylic acid and the produced preparations (content of AA $\geq 5\%$). Furthermore for a site at which corrosive products are handled it is assumed, that the workers will protect themselves even if they handle preparations with irritant effects. The daily dermal exposure for these scenarios is assessed to be low. During activities like filling, transfer, cleaning, maintenance, and repair work, potential exposure is assumed only by single contacts. Considering the exposure level of 0-0.1 mg/cm$^2$/day assessed using the EASE model and an exposed area of 420 cm$^2$, the exposure amounts to 0-42 mg/person/day.
CHAPTER 4. HUMAN HEALTH

Use of special-purpose adhesives in the further processing industries

It is to be assumed that in the industrial sector, adhesives (containing 1-10% acrylic acid) are sometimes handled in open systems during certain activities such as dosage, filling and bonding. Further, if radiation-hardened adhesives are used, acrylic acid can partially evaporate after the (UV) hardening process if the warm workpiece is stored openly. Estimation in application of the EASE model (measurements are not available) produces a potential exposure of 1.5 – 9 mg/m³ (0.5-3 ml/m³) for workplaces with local exhaust ventilation and 30-150 mg/m³ (10–50 ml/m³) for workplaces without local exhaust ventilation. For workplaces without local exhaust ventilation, the lower value of the estimated concentration range (30 mg/m³) appears more realistic in view of the method of use (bonding small areas).

In the case of handling adhesives (labelled as corrosive at ≥ 5%) potential dermal exposure is assumed only by single contacts. The exposure level estimated using the EASE model amounts to 0.-0.01 mg/cm²/day. Generally, small areas of the body are affected. Assuming that an area of 210 cm² (fingers) is exposed a level of 0-2.1 mg/person/day is obtained. Taking into account the corrosive effect of adhesives, daily repeated skin contact is avoided to a large extent by using suitable personal protective equipment, so that daily dermal exposure is assessed to be low.

When preparations not labelled as corrosive (containing < 5% acrylic acid) are handled, it cannot be excluded that gloves are not worn (see Section 4.1.1.2.2). In this case daily dermal exposure is assessed applying the EASE model to 0.005-0.05 mg/cm²/day. Considering an exposed area of 210 cm², dermal exposure amounts to 1-10.5 mg/person/day.

Decomposition of photoresist materials during the production of integrated circuits

Acrylic acid may be released as a decomposition product during the production of integrated circuits or in the galvanic industry, when photoresist materials (e.g. methyl methacrylate) are partially depolymerized by means of UV light. No information could be obtained whether the UV light treatment occurs in closed systems or if the workplaces are equipped with local exhaust ventilation. However, it is assumed that during the UV light treatment only a part of the formed acrylic acid is evaporated, and, unless other information is provided, that inhalation exposure is low.

Because acrylic acid is released during thermal processes, normally no immediate skin contact occurs. The dermal exposure level caused by touching of acrylic acid contaminated surfaces (indirect exposure) is regarded as being low on account of the inhalation exposure levels.

Occupational exposure in the skilled trade

Use of special-purpose adhesives in the skilled trade

Adhesives which contain acrylic acid are used for repair e.g. metal workpieces. It is assumed that rather small areas are bonded and that exhaust ventilation systems are absent, and that suitable personal protective equipment is not worn if non-corrosive adhesives containing < 5% acrylic acid are handled. For the use of adhesives in the skilled trade sector, it has to be taken into account that the overall duration of open handling of adhesives is probably much shorter than the shift duration. Therefore the inhalation exposure level is assumed to be lower than in the comparable industrial sector (without LEV, 30 mg/m³).

The dermal exposure levels may be in the same order of magnitude or even lower than assessed for the use of corrosive and non-corrosive adhesives in the industrial sector (corrosive adhesives:
low, potential exposure: 0-2.1 mg/person/day; non-corrosive: 1-10.5 mg/person/day). It is to be assumed that these repair activities will not be done daily. Since neither workplace measurements nor information on the duration and frequency of exposure are available, no further statements can be made.

**Gas flame removal of acrylic paint**

Acrylic acid may be released as a thermal decomposition product when paints are removed from steel or concrete surfaces by means of gas flames (Henricks-Eckerman et al., 1990). The workplace measurements show that the inhalation exposure level under realistic workplace conditions are below the detection limits of 0.01-0.06 ppm (Työterveyslaitos, 1998). Because of the low not detectable exposure level it is assumed that this exposure scenario is only of minor relevance for the assessment of the occupational exposure.

### 4.1.1.2.6 Summary of exposure data relevant for workplace risk assessment

The following table shows the exposure data of acrylic acid which are relevant for occupational risk assessment.
Table 4.2  Summary of exposure data of acrylic acid which are relevant for occupational risk assessment

<table>
<thead>
<tr>
<th>Area of production and use</th>
<th>Form of exposure Activity</th>
<th>Duration and frequency</th>
<th>Inhalation exposure</th>
<th>Dermal exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Exposure level shift average [mg/m³]</td>
<td>Method</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Method</td>
<td></td>
</tr>
<tr>
<td><strong>Chemical industry</strong></td>
<td></td>
<td></td>
<td>3.0</td>
<td>90th percentile assumed reasonable worst case</td>
</tr>
<tr>
<td>Production, further processing</td>
<td>vapour / liquid</td>
<td>filling, transfer, cleaning, maintenance, repair work</td>
<td>shift length, daily short term, daily single contacts</td>
<td>44.4</td>
</tr>
<tr>
<td>Manufacture of adhesives (1 - 10% acrylic acid)</td>
<td>vapour / liquid</td>
<td>cleaning, maintenance, repair work, drumming</td>
<td>assumed 2 h/ daily single contacts</td>
<td>0.375 - 2.25 EASE with LEV</td>
</tr>
<tr>
<td><strong>Industrial area</strong></td>
<td></td>
<td></td>
<td>0.375 - 2.25</td>
<td>7.5 -37.5</td>
</tr>
</tbody>
</table>

Table 4.2 continued overleaf
Table 4.2 continued Summary of exposure data of acrylic acid which are relevant for occupational risk assessment

<table>
<thead>
<tr>
<th>Area of production and use</th>
<th>Form of exposure Activity</th>
<th>Duration and frequency</th>
<th>Inhalation exposure</th>
<th>Dermal exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>shift average</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>[mg/m$^3$]</td>
<td>[mg/cm$^2$/day]</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Method</td>
<td>Exposed area</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>[cm$^2$]</td>
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<td></td>
<td></td>
<td></td>
<td>Shift average</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>[mg/p/day]</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Method</td>
</tr>
<tr>
<td>Use of adhesives:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- ≥ 5% acrylic acid</td>
<td>vapour / liquid</td>
<td>shift length, daily</td>
<td>1.5 - 9</td>
<td>low</td>
</tr>
<tr>
<td>(labelled as corrosive)</td>
<td></td>
<td>single contacts</td>
<td>30 $^1$</td>
<td>---</td>
</tr>
<tr>
<td></td>
<td>handling, gluing,</td>
<td></td>
<td>---</td>
<td>0 - 0.01</td>
</tr>
<tr>
<td></td>
<td>charging</td>
<td></td>
<td>EASE with LEV</td>
<td>210 (fingers)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>without LEV</td>
<td>0 - 2.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>exp. judg.</td>
<td>EASE</td>
</tr>
<tr>
<td>- &lt; 5% acrylic acid</td>
<td>vapour / liquid</td>
<td>intermittent / assumed</td>
<td>1.5 - 9</td>
<td>low</td>
</tr>
<tr>
<td>(not labelled as corrosive)</td>
<td></td>
<td>shift length, daily</td>
<td>30 $^1$</td>
<td>0.005 - 0.05</td>
</tr>
<tr>
<td></td>
<td>handling, gluing,</td>
<td></td>
<td>EASE with LEV</td>
<td>210 (fingers)</td>
</tr>
<tr>
<td></td>
<td>charging</td>
<td></td>
<td>without LEV</td>
<td>1 - 11</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>exp. judg.</td>
<td>EASE</td>
</tr>
<tr>
<td>Decomposition during</td>
<td>vapour</td>
<td>shift length, daily</td>
<td>low $^2$</td>
<td>low</td>
</tr>
<tr>
<td>production of</td>
<td></td>
<td></td>
<td>exp. judg.</td>
<td>---</td>
</tr>
<tr>
<td>integrated circuits</td>
<td></td>
<td></td>
<td></td>
<td>low $^3$</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>exp. judg.</td>
</tr>
<tr>
<td>Skilled trade</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Use of adhesives:</td>
<td>vapour / liquid</td>
<td>handling, gluing</td>
<td>&lt; 30</td>
<td>low</td>
</tr>
<tr>
<td>- ≥ 5% acrylic acid</td>
<td></td>
<td>shorter than shift</td>
<td>exp. judg.</td>
<td>---</td>
</tr>
<tr>
<td>(labelled as corrosive)</td>
<td></td>
<td>length, not daily</td>
<td></td>
<td>0 - 0.01</td>
</tr>
<tr>
<td></td>
<td></td>
<td>single contacts</td>
<td>---</td>
<td>210 (fingers)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>shorter than shift</td>
<td>exp. judg.</td>
<td>0 - 2.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>length, not daily /</td>
<td></td>
<td>EASE</td>
</tr>
<tr>
<td></td>
<td></td>
<td>intermittent</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>vapour / liquid</td>
<td>handling, gluing</td>
<td>&lt; 30</td>
<td>low</td>
</tr>
<tr>
<td>- &lt; 5% acrylic acid</td>
<td></td>
<td>shorter than shift</td>
<td>exp. judg.</td>
<td>---</td>
</tr>
<tr>
<td>(not labelled as corrosive)</td>
<td></td>
<td>length, not daily /</td>
<td></td>
<td>0.005 - 0.05</td>
</tr>
<tr>
<td></td>
<td></td>
<td>intermittent</td>
<td></td>
<td>210 (fingers)</td>
</tr>
</tbody>
</table>

$^1$ Lower level of the estimated range is assumed to be realistic (expert judgement)

$^2$ Acrylic acid is released as a decomposition product, inhalation exposure is assumed to be low

$^3$ Dermal exposure by touching contaminated surfaces is assumed to be low on account of the inhalation exposure levels
4.1.1.3 Consumer exposure

According to the Swedish Product Register, products containing acrylic acid are used as adhesive or glue and in adhesive substances on the basis of solvents. They are offered in car and motorcycle repair shops for public use. Furthermore, acrylic acid in sealing compounds is also used by consumers.

According to data reported by industry to the BgVV for poison information centres, acrylic acid is used in the Federal Republic of Germany as a component of adhesives (content of acrylic acid up to 8.5%) and as a surface sealing material (content of acrylic acid 6%).

Inhalation exposure

Exposure to UV-hardening adhesives

For the assessment of the inhalation exposure of the consumer, a computer simulation with the aid of the US EPA model SCIES was used showing consumer exposure under different conditions (Appendix B1).

Under conditions of proper use of 1 g UV-hardenable adhesive (content of acrylic acid 6%) 4 times per year for 1 hour each, the consumer using the adhesive is exposed to an average of 0.384 mg/m$^3$ with a peak value of 0.542 mg/m$^3$ during the period of use; after use, a peak concentration of 0.448 mg/m$^3$ is calculated.

It is exclusively the monomer that accounts for the content of acrylic acid in the adhesive.

Dermal exposure

Exposure to sanitary towels, pantyliners and nappy pants

There are no data on the contents of polyacrylates and the weights of the above-mentioned products. Concerning these products, babies may be considered as a worst-case scenario with regard to the duration of the contact, the contact surface and the body weight. As to the assessment of the dermal exposure of babies to acrylic acid from the residual content of monomer acrylic acid in homopolymerisates of acrylic acid which are used as “superabsorbents” in nappy pants, the following data have been submitted by the Industrieverband für Körperpflege- und Waschmittel (industrial association for body care products and detergents/EDANA):

Amounts of acrylic acid in the residual dampness of nappy:

- Daytime: 0.36 µg
- Naptime: 0.43 µg
- Nighttime: 1.08 µg

Under the assumption, that 4 nappies will be used during daytime, a total of acrylic acid of 2.95 µg can be calculated. 20% of acrylic acid is available for absorption (=0.59 µg). The body weight of newborn children is taken as 3.16 ± 0.35 kg, and that of a 1-year-old child 9.74 ± 1.07 kg (mean ± SD). Taken this distribution into consideration, the dermal exposure to acrylic acid will be as follows:

\[ \text{Exposure} = \text{Dermal absorption} \times \text{Body weight} \]

\[ \text{Exposure} = 0.59 \times \text{Body weight} \]

\[ \text{Exposure} = 0.59 \times (3.16 \pm 0.35) \]

\[ \text{Exposure} = 0.59 \times (9.74 \pm 1.07) \]

\[ \text{Exposure} = 1.84 \pm 0.36 \]
Table 4.3  Dermal exposure of children to acrylic acid

<table>
<thead>
<tr>
<th></th>
<th>Median</th>
<th>95% Percentile</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborns</td>
<td>0.18</td>
<td>0.22</td>
<td>0.29</td>
</tr>
<tr>
<td>1-year-old children</td>
<td>0.06</td>
<td>0.073</td>
<td>0.102</td>
</tr>
</tbody>
</table>

* The @-RISK-program (Palisade-Corp., New-York) was used for calculations.

Oral exposure

*Exposure to articles coming into contact with food*

Plastic material that comes into contact with food is regulated by the EU directive 90/128/EEC, 28th of February 1990 (Directive of materials and articles intended to come in contact with food stuff). In this regulation, acrylic acid has not been detailed. Exposure data due to limitations given by the directive are therefore not available.

In comparison with other plastic material (e.g. MMA) the amounts of acrylic acid should be low and therefore may be neglected.

As compared to this, the t-TDI value (temporary tolerable daily intake) is determined as 0.1 mg/kg bw by the Wissenschaftlicher Lebensmittelausschuß (Scientific Committee on Food) of the EU.

Remark

For the assessment of consumer exposure, preferably standard assumptions should be used applicable to typical cases of use; in situations where this is not possible, assumptions will rather be arbitrary. The amounts used per application are based on the data given by the manufacturer. As a rule, an adult of 60 kg bw will be considered as a standard consumer.

On account of the variability of the exposure conditions, individual exposure cannot be defined exactly. Therefore, it would be more appropriate to indicate fields of possible exposure. These have been divided by log ranges (lower range: 1-10, middle range 10-100, upper range 100-1,000).

4.1.1.4  Humans exposed via the environment

According to Appendix VII of Chapter 2 of the TGD the indirect exposure to humans via the environment, i.e. through food, drinking water and air is estimated. As a worst-case scenario, the maximum intake due to exposure in the vicinity of a point source (generic model) is calculated. This is compared to an average intake due to exposure via the regional background concentration. In Appendix A11, the detailed calculations are presented.
The following input parameters were used:

<table>
<thead>
<tr>
<th>Route</th>
<th>Local scenario</th>
<th>Regional scenario</th>
</tr>
</thead>
<tbody>
<tr>
<td>Concentration in grassland soil</td>
<td>4.69 µg/kg (_{ww})</td>
<td>0.02 µg/kg (_{ww})</td>
</tr>
<tr>
<td>Concentration in agricultural soil</td>
<td>24.2 µg/kg (_{ww})</td>
<td>0.02 µg/kg (_{ww})</td>
</tr>
<tr>
<td>Concentration in surface water:</td>
<td>222 µg/l(^*)</td>
<td>0.40 µg/l</td>
</tr>
<tr>
<td>Concentration in the atmosphere:</td>
<td>28 µg/m(^3)</td>
<td>0.002 µg/m(^3)</td>
</tr>
<tr>
<td>Concentration in groundwater:</td>
<td>2.42 µg/l</td>
<td>0.02 µg/l</td>
</tr>
</tbody>
</table>

\(^*\) The annual average local aquatic concentration of 222 µg/l calculated for a generic production and processing site is chosen as a realistic worst case, because the emission to atmosphere and soil were estimated on the basis of the same scenario. The higher result, i.e. 1.74 mg/l for external wet polymerisation was not used to avoid unreasonable combination of worst-case emissions. Although the concentration in surface water mentioned above is 8 times higher than the chosen one, the overall scenario is still regarded as a worst case.

The resulting total daily dose is:

\[ \text{DOSE}_{\text{tot}} = 50 \mu g \cdot \text{kg}_{bw}^{-1} \cdot \text{d}^{-1} \text{ (local scenario)} \]

\[ \text{DOSE}_{\text{tot}} = 15.1 \text{ ng} \cdot \text{kg}_{bw}^{-1} \cdot \text{d}^{-1} \text{ (regional scenario)} \]

The calculated total doses comprise the following routes:

<table>
<thead>
<tr>
<th>Route</th>
<th>% of total dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drinking water</td>
<td>11.8</td>
</tr>
<tr>
<td>Fish</td>
<td>0.3</td>
</tr>
<tr>
<td>Stem</td>
<td>76.6</td>
</tr>
<tr>
<td>Root</td>
<td>&lt; 0.1</td>
</tr>
<tr>
<td>Meat</td>
<td>&lt; 0.1</td>
</tr>
<tr>
<td>Milk</td>
<td>&lt; 0.1</td>
</tr>
<tr>
<td>Air</td>
<td>11.2</td>
</tr>
</tbody>
</table>

The main route of indirect exposure is the intake via plant stems for the local and via drinking water for the regional scenario.
4.1.2 Effects assessment: Hazard identification and Dose (concentration) -
response (effect) assessment

4.1.2.1 Toxicokinetics, metabolism and distribution

Toxicokinetics

Following gavage of an aqueous solution of $[^{14}\text{C}]-\text{acrylic acid}$ (26 µg/kg bw) to rats, acrylic acid was rapidly (within 1 h) absorbed and excreted, mainly as $^{14}\text{CO}_2$. Stomach intubated rats retained 37.0% of the administered radioactivity at 65 min, whereas approximately 60% of the radioactivity had been expired as $^{14}\text{CO}_2$. Relative retention of the radioactivity after 65 min was ≥ 1% in the liver (2.6%), adipose tissue (1.9%), small intestine (1.5%), kidneys (1.2%), and spleen (1.0%) and was less than <1% in lungs, skin, blood, heart, brain and muscle. Approximately 6% of the administered radioactivity was excreted in the urine (Kutzman et al., 1982).

Rats received single oral doses of $[2,3-^{14}\text{C}]-\text{acrylic acid}$ (4, 40 or 400 mg/kg bw in a 0.5% aqueous methylcellulose solution). Within 8 hours, 35-60% of the dose was eliminated from the animal, mostly as expired CO$_2$. After 72 hours, 44-65% of the radioactivity had been eliminated via expired air, while 2.9-4.3% remained in urine, 2.4-3.6% in faeces and 18.9-24.6% in tissues examined (adipose tissue 9-15%, liver 1.7-2.2% and blood 0.8-1.1%) (De Bethizy et al., 1987).

Following single oral administration of 400 mg $[2,3-^{14}\text{C}]-\text{acrylic acid}$/kg bw to rats within 3 days 78% of the radiolabel was exhaled as $^{14}\text{CO}_2$, while 6.3% was excreted in urine, 1.1% in faeces and 12.8% remained in tissues (4.8% in muscle, 3% in liver, 1.3% in fat, 2% in skin). This excretion pattern was consistent with that of $[^{1}\text{C}]-\text{propionate}$ administered in the same manner (Winter et al., 1992).

Following gavage of an aqueous solution of 400 mg $[^{14}\text{C}]-\text{acrylic acid}$ to rats, acrylic acid was well absorbed and excreted primarily (approximately 80%) as $^{14}\text{CO}_2$ within 24 h of administration. Exhalation of volatile compounds was negligible (<0.5% of the dose). Excretion in urine accounted for 5%, excretion in faeces was 9% within 3 days. Tissue concentrations of acrylic acid derived radioactivity were generally low after 3 days, in liver 0.4% of the dose, in muscle 0.4, in skin 0.2, in other tissues below 0.1% (Winter and Sipes, 1993).

Mice and rats, respectively, were treated orally (40 or 150 mg/kg bw) or dermally (10 or 40 mg/kg bw in acetone) with $[^{1}\text{C}]-\text{acrylic acid}$. Mice rapidly absorbed and metabolised orally administered acrylic acid, with about 80% of the dose exhaled as $^{14}\text{CO}_2$ within 24 h. Excretion in urine and faeces accounted for approximately 3% and 1% of the dose, respectively. Elimination of the $^{14}\text{C}$ radiolabel from plasma, liver and kidney was rapid but slower from fat. The disposition of orally administered acrylic acid in rats was similar to the results obtained from mice. After cutaneous application to mice, about 12% of the dose was absorbed, while the remainder was apparently evaporated. Approximately 80% of the absorbed fraction of the dose was metabolised to $^{14}\text{CO}_2$ within 24 h. Excretion in urine and faeces each accounted for less than 0.5% of the dose. Elimination of radioactivity from plasma, liver, and kidney was rapid; however, levels in fat were higher at 72 h (0.5% of the higher dose) than at 8 h (0.1% of the higher dose). After cutaneous administration to rats, 19-26% of the dose was absorbed. Disposition of the absorbed fraction of the dose was similar to results found in mice (Black et al., 1995).

Rats were treated dermally with $[^{14}\text{C}]-\text{labeled acrylic acid}$ (5 mg/kg bw) in either phosphate buffer (pH 6 or 7.4) or acetone for 24 h. The dosing solution (0.1 ml) was spread evenly on the
clipped skin within covered Plexiglas cells (7.3 cm\(^2\) surface area). The rate of appearance of \(^{14}\)CO\(_2\) in exhaled breath was used as a measure of the rate of absorption. The absorption rate was dependent on the vehicle and decreased in the order acetone > buffer pH 6 > buffer pH 7.4. Cumulative absorption after 24 h was 22% from acetone, approximately 19% from buffer pH 6, and 9% from buffer pH 7.4 (D'Souza and Francis, 1988).

Percutaneous absorption of acrylic acid was studied following topical administration of 100 µl of a 4% (v/v) solution of [1-\(^{14}\)C]-acrylic acid in acetone using a skin-mounted, charcoal-containing trap covered with fixed aluminum discs to ensure complete recovery of the radiolabel. Excretion of acrylic acid-derived radioactivity was determined in urine, faeces and expired air over a period of 3 days. After 3 days 73% of the radioactivity had volatilized from the skin, 6% was detected in the skin. 16% of the applied dose, representing 75% of the absorbed dose, was exhaled as \(^{14}\)CO\(_2\) within 12 h. Only 0.9% of the applied radioactivity was found in urine, 0.2% in faeces, and 0.4% was in the major tissues after 72 h (Winter and Sipes, 1993).

Rats were nose-exposed to gaseous \([^{11}\)C]-acrylic acid for 1 minute. At 1.5 minutes 18.3% of the delivered dose was retained in the rats. Relatively large amounts of radioactivity were found in the upper respiratory tract. After 65 minutes the radioactivity in the snout was reduced to 8.1% and approximately 60% of the radiolabel had been expired as \(^{11}\)CO\(_2\). The elimination of \(^{11}\)CO\(_2\) was biphasic with \(t_{1/2}\) of the \(\alpha\) phase of 30.6 min. The amount of radioactivity retained in liver, fat and stomach increased markedly between 1.5 and 65 minutes post exposure. The authors postulate that a portion of acrylic acid was ingested after inhalation. Urinary and fecal excretion was estimated to be 15% within 65 minutes (Kutzman et al., 1982).

A hybrid computational fluid dynamics (CFD) and physiologically-based pharmacokinetic (PBPK) dosimetry inhalation model was constructed to estimate the regional tissue dose of acrylic acid in the rat and human nasal cavity, respectively (Frederick et al., 1998). The rodent model uses two olfactory compartments to incorporate both the olfactory epithelium in the projection extending along the dorsal meatus and the ethmoid olfactory region. This model was based on a compartmental rat nasal model of Bush et al., 1998. The human model uses one olfactory compartment since the human nasal cavity lacks a counterpart for the rodent ethmoid olfactory region (Subramaniam et al., 1998). The liquid phase of the model of Bush et al. was modified to include the effect of buffering capacity on the ionization of the acid in the mucus, diffusion of both the ionized form of the acid and the non-ionized species, liquid:air partition coefficients, tissue: blood partition coefficients (Black and Finch, 1995), and metabolism of acrylic acid (Black and Finch, 1995).

CFD simulations provided estimates of the volume of air flowing through various regions of the rat and human nasal cavities, respectively, at inhalation flow rates respective of resting to light activity physiological conditions (rat: 100-500 ml/min, human: 11,400 or 18,900 ml/min, laminar flows). The simulated regional gas phase mass transport coefficients for the rat nasal cavity are 1-2 orders of magnitude higher than those of the human nasal cavity.

A hybrid CFD-PBPK inhalation model was constructed with the aim to evaluate the relationship between inhaled acrylic acid vapour concentration and the tissue concentration in various regions of the nasal cavity of rats and humans, respectively. An explicit effort was made to derive the parameters for rat and human used in the model either from experimental data or from physicochemical principles without “fitting” model parameters (gas phase diffusivity: 0.1 cm\(^2\)/sec; air minute volumes: 250 ml/min (rat), 7,500 ml/min (human); blood flow to nasal cavity (human) estimated). The results of the quantitative sensitivity analysis of the model parameters are not available completely. Deposition of vapours in the rat nasal cavity is relatively insensitive to significant variation in the gas phase mass transport coefficients, but the
human CFD-PBPK model was sensitive to variation in air phase and liquid phase parameters (liquid diffusivity, mucus:air partition coefficient). The diffusivity of acrylic acid (ionized and non-ionized) in mucus and epithelium was defined as 0.01 cm$^2$/h as an adjustable parameter. The mucus:air partition coefficient was defined as 1,780 (saline, pH 2.0; the liquid:air partition coefficient value for saline, pH 7.4, is 3,210).

Unidirectional simulations were conducted with the model at a flow rate of 500 ml/min (rat) to estimate the steady-state tissue concentration in the anterior olfactory epithelium lining the dorsal meatus of the rat nasal cavity over a wide range of acrylic acid vapour concentrations (0 to 25 ppm for one hour). A dose-response of acrylic acid exposures was simulated for an adult resting male rat and an adult resting male human using the appropriate inspiratory flow rate (based on the minute volumes of each species), nasal anatomy, and nasal air flow patterns from CFD simulations. The cyclic flow simulation was conducted for a reference resting rat and human exposed to 2 ppm acrylic acid for 3 min (minute volume 250 ml/min (rat), 7,500 ml/min (human)).

The CFD-PBPK model simulations predict that olfactory epithelium of the human nasal cavity is exposed to 2-3 fold lower tissue concentrations of acrylic acid than the olfactory epithelium of the rodent nasal cavity under either unidirectional flow exposure conditions or cyclic flow conditions. Frederick et al. (1998) are of the opinion that the model predicts olfactory tissue concentrations for acrylic acid that correlate with acute histopathological lesions observed in vivo (rats, exposed with 75 ppm acrylic acid for 3 or 6 h in a chamber) and with those observed in vitro (rats nasal septa incubated for two hours at 37°C with concentrations from 0.0 to 6.0 mM acrylic acid).

Metabolism

After oral administration of 4, 40, or 400 mg/kg bw [2,3-$^{14}$C]-acrylic acid in a 0.5% aqueous methylcellulose solution to rats within 72 h 44-65% of the radioactivity had been eliminated via expired air and 2.9-4.3% remained in the urine. The HPLC profile of metabolites observed in the urine of rats indicated two major metabolites. One of the major metabolites co-eluted was 3-hydroxypropionic acid. Radioactivity could not be detected at the retention times corresponding to that of 2,3-epoxypropionic acid or N-acetyl-S-(2-carboxy-2-hydroxyethyl)cysteine. One hour following an oral dose of acrylic acid (4, 40, 400 or 1,000 mg/kg) in rats a significant depletion of NPSH in the glandular stomach was reported at doses above 4 mg/kg. In the forestomach NPSH depletion occurred at a dose of 1,000 mg/kg. No significant effect of acrylic acid on NPSH in the blood or liver was observed (DeBethizy et al., 1987).

Winter et al. (1992) compared the metabolites of acrylic acid and propionic acid using $^{13}$C-NMR analysis of the urine of rats after gavage of single doses (400 mg/kg bw). 3-Hydroxypropionic acid, N-acetyl-S-(2-carboxyethyl)cysteine and N-acetyl-S-(2-carboxyethyl)cysteine-S-oxide were identified as metabolites of acrylic acid. No unchanged acrylic acid was detected. In contrast, the spectra of urine from a propionic acid-treated rat revealed only a few minor $^{13}$C-enriched signals that were assigned to methylmalonic acid. These metabolites (CO$_2$ and methylmalonic acid) are consistent with the known major vitamin B$_{12}$-dependent pathway of propionate metabolism in mammals. An alternative pathway involves β-oxidation. Acrylyl-CoA forms 3-hydroxypropionic acid that can then be oxidized to malonic semialdehyde. Further catabolism yields acetyl-CoA and CO$_2$. It is conceivable that excretion and detection of the mercapturates is a consequence of the high dose used in this experiment.

Following single doses (40 or 150 mg/kg) of [1-$^{14}$C]-acrylic acid to rats urinary metabolites and tissues were analyzed by HPLC. A major polar metabolite which could not be identified accounted for approximately 2 to 3% of the dose. A metabolite that coeluted with
3-hydroxypropionic acid was also detected. Small amounts of several other metabolites were detected. Plasma and liver from orally dosed rats were also analyzed for acrylic acid and metabolites by HPLC. One hour after dosing, a metabolite in plasma that co-eluted with 3-hydroxypropionic acid accounted for about 0.5% of the dose after 40 mg/kg bw. This metabolite was also detected in plasma after application of the higher dose. Neither acrylic acid nor metabolites were detected in plasma or liver at times later than 1 h. They were not detected in kidney at any time after administration (Black et al., 1995).

In other experiments, livers from mice dosed by gavage following a similar dosing regime were analyzed for acrylic acid and metabolites by HPLC. Several metabolites of higher polarity than those of acrylic acid including 3-hydroxypropionic acid were detected 1 h after administration, but not at times later than 1 h. Acrylic acid was not detected in livers from mice at any time after cutaneous administration of 40 mg/kg bw. After cutaneous dosing in rats, a peak that coeluted with acrylic acid was detected in urine along with the major metabolite found after oral dosing. A trace amount of another metabolite was detected in urine from the 40 mg/kg bw cutaneous dose group but not after dosing 10 mg/kg bw (Black et al., 1995).

**In vitro studies**

Hepatic microsomes were prepared using conventional methods from rats and incubations were started by the addition of 10 µl of [2,3-¹⁴C]-acrylic acid. No epoxidized metabolites could be detected and the parent compound was recovered from the incubation mixture unchanged (DeBethizy et al., 1987).

**In vitro** percutaneous absorption studies using excised human cadaver skin have indicated that acrylic acid absorption can vary significantly as a function of the pH value and nature of the vehicle. **In vitro** flux estimated after a 1 mg dose varied 600-fold within the treatments studied and decreased in the order acetone >> buffer pH 6.0 > buffer pH 7.4 (D'Souza and Francis, 1988).

Miller et al. (1981c) have studied the metabolism of acrylic acid in rat tissue homogenates. Acrylic acid did not react with reduced glutathione either in presence or absence of the soluble enzyme fraction. Non-protein sulphydryl concentrations were not appreciably lower in blood after addition of acrylic acid **in vitro**.

The rate of ¹⁴CO₂ formation from [¹⁴C]-acrylic acid was measured **in vitro** with preparations from rat liver hepatocytes. Rapid oxidation of acrylic acid to CO₂ was observed. Mitochondria isolated from the liver homogenates were incubated with acrylic acid under the same conditions and yielded higher rates of acrylic acid-oxidation than homogenates. HPLC analysis of the mitochondrial incubation mixtures indicated 3-hydroxypropionic acid as a major metabolite (Finch & Frederick, 1992).

Black et al. (1993) determined the rate of the **in vitro** oxidation of acrylic acid in 13 tissues of mice. The maximal rate of acrylic acid oxidation in kidney, liver and skin was 2,890, 616 and 48 nmol/h/g, respectively. In remaining organs acrylic acid was oxidized at rates less than 40% of the rate in liver. 3-Hydroxypropionic acid was the only metabolite detected by HPLC analysis.

Acrylic acid oxidation rates and blood tissue partition coefficients were studied in slices of rat tissue using [1-¹⁴C]-acrylic acid. Acrylic acid oxidation in rat kidney and liver slices was described by saturable kinetics with maximal rates of about 4 and 2 µmol/h/g, respectively. Acrylic acid oxidation rates in 11 additional tissues were 40% or less than that in liver (Black & Finch, 1995).
Conclusion

Acrylic acid is rapidly absorbed in rats and mice after oral or inhalation administration. A hybrid computational fluid dynamics and physiologically-based pharmacokinetics inhalation dosimetry model was constructed for interspecies (rat-human) extrapolation of acrylic acid tissue dose in the olfactory region of the nasal cavity. The model simulations indicate that under similar exposure conditions human olfactory epithelium is exposed with acrylic acid to 2-3 fold lower than rat olfactory epithelium. After dermal administration some acrylic acid is evaporated, the remainder undergoes rapid absorption in these animals. Dermal absorption is strongly dependent on the vehicle and the pH value of the solution.

Acrylic acid is rapidly metabolised by oxidative pathways to CO$_2$. The main metabolic pathway of acrylic acid seems to be a secondary, non-vitamin-B$_{12}$ dependent pathway of propionic acid metabolism consisting in reactions similar to fatty acid β-oxidation. In urine poorly characterized substances of a higher polarity than those of acrylic acid are detected. Unmetabolised acrylic acid could not be detected in urine, however small amounts of 3-hydroxypropionic acid were found. Epoxide intermediates were not detected. In vitro (stomach tissue) and in vivo acrylic acid reacts with GSH and NPSH to a very low extent. High dosages of acrylic acid leading to tissue damage cause the formation of small amounts of mercapturic acid derivates.

4.1.2.2 Acute toxicity

Studies in animals

In tests with animals, acrylic acid causes acute harmful effects by the oral and dermal routes of exposure. A review on acute toxicity studies with acrylic acid is given in a publication by Tyler et al., 1993.

The oral LD$_{50}$ values reported for rats cover a range from as low as 140 mg/kg up to 1,400 mg/kg (Dow Chemical 1979a, unpublished report) depending on the concentration of the test substance, similar acute oral toxicity is demonstrated for mice and rabbits (Carpenter et al., 1974). The only clinical signs observed during acute oral toxicity testing with this corrosive substance were a short reflex period of motor excitation followed by lethargy.

More detailed information on lesions caused after oral application of the acid are given within the report on a test with male rats treated with a 10% aqueous solution of acrylic acid (purity 99%, pH of the aqueous solution 2.5) where an oral LD$_{50}$ value of 1,350 mg/kg bw was detected. This aqueous solution caused mortalities within 2 days after treatment (no further data on methodology). In rats of the 700 mg/kg (non-lethal dose), 900 and 1,100 mg/kg dose groups that were killed 48 hours after treatment, histopathology revealed necrosis in the gastric epithels and irritation infiltrates in the gastric mucosa in approximately 50% of animals assessed. In addition, these animals demonstrated acute degeneration of liver parenchyme and in some cases liver necrosis. Animals necropsied 14 days after treatment did not show relevant pathologic changes (Majka et al., 1974).

Acute dermal toxicity is dominated by the severe local corrosivity caused by dermal contact; dermal LD$_{50}$ values of 300 mg/kg (Carpenter et al., 1974) and 640 mg/kg (BASF AG 1979, unpublished report) are demonstrated for rabbits. In the BASF study doses of 400 and 640 mg/kg of undiluted acrylic acid were occlusively applied for 24 hours to the skin of 5 male and 5 female rabbits per dose. After application of 400 mg/kg 1/5 males and 1/5 females died on day 7 or later; after application of 640 mg/kg 2/5 males and 3/5 females died within 24 hours. In addition
to severe local necroses, apathy, laboured respiration and poor general state were observed; necropsy demonstrated dilatated heart and lung oedema.

Data on acute inhalation toxicity of acrylic acid normally demonstrate severe irritation in the respiratory tract but no mortalities. Majka et al. (1974), however, state an inhalation LC\text{50} of 3,600 mg/m\textsuperscript{3}/4 h (3.6 mg/l/4 h) for male rats in a poorly reported study. The animals were exposed to acrylic acid vapours (purity of the acid 99\%) in an inhalation chamber of 0.045 m\textsuperscript{3} volume (dynamic system with air flow of 100-120 l/hour, no more data on methodology). A LC\text{50} of 3,600 mg/m\textsuperscript{3}/4 h was detected with mortalities occurring within 48 hours after treatment. Histopathology in rats killed 48 hours after treatment revealed in the 2,970 mg/m\textsuperscript{3} (non-lethal concentration) and 3,600 mg/m\textsuperscript{3} (concentration of the LC\text{50} value) groups hyperemia of the inner organs. In the respiratory system severe irritation of the bronchial mucosa, exudate into the bronchial lumen, macrophages in the vesicle lumen and focal intraparenchymal irritation in the lungs was observed. Necropsy at the end of the 14-day observation period demonstrated signs of respiratory irritation.

Carpenter et al. (1974) reported data from vapour inhalation tests with rats using glacial acrylic acid (no data on purity) as test substance within a list of test results for many different chemicals. The following data on inhalation toxicity of acrylic acid are given: Maximum inhalation time of 1 hour caused no deaths in rats inhaling “concentrated vapours” of acrylic acid (no information on this concentration); none of six rats died after a 4-hour inhalation of 2,000 ppm (5.9 mg/l/4h) of the substance (no data on testing methodologies).

In the majority of the other test reports acute inhalation toxicity is stated to be low - supposedly because acrylic acid interacts with humidity of the air prior to reaching the respiratory tract (BAMM, 1988, unpublished report) and causes respiratory irritation instead of acute inhalation toxicity (BASF AG, 1980, unpublished report). The LC\text{50} for rats inhaling acrylic acid vapours is reported to be >5.12 mg/l/4 h (BASF AG, 1980, unpublished report). The mentioned acute inhalation toxicity studies were conducted with rats using whole body exposure to “saturated” acrylic acid vapours. No mortalities occurred in these studies and the clinical signs of “respiratory irritation” were perinasal wetness and encrustation and abdominal breathing. No pathological changes were observed at necropsy. In addition, the laboratory staff of the 1988 study judged that the vapours interacted with the relative humidity of the water soluble test material prior to reaching the respiratory tract. Thus, the observed clinical signs of respiratory irritation demonstrate that substance vapours normally will not reach the lungs.

Studies in humans

No data available.

Conclusion on acute toxicity

Data on human experience with acute exposure to acrylic acid are not available. Pure acrylic acid is a very reactive chemical substance and accordingly exhibits severe corrosive properties in contact with biological material. Acute toxicity detected in animal tests consequently is dominated by chemical interactions with water and/or biological material. Thus, acrylic acid causes acute harmful effects by the oral and dermal routes of exposure. The oral LD\text{50} values for rats cover a range from as low as 140 mg/kg up to 1,400 mg/kg depending on the concentration of the test substance. An oral LD\text{50} of 1,350 mg/kg was detected for male rats in a study with a 10\% aqueous solution of acrylic acid - pH of this solution was 2.5 and thus, corrosive effects demonstrated in this study are not caused by the pH of the test substance (Majka et al., 1974); a dermal LD\text{50} of approximately 640 mg/kg was detected for rabbits in a study with undiluted
acrylic acid (BASF AG, 1979). Acute inhalation toxicity however is normally stated to be low because acrylic acid interacts with the humidity of the air prior to reaching the depth of the respiratory tract. Despite this an inhalation LC\textsubscript{50} of 3.6 mg/l/4 hours is detected for male rats in a study by Majka et al. The findings of acute toxicity the respiratory tract seem to depend on the mode of exposure. For classification, see Section 1.4.

4.1.2.3 Irritation/Corrosivity

Studies in animals

Acrylic acid causes severe burns to skin and eyes and severe irritation in the respiratory tract. Reported dose-response assessment for acute toxic effects caused by acrylic acid are based on demonstration of dose dependency of the severe corrosive properties of this chemical:

In a test according to EEC and OECD guidelines, a single topical application of 0.5 ml of 99.8% pure glacial acrylic acid to the intact skin of 5 Albino rabbits for 3 minutes under a semi-occlusive dressing caused in all animals brownish discolouration of the skin within 1 hour after substance application. In 2 animals the finding was assessed after the 1-hour reading by macroscopic pathology indicating superficial necrosis, slight oedema and discolouration of the application area. The brownish discolouration of the remaining 3 animals was assessed after 14 days by histopathology. This examination revealed deep focal necrosis (full thickness necrosis), loss of epidermal adnexa in necrosis area, perifocal moderate epithelial hyperplasia and diffuse inflammatory reaction (corium to subcutis) in the application area (BASF AG, 1998, unpublished report).

After 24 hours of occluded application of 400 mg/kg to the skin of rabbits 2 out of 10 animals died 7 days after exposure or later; after application of 640 mg/kg 5 out of 10 rabbits died within 24 hours (BASF AG, 1979, unpublished report). After 1 minute of exposure to undiluted acrylic acid or of a 50% aqueous substance solution rabbit skin exhibited necrosis; a 10% aqueous solution caused skin irritation after 5 minutes of exposure (BASF AG, 1958, unpublished report).

The serious damage to eyes caused by acrylic acid is not due to the acidic properties of this chemical. In an experiment with acrylic acid neutralized with potassium hydroxide (forming a neutral 60% aqueous solution of potassium acrylate) severe ocular damage was demonstrated for rabbit eyes not flushed with water immediately after instillation of the neutral solution: 0.1 ml of 60% (neutral) aqueous solution of potassium acrylate was instilled into each of the eyes of 9 albino rabbits. The eyes of 6 animals were flushed with lukewarm water. Three animals flushed 2 seconds after instillation and 3 animals flushed 4 seconds after instillation developed corneal opacities that cleared within 7 days. The 3 animals with unwashed eyes demonstrated irreversible severe ocular damage with corneal opacity occurring after 1 hour and persisting for the duration of the study (18 days). In addition, 1 animal was administered 0.1 ml and flushed with water 20 seconds later, and 1 animal was treated with 0.1 ml solution and flushed 4 seconds after instillation for a period of 1 minute; both animals developed irreversible corneal opacity (Hoechst Celanese Corp., 1992, unpublished report).

In a series of poorly reported Draize tests with rabbits the following relationship between eye lesions and concentrations of aqueous solutions of acrylic acid (purity 99%) was detected: The undiluted acid caused severe irritation that reversed within 20 days resulting in irreversible tissue changes such as scarring of the eyelids and corneal opacity. Similar but less pronounced lesions resulted after instillation of a 10% aqueous solution. A 3% solution caused irritation that
reversed within 6 days, lesions caused by a 1% aqueous solution disappeared 2 days after instillation of the test substance. No more data are reported (Majka et al., 1974).

All studies conducted in order to assess acute inhalation toxicity of acrylic acid demonstrate severe irritation in the respiratory tract. Majka et al. exposed rabbits to acrylic acid vapours (purity of the acid 99%) in an inhalation chamber of 0.045 m$^3$ volume (dynamic system with air flow of 100-120 l/hour, no more data on methodology). A LC$_{50}$ of 3,600 mg/m$^3$/4 h was detected with mortalities occurring within 48 hours after treatment. Histopathology in rats killed 48 hours after treatment revealed in the 2,970 mg/m$^3$ (non-lethal concentration) and 3,600 mg/m$^3$ (concentration of the LC$_{50}$ value) groups hyperemia of the inner organs. In the respiratory system severe irritation of the bronchial mucosa, exudate into the bronchial lumen, macrophages in the vesicle lumen and focal intraparenchymal irritation in the lungs was observed. Necropsy at the end of the 14-day observation period demonstrated signs of respiratory irritation (Majka et al., 1974). For further information see Section 4.1.2.2 on Acute toxicity of acrylic acid.

Studies in humans

Human data are submitted by industry mentioning that 3 accidents with acrylic acid occurred within the time period of 1967-1992: Two workers needed hospitalisation because of skin corrosion and 1 worker because of irritation of the respiratory tract (BASF AG, 1992, unpublished information).

Conclusion on irritation and corrosivity

Data on accidents at the workplace demonstrate that acrylic acid causes skin corrosion and irritation of the respiratory tract in humans. In tests with rabbits pure acrylic acid caused severe burns to skin and eyes; a 50% aqueous substance solution caused necrosis to rabbit skin after 1 minute of exposure, even a 10% aqueous solution caused skin irritation within 5 minutes. Severe ocular damage caused by acrylic acid cannot be avoided by neutralizing the acid. For classification, see Section 1.

4.1.2.4 Corrosivity

See Section 4.1.2.3

4.1.2.5 Sensitisation

Studies in animals

Eight acrylates and methacrylates including acrylic acid, hydroxyethyl acrylate, hydroxypropyl acrylate, aminoethyl methacrylate hydrochloride, Na-2-sulphoethylmethacrylate, 2-sulphoethyl methacrylate, hydroxyethyl methacrylate, hydroxypropyl methacrylate and an additional 64 substances were tested in guinea pigs with the use of a modified Split Adjuvant Test. The purity of the test substances is not mentioned. The highest concentration which did not cause primary irritation was used but no data are given on the test concentrations. Ten animals per test received a 0.1 ml aliquot of the test material to the backs four times in 10 days. At the time of the third application, 0.2 ml Freund’s adjuvant was injected at one point adjacent to the insult site. After a 2-week rest period, the guinea pigs were challenged with the test material on one flank and a solvent (if used) on the other flank. The challenge site was evaluated for erythema and oedema at
24 and 48 hours. Acrylic acid and six other acrylates and methacrylates were negative (0/10) and only aminoethylmethacrylate hydrochloride was positive in 1/10 animals (Rao et al., 1981).

In a modified Freund’s Complete Adjuvant test 8 guinea pigs/group received 3 intradermal injections during the induction phase on days 0, 5 and 9 (the test substance was mixed with FCA in a volume of 0.1 ml). Non-irritant test concentrations were used for challenge at day 21. The test concentrations for intradermal injections were 1.2% and for challenge 7.2% in Aramek, a mixture of 2 parts methyl ethyl ketone and 1 part of peanut oil. Distilled acrylic acid was negative but commercial acrylic acid is a strong skin sensitizer. The skin reactions were due to the presence of varying quantities (up to 7%) of \(\alpha,\beta\)-diacryloxypropionic acid (DAPA). Positive skin reaction was still present after a third challenge on day 49 (Waegemakers and van der Walle, 1984).

Recent investigations on the occurrence of DAPA in industrial acrylic acid have shown that DAPA is not present (at a detection limit of 20 ppm) in current commercial samples of acrylic acid (Elf Atochem, 1998).

The stabilizer added to the trade product (hydroquinone monomethyl ether) is a known skin sensitizer (EHC 191), however the concentration added (0.02% w/w) is too low to propose labeling.

There is no information available on the potential of acrylic acid to produce respiratory sensitisation in animals.

**Studies in humans**

Human data are available showing the severe local corrosive properties of acrylic acid. Exposed persons can exhibit contact dermatitis. One worker showed a positive reaction in a Patch test with acrylic acid but not with acrylic resin compounds (Fowler, 1990). One woman was patch tested with individual components of Fixomull® tape adhesive. A positive response to acrylic acid, a component of the tape, was demonstrated (Daecke et al., 1993). Negative results in six workers patch tested with 0.1% acrylic acid in petrolatum are also reported (Conde-Salazar et al., 1988). However, data on the purity of the acrylic acid products were not given by the different authors. Since 1989 more than 450 workers in production plants using acrylic acid as a base material have been regularly medically examined. No cases of sensitisation to acrylic acid have been observed (information by BASF AG, 1998).

Respiratory sensitisation has not been observed.

**Conclusion on sensitisation**

Pure acrylic acid does not show skin sensitising properties in animal sensitisation tests. But skin sensitisation was observed in humans. This might be caused by an impurity of acrylic acid, DAPA. However, data on the impurity in the acrylic acid samples tested in humans were not available. Recent investigations on the occurrence of DAPA in industrial acrylic acid have shown that DAPA is not present (at a detection limit of 20 ppm) in current commercial samples of acrylic acid. Medical examinations performed with more than 450 workers in production plants using acrylic acid as a base material demonstrated that since 1989 commercial acrylic acid and present occupational health protection precautions are sufficient to avoid sensitisation hazard at the workplace.

Respiratory sensitisation has not been observed in humans.
4.1.2.6 Repeated dose toxicity

Studies in animals

Several studies investigating the effects of acrylic acid after repeated oral and inhalation exposures of rats and mice were regarded as valid according to the requirements of the directive 793/93/EEC.

Oral administration

In an oral 90-day study (BASF AG, 1987; Hellwig et al., 1993) acrylic acid (approx. 99%) was administered by gavage in two dosages (150 and 375 mg/kg bw/d) to Wistar rats.

The clinical examinations revealed vocalization beginning after the first week of treatment and tympanies of the gastrointestinal tract, which was frequently connected with cyanosis and dyspnoea, in most of the animals from week 3 onwards. 6 males and 9 females of the high-dose group and 5 males and 5 females of the low-dose group died prematurely (day 14-81 of the treatment) showing apathy, hypothermia and piloerection before death. Dose-related severe toxic effects were recorded in both dose groups consisting of reduced body weight gain, thickening of the plica marginata and hyperaemia or erosions/ulcerations of the gastric mucosa. Degeneration/necrosis of renal tubules were observed in the five males in each of both dose groups and four and seven females of the low and high dose groups, respectively, that died during the study. The testing parameters did not include hematology, clinical chemistry, urinalysis; histopathological examination was done on selected organs of the gastrointestinal tract, liver, kidney, urinary bladder, adrenals, tongue, buccal and nasal mucosa. The LOAEL of this study was 150 mg/kg, no NOAEL was derived.

Wistar rats which received acrylic acid in the drinking water at doses of 120, 800, 2,000 or 5,000 ppm (approx. 6, 40, 100 or 200 mg/kg bw/d in males, and 10, 66, 150 or 375 mg/kg bw/d in females) for 3 months (10 rats/group/sex) and 12 months (20 rats/group/sex) showed reduced water consumption at 2,000 ppm and 5,000 ppm dosages (BASF AG, 1987; Hellwig et al., 1993). No treatment-related premature deaths occurred. Lower food consumption was seen in high dose males and reduced body weight gain was observed in males from 2,000 ppm and 5,000 ppm groups. Although there were transient decreases of red cell counts and hematocrit values and increased MCH and MCHC values for high dose males at week 12 only, no treatment-related effect on hematology, clinical chemistry, and urine parameters were found. No substance-related toxic effect could be microscopically demonstrated in a comprehensive list of organs examined in the two high doses. Obviously due to the bad palatability treated animals had lower drinking water uptake, which was considered to result in lower food consumption and body weight gain. Mortality and toxic effects in the kidney and the stomach found in the gavage study were not confirmed in this study at comparable doses indicating that these effects were attributable to high local and blood peak values after bolus administration by gavage. Based on the reduced body weight gain in males and lower water consumption in both sexes, the NOAEL of this study was considered to be 800 ppm (40 mg/kg). In females the NOAEL was considered to be 5,000 ppm (331 mg/kg) because reduced water consumption was not interpreted as a clear adverse health effect.

Fischer 344 rats (15 animals/sex/group) in another 90-day drinking water study (Bushy Run Research Center, Inter-Company Acrylate Study Group, 1980) were administered doses of 83, 250, or 750 mg/kg bw/d of acrylic acid. No deaths occurred during the treatment period. However, clear dose-related effects were observed.
At the high-dose level there was reduced food and water consumption, reduced body weight gain, lower organ weights of liver, kidneys, spleen, heart, brain, and elevated testes weight and some altered clinical chemistry parameters (increased levels of serum urea nitrogen, glucose, alkaline phosphatase and aspartate transaminase). Furthermore, there was a statistically significant decrease in total serum cholesterol noted for the high level females. In both sexes increases of urinary protein and specific gravity and a decrease in urinary pH values were noted. No significant prevalence of microscopic lesions was found in any of the animals.

At 250 mg/kg bw, a decrease in water consumption was noted for both sexes. Body weight gain was lower in females. Kidney weights were increased in both sexes and relative testes weights were increased in males. Effects on serum urea nitrogen, cholesterol and alkaline phosphatase in female rats and urinary specific gravity and protein in both sexes were similar, but less pronounced than those observed at the high dose level.

At 83 mg/kg bw the only effects noted were a reduction of water consumption by male rats and a slight increase in red blood cells in female rats. Both findings were not considered to be of toxicological relevance, therefore 83 mg/kg was the NOAEL of this study.

**Inhalation route**

A 90-day inhalation study on Fischer 344 rats and B6C3F1 mice (Miller et al., 1981a, 1981b; Dow Chemical Company, 1979b) using 15 animals/sex/group exposed to doses of 5, 25 or 75 ppm (approx. 0.015, 0.074 or 0.221 mg/l) of acrylic acid vapour on 6 hours/day on 5 days/week revealed lower mean body weight gains in female mice from 25 ppm and 75 ppm dose groups. Histopathological examinations were performed on tissues of 10 animals/sex/group.

Male mice of the 25 ppm and 75 ppm groups and in female mice of the 75 ppm group had a slight decrease of the mean hemoglobin concentration without further corroborative alterations of the hematological parameters. There were no relevant treatment-related effects on organ weights, hematological parameters, clinical chemistry parameters or urinary parameters.

Acrylic acid vapour induced slight focal degeneration of the nasal olfactory epithelium in 7/10 male and 10/10 female rats at the 75 ppm dose. No lesions of the nasal mucosa were observed in the mid and low dose groups.

Similar dose-related lesions on the nasal mucosa were demonstrated in all groups of treated mice which were examined microscopically. Slight to moderate focal degeneration of the olfactory epithelium was observed in 10/10 males and 11/11 females of the high dose group, 11/11 males and 9/10 females of the mid group and 1/10 males and 4/12 females of the low dose group. Nasal lesions in high dose animals showing degenerated olfactory epithelium were corroborated by focal replacement of the olfactory epithelium by a lower columnar-type epithelium which resembled respiratory-type epithelium. There also appeared to be very slight focal hyperplasia of the submucosa glands and infiltrations of inflammatory cells in the mucosa and submucosa in all animals affected. Areas normally lined by respiratory epithelium appeared to be totally unaffected. In the mid-dose group, 1/11 male and 2/10 females had also focal infiltration of inflammatory cells. Similar lesions were not observed in any mice of the control groups.

In this study, histopathology of four cross-sections were examined in 10 animals/sex/group at different levels of the nasal turbinates, being the target organ identified.

For local effects, this study revealed a NOAEC of 25 ppm for rats. No local NOAEC was derived in mice, the LOAEC (local) is 5 ppm. There was no systemic toxicity in rats and male
mice and systemic NOAEC was therefore 75 ppm. Because of lower body weight gain, the NOAEC for female mice was 5 ppm.

Other studies which were not in full compliance to the minimal requirements of OECD/EEC guidelines for 28-day studies on repeated dose toxicity are described hereafter to give further information on the substance.

In a range-finding study acrylic acid vapour was inhaled by Fischer 344 rats and B6C3F1 mice (5 animals/sex/dose) during 2 weeks (6 h/d, 5 d/week) at doses of 25, 75, and 225 ppm (approx. 0.074, 0.221 or 0.662 mg/l) (Miller et al., 1979). The high dosage resulted in clinical signs as nose scratching and reduced body weight gains in rats and mice of both sexes as well as in reduced fat depots in female rats. Dose-related inflammatory and degenerative lesions of the nasal mucosa were recorded in all rats and mice of the high dose groups, in all mice of the mid dose group and in 2/5 male mice and 4/5 female mice of the low dose group. All male and female rats in the 225 ppm group had some areas with focal squamous metaplasia of the nasal tissue.

Inhalation by 15 female B6C3F1 mice of 5 or 25 ppm acrylic acid for a period of 15 days (4.4, 6, or 22 h/d) resulted in disorganization and atrophy of the olfactory epithelium, basal cell hypertrophy with squamous differentiation, epithelial necrosis with desquamation, and Bowmans' gland degeneration (Lomax et al., 1994, Rohm and Haas, 1994). After a six-week recovery period of 5 females/group, animals which were exposed 22 h/d to 25 ppm acrylic acid vapour exhibited regions of respiratory metaplasia.

Following repeated inhalation of acrylic acid vapour 6 h/d in four male and four female rats/group toxic effects were briefly reported (Gage, 1970). Four male and four female rats exposed for 4 days to 1,500 ppm acrylic acid vapour showed nasal discharge, lethargy, weight loss and congested kidneys. Inhalation of 300 ppm acrylic acid vapour for 20 days resulted in nasal irritation, lethargy and reduced body weight gain. Vapour concentration of 80 ppm induced no sign of toxicity.

Respiratory function (respiratory minute volumes (-23% in rats, -27-34% in mice) and respiratory rates (-17% in rats, -32-37% in mice) were slightly depressed after inhalation of 75 ppm acrylic acid vapour by male rats and male mice for 5 days (6 h/d) (Barrow, 1986). Assuming an even distribution of acrylic acid, the dose expressed as acrylic acid concentration (µl/litre)/minute volume (l/min)/nasal cavity surface area (cm²) was nearly twice in mice (3.5-3.8 µl/min/cm²) than in rats (1.8-2.1µl/min/cm²). Histopathology of sections from four levels of the nasal cavity was characterised by severe lesions in both species. Mice had more severe lesions, as seen by the presence of more cellular exudate in the lumen of the nose and a much greater loss of sensory cells. Under the assumption of an evenly distributed chemical the damage of the tissue should be distributed regularly throughout the nasal cavity. However, the principal location of the lesions was on the dorsal meatus of level 3, where epithelial cell counts indicated a 50 percent decrease in mice versus 15 percent in rats. Cell proliferation studies showed a statistically significant increase in cell turnover of the olfactory epithelium in the dorsal meatus of both species. Turnover rate was 4 percent in treated rats (versus 0.9 percent in controls) and 1.7 percent in treated mice (versus 0.1 percent in controls).
Dermal route

Skin irritation becomes prevalent with higher incidence and severity in mice treated with 4% acrylic acid compared to 1% acrylic acid or vehicle control (acetone) after 13 weeks of dermal application (3 d/wk) (Basic Acrylate Monomer Manufacturers, 1991; Tegeris et al., 1988). No irritant effect was evident after long-term application of 1% acrylic acid in acetone in mice (Inter-Company Acrylate Study Group, 1982, see Section 4.1.2.8).

Studies in humans

No data available.

Effect levels used for the risk characterization

No-observed-adverse-effect-level (NOAEL/NOAEC)

Oral administration: 40 mg/kg bw/d (90 d, male rats)
(BASF AG, 1987; Hellwig et al., 1993)
83 mg/kg bw/d (90 d, female rats)
(Bushy Run Research Center, Inter-Company Acrylate Study Group, 1980)

Inhalation exposure: LOAEC for local effects
5 ppm (resp. 0.015 mg/l), 90 d, mice
(Miller et al., 1981a, 1981b; Dow Chemical Company, 1979b)

Inhalation exposure: NOAEC for systemic effects
5 ppm (0.015 mg/l) (90 d, female mice)
75 ppm (0.221 mg/l) (90 d, male and female rats, male mice)
(Miller et al., 1981a, 1981b; Dow Chemical Company, 1979b)

Conclusion on repeated dose toxicity

Overall, the toxic profile of acrylic acid is dominated by its local irritation effects irrespective of the way of application. Prolonged inhalation of concentrations from 5 ppm or higher in mice and 75 ppm in rats induced degeneration of the olfactory mucosa. It causes severe mucosal damage to the stomach after repeated gavage administration of \( \geq 150 \text{ mg/kg bw/d} \), but not after application via drinking water at similar or higher doses. Long-term exposure of the skin to acrylic acid at a concentration of \( \geq 1% \) resulted in irritation whereas no effect on the skin was evident at 1% (see also dermal carcinogenicity studies in Section 4.1.2.8). Following oral, dermal or inhalation administrations no other systemic toxic effects were detected except premature deaths and tubular degeneration/necrosis in the kidneys which were evident after gavage administration of dosages \( \geq 150 \text{ mg/kg bw/d} \) in a rat 3-month study. Effects were attributed to the high peak concentrations and did not occur in drinking water studies at similar or higher doses. Some studies with repeated application revealed minimal changes of single red blood cell parameters, however no clear hematotoxic effect was found. Changes in clinical chemistry parameters, observed in drinking water studies, were assumed to be associated with reduced consumption of water and/or food.
4.1.2.7 Mutagenicity

Bacterial systems

A bacterial mutation test with Salmonella typhimurium tester strains TA98, TA100, TA1535 and TA1537 was negative in concentrations up to 5,000 µg/plate with and without S-9 mix. Doses from 1,000 µg/plate upwards induced toxic effects (Cameron et al., 1991). Negative results were also found in further bacterial mutation studies (Zeiger et al., 1987; BASF, 1977).

In vitro systems with mammalian cells

A mammalian cell gene mutation test with CHO cells (HPRT locus) was negative at doses up to 1.9 µl/ml (2,000 µg/ml) without S-9 mix and up to 2.4 µl/ml (2,500 µg/ml) with S-9 mix. Survival at the highest concentrations was 35% and 24%, respectively (McCarthy et al., 1992).

In the mouse lymphoma assay increases of mutation frequencies were found in two independent investigations. Cameron et al. (1991) reported on a positive mouse lymphoma assay with and without metabolic activation: without S-9 mix 3-fold to 6-fold dose-related increases of mutant frequencies were induced in the dose range 2.65 to 5.44 mmol/l (191 to 392 µg/ml); with S-9 mix 3-fold to 8-fold increases were found in the dose range 16.2 to 26.5 mmol/l (1,167 to 1,910 µg/ml). The relative total growth at the highest concentrations was 15% and 20%, respectively. Another mouse lymphoma assay was run only without metabolic activation: in the dose range 300 to 600 µg/ml 4-fold to 8-fold increases of mutant colonies were induced in a dose-related and reproducible manner; the survival was about 20% at the highest concentration (Moore et al., 1988). In both studies the majority of the mutants gave small colonies.

Positive effects were described also for in vitro chromosomal aberration assays. McCarthy et al. (1992) reported a positive chromosomal aberration assay in CHO cells with and without metabolic activation. Without S-9 mix, doses of 3.8 and 5.0 µl/ml (3,942 and 5,230 µg/ml) induced 11% and 30% aberrant cells (2% in the negative control); with S-9 mix, in the dose range 1.6 to 2.8 µl/ml (1,689 to 2,977 µg/ml) aberration frequencies of 9% to 28% were found in a dose-related manner (1% in the negative control). The positive response was not bound to drastic cytotoxic effects (42% and 35% relative cloning efficiency at the highest concentrations) or to decreases in pH (pH was adjusted to pH 7.0). A chromosomal aberration assay with L5178Y mouse lymphoma cells was only run without S-9 mix. In the dose range 300-500 µg/ml 7%-21% aberrant cells were induced in a dose-related manner (4% in the control culture); the effect was not related to drastic cytotoxicity (Moore et al., 1988). Furthermore, Ishidate (1988) reported that acrylic acid induced chromosomal aberrations in CHL cells without S-9 mix at the highest dose tested of 750 µg/ml; cytotoxicity data are not given.

In primary rat hepatocytes acrylic acid did not induce unscheduled DNA synthesis (UDS) in non-toxic doses up to 0.4 µl/ml (420 µg/ml). Higher doses could not be analysed due to high cytotoxicity (McCarthy et al., 1992).

Wiegand et al. (1989) investigated induction of micronuclei, UDS and cell transformation in tertiary cultures of Syrian hamster embryo (SHE) cells without addition of an external metabolising system. Results, although negative for all three endpoints, are of low reliability due to the screening character of the test, poor description and methodological insufficiencies (e.g., in the micronucleus test only doses in the non-toxic range were analyzed; there is confusion whether diethylstilbestrol or B(a)P was used as positive control, concentrations were not given, nor the percentage of micronucleated cells).
Drosophila test

A test with Drosophila melanogaster for induction of sex-linked recessive lethal mutations was negative after administration via feeding or injection (McCarthy et al., 1992).

In vivo systems with mammals

Two in vivo bone marrow chromosomal aberration assays with rats gave negative results (McCarthy et al., 1992). Chromosome aberrations were analyzed (5 animals per sex, 50 metaphases per animal) at 6, 12, and 24 h after oral doses of 100, 333 or 1,000 mg/kg or after exposure to 2,000 or 5,000 ppm acrylic acid in drinking water for 5 days. In the acute as well as in the repeated dosing regimens the highest doses led to reduced body weight gains. Mitotic activity of bone marrow cells was not influenced by the treatments.

A dominant lethal assay led to negative results. Male mice were given single oral doses (gavage) up to 324 mg/kg or five daily oral doses up to 162 mg/kg. Immediately after dose administration male mice were mated. Females were checked for vaginal plugs each morning. Each mated female was replaced with a virgin female. The mating process was continued for 46 days. An analysis of the uterine contents of female mice was made 12-15 days after observation of the vaginal plug (McCarthy et al., 1992).

Mutagenicity data for structurally-related acrylic compounds

Negative results from in vivo bone marrow tests (micronucleus or chromosomal aberration assays) were reported for several structurally-related acrylic compounds:

- methyl methacrylate (Hachiya et al., 1982);
- ethyl acrylate (Ashby et al., 1989; Morita et al., 1997);
- methyl acrylate (Fh-ITA, 1994);
- butyl acrylate (Engelhardt and Klimisch, 1983).

Positive micronucleus assays were also reported, however, they seem to be of low reliability (Przybojewska et al., 1984; methodological insufficiencies) (Kligerman et al., 1991; weak effect in mouse splenocytes for doses in the toxic range paralleled by a negative in bone marrow chromosomal aberration test).

Conclusion on mutagenicity

Acrylic acid did not induce gene mutations in Salmonella or CHO cells (HPRT locus) but was clearly positive in the mouse lymphoma assay and in the in vitro chromosomal aberration test. Since in the mouse lymphoma assay small colonies were induced preferentially, the mutagenic potential of acrylic acid seems to be limited to clastogenicity. In vivo, acrylic acid did not induce mutagenic effects in either rat bone marrow cells or mouse germ cells after oral administration. Based on the present results and taking into account data on structurally-related acrylic compounds, it is unlikely that acrylic acid is mutagenic in vivo.
4.1.2.8 Carcinogenicity

Studies in animals

In a valid carcinogenicity study (BASF AG, 1989; Hellwig et al., 1993) Wistar rats were exposed to doses of 120, 400 or 1,200 ppm (mean substance uptake 9, 31, or 88 mg/kg bw/d) acrylic acid (99%, stabilized with 200 ppm hydroquinone monomethylether) in drinking water for 26 months (males) or 28 months (females). Except a slightly reduced water consumption in high-dose males and females no treatment-related clinical, hematological or histopathological changes were detected in comparison with the controls. The incidence and organ distribution of tumours found in the groups treated with acrylic acid did not differ from those of the controls (Table 4.4).

Studies which did not fulfil the requirements of guideline testing protocols for regulatory purposes give additional information on acrylic acid (see also Table 4.5):

In a dermal carcinogenicity study no tumours of the skin or subcutis were induced in treated mice or in the vehicle controls (Intercompany Acrylate Study Group, 1982). A group of 40 C3H/HeJ male mice received 25 µl applications of acrylic acid as 1.0% (v/v) dilutions in acetone. A negative control group received acetone only. The substances were applied to the skin of the back three times weekly for lifetime. Histological examination was performed on the dorsal skin of all treated mice and on gross lesions. The mortality rate was not affected by treatment (mean survival time in the acrylic acid group 515 days, in the acetone group 484 days). No signs of skin irritation were observed. One male of the acrylic acid group showed an epidermal hyperplasia.

In another dermal carcinogenicity study 25 or 100 µl of 1% (v/v) acrylic acid in acetone was administered to two strains of mice (C3H/HeN Hsd BR, Hsd:(ICR)BR) during 21 months (3 times/week). Histopathology was done on the skin, some internal organs and unusual gross lesion. No treatment-related signs of skin irritation, toxicity, clinical signs or skin tumors were observed. There was no treatment-related effect on body weight gain or mortality rate. 7/50 female C3H-mice of the 100 µl acrylic acid treated group revealed a significant increased frequency of lymphosarcoma compared to the acetone control group (BAMM 1990, 1991; TSCATS, 1992a), but lymphosarcomas are commonly seen in most strains of mice which are 18-24 months of age (Frith and Wiley, 1981) and their relation to the treatment was considered to be uncertain.

After subcutaneous administration of 1.4 mg acrylic acid in 0.5 ml trioctanion in 2/30 female Hsd-(ICR)Br mice two sarcomas were observed at the application site after 49.5 weeks (once weekly) (Segal et al., 1987). Malignancies were not observed in 20 mice receiving solvent (trioctanion) alone and in 100 untreated mice. These results were not considered to be relevant because of the application route (Grasso, 1987).

Studies in humans

No information on potential human carcinogenicity is available.

Conclusion on carcinogenicity

There is no evidence that acrylic acid administered orally to rats or applied dermally to mice is carcinogenic. There are no cancer data available with respect to human exposure.
### Table 4.4 Oral carcinogenicity study on acrylic acid (AA)

<table>
<thead>
<tr>
<th>Species/strain No. of animals/sex/group</th>
<th>Exposure time</th>
<th>Treatment schedule</th>
<th>Mortality rate</th>
<th>Treatment-related tumour response</th>
<th>Study design according to the B32/B33 method</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rat/Wistar, 50/sex/group</td>
<td>26/28 months</td>
<td>120, 400, 1200 ppm AA in drinking water</td>
<td>Ø</td>
<td>no</td>
<td>yes</td>
<td>BASF (1989)</td>
</tr>
</tbody>
</table>

Ø No treatment-related effects on the mortality rate and mean survival time

### Table 4.5 Dermal carcinogenicity studies with acrylic acid (AA)

<table>
<thead>
<tr>
<th>Species/strain No. of animals/sex/group</th>
<th>Exposure time</th>
<th>Treatment schedule</th>
<th>Mortality rate</th>
<th>Skin irritation</th>
<th>Skin hyperplasia</th>
<th>Skin tumours</th>
<th>Tumour response of internal organs</th>
<th>Study design according to the B32/B33 method</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mouse/C3H/HeJ 40 males</td>
<td>life time</td>
<td>25 µl AA (1% v/v in acetone)</td>
<td>Ø</td>
<td>no</td>
<td>1/40</td>
<td>0/40</td>
<td>no</td>
<td>no</td>
<td>Intercompany Acrylate Study Group (1982)</td>
</tr>
<tr>
<td>Mouse/C3H/HeN Hsd BR, 50/sex/group</td>
<td>life time</td>
<td>25 or 100 µl AA (1% v/v in acetone)</td>
<td>Ø</td>
<td>no</td>
<td>0/50 for each sex</td>
<td>0/50 for each sex</td>
<td>no</td>
<td>100 µl AA: 7/50 females with lympho sarcoma</td>
<td>no</td>
</tr>
<tr>
<td>Mouse/Hsd: (ICR)BR 50/sex/group</td>
<td>life time (86-92 weeks)</td>
<td>25 or 100 µl AA (1% v/v in acetone)</td>
<td>Ø</td>
<td>no</td>
<td>0/50 for each sex</td>
<td>0/50 for each sex</td>
<td>no</td>
<td>no</td>
<td>BAMM (1990)</td>
</tr>
</tbody>
</table>

Ø No treatment-related effects on the mortality rate and mean survival time
4.1.2.9 Toxicity for reproduction

Fertility

Possible effects on reproductive performance were investigated by oral administration (via drinking water) in two different studies with rats.

In a one-generation study with F334/N rats (DePass et al., 1983), the animals (10 males and 20 females per dose group) received acrylic acid at dose levels corresponding to 0, 83, 250 or 750 mg/kg bw/d for 13 weeks. Each male was then mated with 2 females and exposure continued for both sexes throughout gestation and lactation. Dose-related reductions in food and water consumption and consequently in body weight gain were observed in the F₀ animals, most pronounced and statistically significant at the 750 mg/kg bw/d dose level. At the high-dose level the fertility index of males and females, the gestation index, the number of pups born alive and the percentage of pups weaned were clearly reduced. In the high-dose group, pups of both sexes showed decreased body weight gain, also in males a reduction in absolute and relative liver weights and in females a reduction in both absolute and relative spleen weights was observed. Nevertheless, these findings were not considered as an indication of any substantial deleterious effect of acrylic acid on reproductive performance, because there were no statistically significant differences among the treated and the control groups. However, the fertility index and litter size of the control group of this study were atypically low.

In a two-generation study (OECD 416) acrylic acid was administered orally (in drinking water) to Wistar rats at doses of 0, 500, 2,500, 5,000 mg/l (53, 240, 460 mg/kg bw/d). The following results were observed (BASF, 1994c; Hellwig et al., 1997): in the male F₀ parental generation there were no signs of general toxicity. In the female parental generation reduction of food and drinking water consumption were observed at 5000 mg/l during the period of pregnancy. Dose dependent reduction of food and drinking water consumption during the lactation period were observed at 2,500 mg/l.

In the F₁ generation in both sexes dose-dependent reduction of food and drinking water consumption at 2,500 mg/l were observed. Body weight and body weight gain in both sexes were reduced. But in both sexes of the F₀ and F₁ generation there were no abnormal clinical signs. No adverse effects on fertility and pre-implantation development could be detected; no effects on reproductive organs have been observed. The mating index of males in both generations and in all dose groups was 100%.

The fertility rate in the F₀ generation was between 92-96%; in the F₁ generation in all dose groups the fertility rate was 100%. The rate of pregnancy in both generations was not reduced. In both generations there were no differences in numbers of pups born alive.

NOAEL for reproductive function was 460 mg/kg bw/d.

NOAEL for general toxicity was 240 mg/kg bw/d for the F₀ generation, but 53 mg/kg bw/d for the F₁ generation.

Developmental Toxicity

Oral

Developmental studies with the oral route of administration are not available. However, in the above-mentioned two reproductive toxicity studies in rats acrylic acid (in drinking water) produced some signs of postnatal developmental toxicity in offspring, predominantly decreased
body weight gain, following the exposure of the parental generation at a dose level leading to reduced food and water intake and to reduced body weight gain in the F₁ animals. No gross abnormalities were observed in the offspring in either study (DePass et al., 1983; BASF, 1994 c; Hellwig et al., 1997).

NOAEL (offspring): 53 mg/kg bw/d.

**Inhalation**

Groups of 30 pregnant Sprague-Dawley rats were exposed (6 h/d, whole-body) to atmospheres containing acrylic acid at 0, 40, 120, and 360 ppm (= 0, 120, 350 and 1,060 mg/m³) during days 6 to 15 of gestation. After exposure the dams were observed up to day 20 of gestation (Klimisch and Hellwig, 1991). The animals’ body weight and food consumption were determined on gestation day 0 and subsequently on every third day up to gestation day 20. After sacrifice dams were subjected to a gross pathological examination. After external examination of each foetus their body weights and lengths were measured and they were further processed for skeletal and visceral examination. In the dams, irritation of the respiratory tract and the eyes was observed in the highest dose group. A dose-related reduction in food and water intake resulting in a decrease in body weight gain was observed in the 120 and 360 ppm groups. Also in the 40 ppm group a slight but statistically significant effect was seen on body weight gain of the dams. A NOAEL for maternal toxicity could therefore not be derived from this study. But no effects on reproductive performances were observed. There were no signs of group-related trends or significant differences between groups in terms of pre-implantation losses, live foetuses, or resorptions. There were also no signs of group-related differences in the incidences of abnormalities, variations, or retardations in the foetuses in terms of general appearance, foetal body weights and the conditions of the internal organs or the skeleton.

Groups of 16 pregnant New Zealand rabbits were exposed (6 h/d, whole-body) to atmospheres containing acrylic acid at 0, 25, 75, and 225 ppm during days 6-18 of gestation (Bushy Run Research Center, 1993; Neeper-Bradley et al., 1997). All doses were observed daily for morbidity and mortality. During the exposure period, animals were observed for clinical signs preceding and subsequent to daily exposures and from outside during actual exposures. Maternal body weights were measured on gestation day 0, 3, 6, 12, 24, and 29. Food consumption was measured daily throughout the study beginning on gestation day 3. After sacrifice on gestation day 29, maternal liver and kidney weights were determined. All foetuses were weighed and examined for external malformations and variations, for thoracic and abdominal visceral abnormalities including internal sex organs, for craniofacial abnormalities and for skeletal malformations and variations. Dose-related clinical signs (as perinasal/perioral wetness and nasal congestion, as well as reduced body weight gain and food consumption) were observed in the 75 and 225 ppm groups. The overall pregnancy rate was equivalent for all groups (94-100%). No dose-related effects were observed in the reproduction function of the dams. There were no effects on the number of ovarian corpora lutea, the number of total viable or non-viable (early and late resorptions and dead foetuses) implantations/litter. Percentage live foetuses and sex ratio were equivalent across groups. Foetal body weights were unaffected by test substance exposure. There were no exposure-related increases in the incidences of external, visceral or skeletal malformations or variations.

NOAEL for maternal toxicity was 25 ppm (Bushy Run Research Center, 1993; Neeper-Bradley et al., 1997).
Inhalation exposure of pregnant rats and rabbits to atmospheres containing acrylic acid at concentrations up to 360 ppm (rats) and 225 ppm (rabbits) produced no evidence of developmental toxicity in either species.

NOAEL (rats): 360 ppm = 1,060 mg/m$^3$

NOAEL (rabbits): 225 ppm = 663 mg/m$^3$

(BASF, 1983; Klimisch and Hellwig, 1991; Bushy Run Research Center, 1993; Neeper-Bradley et al., 1997).

Human data: not available.

Conclusion on toxicity for reproduction

In oral reproductive toxicity studies (rats) no effects on reproductive function (fertility) were observed. Some signs of postnatal developmental toxicity (retarded body weight gain of the pups) were seen following exposure of the parental generation, however at dose levels that led to reduced food intake and weight gain in the dams. No gross abnormalities were observed in the offspring. A NOAEL/fertility of 460 mg/kg bw/d was derived from a guideline 2-generation study in rats (BASF, 1994c; Hellwig et al., 1997). No prenatal developmental toxicity was observed (rats and rabbits, inhalation), even at concentration levels that produced some signs of maternal toxicity. No specific teratogenic potential could be revealed for dose levels up to and including 360 ppm (rats), resp. 225 ppm (rabbits). A NOAEL/developmental toxicity of 225 ppm (according to 663 mg/m$^3$) was derived from the developmental toxicity study in rabbits (Bushy Run Research Center, 1993; Neeper-Bradley et al., 1997). According to the present database for toxicity for reproduction there are no reasons to classify acrylic acid as a reproductive toxicant.
4.1.3 Risk characterisation

4.1.3.1 General aspects

Acrylic acid is absorbed via the lungs in animals and humans, absorption via the oral and dermal routes of exposure is demonstrated. In animals with solely nasal respiration, it is resorbed at the nasal mucosa. A hybrid computational fluid dynamics and physiologically-based pharmacokinetics inhalation dosimetry model was constructed for interspecies (rat-human) extrapolation of acrylic acid tissue dose in the olfactory region of the nasal cavity. The model simulations indicate that under similar exposure conditions human olfactory epithelium is exposed with acrylic acid to 2-3 fold lower than rat olfactory epithelium. After dermal administration some acrylic acid is evaporated; the remainder undergoes rapid absorption. The extent of absorption depends on pH and solvent with direct dependency on substance concentration. Mouse skin shows better permeability than human skin. Whole body distribution was observed. In mice acrylic acid is rapidly and completely metabolised mainly in liver and kidney by the normal catabolic pathways by beta-oxidation of fatty acids and in the citrate cycle. Elimination preferably occurs as carbon dioxide (exhalation). Small amounts of 3-hydroxypropionic acid but no unmetabolised acrylic acid could be detected in urine.

Acrylic acid causes acute harmful effects following oral and dermal exposure in rats (oral LD$_{50}$: 140 up to 1,400 mg/kg bw; dermal LD$_{50}$: 300-600 mg/kg bw). Acute inhalation toxicity is normally stated to be low because acrylic acid interacts with humidity of the air prior to reaching the respiratory tract and causes respiratory irritation. Inhalation LC$_{50}$ values ranging from 3.6 to >5.1 mg/l/4 h have been determined. Human data are not available. Acrylic acid causes severe burns to skin and eyes in animals and severe irritation in the respiratory tract. The severe corrosive properties of the substance are demonstrated in a dose-dependent manner. In humans acrylic acid causes skin corrosion and irritation of the respiratory tract.

Persons exposed to acrylic acid can exhibit contact dermatitis. This was attributed to oligomeric impurities in the raw material; the pure acid does not show skin sensitizing properties.

Following repeated oral and inhalation exposure of acrylic acid in rats and mice, dose-related severe toxic effects were recorded. Gavage treatment with acrylic acid for 90 days revealed dose-dependent mortality, irritation and ulceration of the stomach, and renal tubular necrosis in rats (LOAEL 150 mg/kg bw/d).

No specific toxic effects were noted in further studies where acrylic acid was given with the drinking water. Reduced palatability (decreased water consumption) and non-specific signs of toxicity (decreased food consumption, body weight gain) at dosages >2,000 ppm (100 mg/kg bw/d in male rats, 150 mg/kg bw/d in females) were observed in subchronic and chronic studies A NOAEL of 40 mg/kg bw/d was derived for male rats, and of 83 mg/kg bw/d for female rats.

Toxic effects of relevance were seen in inhalation studies on rats and mice. In a 90-day inhalation study, acrylic acid vapour induced degenerative lesions on the olfactory mucosa in mice at 5 ppm (0.015 mg/l) and in rats at 75 ppm (0.0221 mg/l). Mice seemed to be more sensitive than rats, thus a LOAEC of 5 ppm (0.015 mg/l) was derived for local effects.

Long-term dermal exposure at concentrations >1% resulted in skin irritation.

There is no information on the health effects in humans of repeated exposure to acrylic acid.
Acrylic acid did not induce gene mutations in Salmonella or CHO cells (HPRT locus) but was clearly positive in the mouse lymphoma assay and in the in vitro chromosomal aberration test. Since in the mouse lymphoma assay small colonies were induced preferentially, the mutagenic potential of acrylic acid seems to be limited to clastogenicity. In vivo, acrylic acid did not induce mutagenic effects in either rat bone marrow cells or mouse germ cells after oral administration. Based on the present results and taken into account data on structurally-related acrylic compounds, it is unlikely that acrylic acid is mutagenic in vivo.

There is no concern for carcinogenicity from long-term studies on animals.

In oral reproductive toxicity studies (rats) no effects on reproductive function (fertility) were observed but some signs of postnatal developmental toxicity (retarded body weight gain of the pups) were seen following exposure of the parental generation. No gross abnormalities were observed in the offspring. No prenatal developmental toxicity was observed (rats and rabbits, inhalation).

### 4.1.3.2 Workers

In Table 4.6, a summary of the effects which are relevant for the occupational risk assessment is given.

<table>
<thead>
<tr>
<th>Acrylic acid</th>
<th>Inhalation</th>
<th>Dermal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute toxicity</td>
<td>LC50 rat: 3,600 mg/m³/4 h</td>
<td>LD50 values for rabbits: 300 and 640 mg/kg bw</td>
</tr>
<tr>
<td>Irritation/Corrosivity</td>
<td>Respiratory tract irritant;</td>
<td>Pure substance: corrosive;</td>
</tr>
<tr>
<td>Sensitisation</td>
<td>No data; not suspected to be a respiratory tract sensitizer</td>
<td>Not a skin sensitizer (purified acrylic acid)</td>
</tr>
<tr>
<td>Repeated dose toxicity (local)</td>
<td>Respiratory tract irritant; LOAEC in mice: 5 ppm (15 mg/m³), a CFD/IPPK-model indicates a lower sensitivity of humans</td>
<td>Pure substance: corrosive; long term irritation threshold concentration between 4% and 1%</td>
</tr>
<tr>
<td>Repeated dose toxicity (systemic)</td>
<td>N(O)AEC (with and without extrapolation): 5 ppm (15 mg/m³)</td>
<td>Extrapolated NAEL greater than 600 mg/person/day</td>
</tr>
<tr>
<td>Mutagenicity</td>
<td>Unlikely to be mutagenic in vivo</td>
<td></td>
</tr>
<tr>
<td>Carcinogenicity</td>
<td>Not suspected to be carcinogenic (experimental data)</td>
<td></td>
</tr>
<tr>
<td>Fertility impairment</td>
<td>Not considered to be reprotoxic (experimental data)</td>
<td></td>
</tr>
<tr>
<td>Developmental toxicity</td>
<td>Not considered to be reprotoxic (experimental data)</td>
<td></td>
</tr>
</tbody>
</table>

For the purpose of risk assessment it is assumed that inhalation of vapour and skin contact are the main routes of exposure. Oral exposure it not considered to be a significant route of exposure.
4.1.3.2.1 Acute toxicity

Inhalation

Experimental investigation of the acute inhalation toxicity (rat, 4 h) of acrylic acid indicated a LC50 of 3,600 mg/m³. No lethality but local effects in the respiratory tract and hyperemia of the inner organs were observed at 2,970 mg/m³. Concerning respiratory tract irritation following exposure by inhalation please refer to “Irritation/Corrosivity/Inhalation” and “Repeated dose toxicity/Inhalation (local)”.

This value of 2,970 mg/m³ without lethality is much higher than the measured short-term value of 44.4 mg/m³ (assumed reasonable worst case) in the chemical industry. Therefore lethality due to inhalation exposure is not anticipated to occur.

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

Dermal

Dermal LD50 values of 300 mg/kg and 640 mg/kg bw are demonstrated for rabbits. Acute dermal toxicity is accompanied by corrosive effects to the skin.

The highest dermal exposure is estimated to be 42 mg/p/d (= 0.6 mg/kg/d) in the chemical industry and in industrial area when single skin contacts to acrylic acid are possible. Lethality due to skin contact is not anticipated to occur.

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

4.1.3.2.2 Irritation/Corrosivity

Dermal/Eyes

Acrylic acid led to severe burns of the skin. A 50% solution caused necrosis to rabbit skin after 1 minute and even a 10% solution led to irritation within 5 minutes. The repeated administration of a 4% solution to the skin of mice over 13 weeks (3 d/w) caused irritation while the administration of a 1% solution for 13 weeks or lifetime led to no effects. Acrylic acid caused also severe burns to the eyes. Irreversible changes were observed after application of the pure substance and a 10% solution to the eyes of rabbits. A 3% and a 1% solution led to irritation that reversed within 6 and 2 days respectively.

Acrylic acid is labelled with R 35 (Causes severe burns) and according to the preparations directive (88/279/EEC) a concentration of ≥ 10% should be labelled with R 35, a concentration of ≥ 5% and < 10% with R 34 and a concentration of ≥ 1% and < 5% with R 36/38. A 10% solution did not result in corrosivity as supposed by the labelling scheme, but the observation time of 5 minutes was too short, and corrosivity after a longer period is not excluded. In addition the 1% solution did not result in skin irritation. But overall the experimental data are not in a noteworthy contradiction to the above-mentioned labelling. The scheme is considered to be a pragmatic estimate of local effects of preparations.
Scenarios with corrosive AA preparations

In exposure scenarios where pure acrylic acid or corrosive preparations of acrylic acid (≥ 5% AA) are handled skin and eye contact is avoided by using suitable PPE. Potential exposure is assumed only by single contacts. These scenarios are all exposure situations in the chemical industry, the manufacture of adhesives in the industrial area and the use of adhesives with ≥ 5% AA in industrial and skilled trade applications.

Scenarios with irritant preparations

There are two scenarios where irritant preparations of acrylic acid (content < 5% AA) are handled. These are the use of adhesives with < 5% AA in the industrial area and skilled trade. In these situations it cannot be excluded that PPE is not worn. Skin and eye irritation may occur. Acute irritation (skin, eye) is a reversible adverse effect, which can be immediately recognised and prevented. These risk situations are not anticipated to result in additional types of risk reduction measures beyond those which are already being applied (e.g. appropriate hygiene measures).

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

Inhalation

Acute irritation testing revealed severe irritation of the respiratory tract of rats at 2,970 mg/m³ (4 h). A threshold for acute respiratory irritation is not described. With reference to the section on repeated dose toxicity, it is anticipated, that the respiratory tract irritation threshold for single exposure does not significantly differ from that for repeated exposure. This consideration implies that the assessment of local effects after repeated inhalation (see “Repeated dose toxicity”) may be used for the assessment of short-term exposure as well. Experimental data concerning different exposure durations per day are not available. As a pragmatic approach it is assumed that the LOAEC of 5 ppm (6 h/d) is also appropriate to assess short-term exposure.

With regard to local chronic inhalation toxicity exposure situations in industrial areas like manufacture of adhesives (without LEV) and use of adhesives (with and without LEV) are evaluated as being of concern (see Section Repeated dose toxicity Inhalation (local) and Table 4.7). Additionally exposure situations either short-term or not occurring on a daily basis like production and further processing in the chemical industry (with an assumed reasonable worst case of 44.4 mg/m³ as short-term value) and use of adhesives in skilled trade applications (exposure level: < 30 mg/m³, shorter than shift length) give rise to concern with regard to acute respiratory irritation.

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

4.1.3.2.3 Sensitisation

Dermal

Acrylic acid is not considered to be a skin sensitiser. Dermal exposure of workers to acrylic acid is not anticipated to result in skin sensitisation.

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

Respiratory sensitisation has not been observed in humans and a sensitising potential was not observed in skin sensitisation testing. Overall acrylic acid is not suspected to be a respiratory sensitiser.

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

### 4.1.3.2.4 Repeated dose toxicity

**Inhalation (local)**

Vapours of acrylic acid are irritating to the upper respiratory tract. The assessment of its local irritation potency is based predominantly upon the available results of the 90-day inhalation studies in rats and mice. In both species tested degenerative changes to the olfactory epithelium were observed. For rats the NOAEC is 25 ppm, the LOAEC is 75 ppm. In mice, slight degeneration of olfactory epithelium was observed in some animals even at the lowest concentration of 5 ppm; at 25 ppm significant irritating effects were observed in almost all animals.

Comparison of results of the 2-week and 90-day inhalation studies with acrylic acid (see hazard assessment and discussion in WHO/IPCS Environmental Health Criteria 191, p 74-75) reveals that the effects caused by acrylic acid are largely determined by the exposure concentration and are relatively less affected by the duration of exposure in repeated exposure studies. Furthermore, results of a chronic inhalation study with the acrylic acid methyl ester indicate that most changes in the rat nasal mucosa developed during the first 12 months of exposure and increased only moderately with ongoing exposure up to 24 months (Reininghaus et al., 1991). In addition, comparison of methyl methacrylate chronic and subacute inhalation studies (see EU Risk Assessment Report for methyl methacrylate) supports the conclusion that progression of lesions of the olfactory epithelium might be minimal. Thus in conclusion, it may be assumed that the nasal irritation threshold for acrylic acid will not substantially change when extrapolation is made from experimentally-tested subchronic exposure to chronic exposure.

The main problem in the acrylic acid risk assessment is the species extrapolation from rodents to humans. Rodents show a nasal anatomy and respiratory physiology different from man. For instance, the architecture of nasal passages is more complex in rodents than in humans. These differences will influence the toxicokinetics of substances in the upper respiratory tract of species. A CFD/PBPK-model was constructed for interspecies (rat-human) extrapolation of acrylic acid tissue dose in the olfactory region of the nasal cavity. The model simulations indicate that under similar exposure conditions human olfactory epithelium is exposed to a 2-3-fold lower dose compared to rat olfactory presentation with regard to the uncertainty of the model and the sensitivity parameters (see Section 4.1.2.1). Therefore the calculation of both the direct and adjusted MOS is performed with the LOAEC of 5 ppm (15 mg/m³), that resulted in slight effects in some mice (near to NAEC).

The LOAEC of 15 mg/m³ is compared with the exposure information for scenarios with repeated daily inhalation exposure. For details see **Table 4.7**. The most critical exposure scenarios are found in the industrial area outside the chemical industry (manufacture of adhesives without LEV: MOS 0.4-2, use of adhesives without LEV: MOS 0.5, with LEV MOS 2-10). These MOS values are considered to be of concern.

**Conclusion (iii)** There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.
Table 4.7  Repeated dose toxicity (inhalation/local and systemic and dermal contact/systemic): MOS values and conclusions

<table>
<thead>
<tr>
<th>Area of production and use</th>
<th>Inhalation (local, systemic)</th>
<th>Dermal contact (systemic effects)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Shift-average value [mg/m³]</td>
<td>Direct MOS = adjusted MOS 1,2</td>
</tr>
<tr>
<td></td>
<td>local</td>
<td>systemic</td>
</tr>
<tr>
<td>Chemical Industry</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Production, further processing (filling, transfer, cleaning, maintenance, repair work)</td>
<td>3.0 4)</td>
<td>5</td>
</tr>
<tr>
<td>Manufacture of adhesives (1 - 10% acrylic acid) (filling, transfer, cleaning, maintenance, repair work, drumming)</td>
<td>0.375 - 2.25 6)</td>
<td>7 - 40</td>
</tr>
<tr>
<td>Industrial area</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Manufacture of adhesives (1 - 10% acrylic acid) (filling, transfer, cleaning, maintenance, repair work)</td>
<td>0.375 – 2.25 6)</td>
<td>7 – 40</td>
</tr>
<tr>
<td>Use of adhesives</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- ≥ 5% acrylic acid (handling, glueing, charging)</td>
<td>1.5 - 9 6)</td>
<td>2 - 10</td>
</tr>
<tr>
<td>- &lt; 5% acrylic acid (handling, glueing, charging)</td>
<td>1.5 - 9 6)</td>
<td>0.5</td>
</tr>
<tr>
<td>Decomposition during production of integrated circuits</td>
<td>low 5)</td>
<td>high</td>
</tr>
</tbody>
</table>

1) LOAEC (local): 15 mg/m³
2) NO(A)EC (systemic): 15 mg/m³
3) NAEL 150 mg/p/d resp. > 600 mg/p/d
4) 90th percentile
5) expert judgement
6) EASE (inhalation, with LEV)
7) EASE (inhalation, without LEV)
8) EASE (dermal, without PPE
Inhalation (systemic)

There was no systemic toxicity in rats and male mice, systemic NOAEC therefore was 75 ppm. Because of lower body weight gain, the NOAEC for female mice was 5 ppm with a systemic LOAEC of 25 ppm. Overall, comparison of local (LOAEC of 5 ppm) and systemic dose responses shows that the toxic profile of acrylic acid is dominated by its local effects in the upper respiratory tract. The formal systemic LOAEC is 5 times greater than the local LOAEC, a clear systemic target organ was not found and it is not excluded, that lower body weight gain might be secondary to the predominant local effects at 25 ppm. A comparison of subchronic and chronic oral studies with acrylic acid shows that a specific duration adjustment is not necessary for systemic effects. So a MOS-calculation (direct and adjusted) of systemic effects could be based on the N(O)AEC of 5 ppm (= 15 mg/m³).

On the basis of this N(O)AEC the corresponding MOS values for repeated dose toxicity (systemic) are the same figures as the local MOS values the latter being based on the local LOAEL. Referring to the hazard assessment part of the report the lower body weight gain at 25 ppm is not described as secondary to local effects. So local and systemic risks need to be evaluated separately.

The MOS values calculated for the manufacture of adhesives without LEV (0.4-2) and use of adhesives without LEV (0.5) in industrial applications are evaluated as being of concern. Unlike the evaluation of local chronic inhalation risks the industrial area of use of adhesives with LEV is not considered of concern with regard to systemic toxicity. The different starting point for MOS calculation [LOAEC (local) resp. N(O)AEC (systemic)] has to be taken into consideration (results see Table 4.7).

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

Dermal (local)

Please refer to the preceding Section “Irritation/Corrosivity: Dermal/ Eyes”.

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

Dermal (systemic)

For chronic dermal contact to acrylic acid preparations which are not labelled as corrosive possible systemic toxicity needs to be discussed. The results of the 90-day inhalation studies and the drinking water studies may be used for route-to-route extrapolation.

In the inhalation studies (with nasal cavity toxicity as primary effect) lower body weight gain with a NOAEC of 5 ppm (15 mg/m³) was observed in female mice (respectively a NOAEC of 75 ppm for rats and male mice). The obstacles that complicate the identification of an independent systemic toxicity are already mentioned. Assuming a breathing volume of 10 m³ per shift, an inhalation intake of 150 mg/person/day (based on female mice) respectively 2,250 mg/person/day (based on rats and male mice) may be calculated as dosage without systemic effects. The dose of 150 mg/person/day is used for the formal calculation of the direct MOS. For the calculation of an adjusted MOS route-specific differences in systemic availability should be considered due to differences in absorption. For a hydrophilic vapour like acrylic acid a nearly quantitative absorption (100%) via inhalation is assumed. In Section 4.1.2.1 dermal absorption rates of 12-26% are described. The highest value of about 25% is used for a calculation of route-specific
differences. A further duration adjustment is considered to be not necessary (see under Inhalation (systemic)). For the calculation of an adjusted MOS a dermal NAEL (human, chronic) of about 600 (150·4) mg/person/day is assumed.

For repeated oral administration a lowest NOAEL of 40 mg/kg/day (male rats) is derived from a drinking water study. Obviously the bad palatability led to a lower drinking water intake, which was considered to result in lower food consumption and body weight gain. Other effects, observed at higher doses of the drinking water studies, were not indicative of a clear target organ and might be partly based on the above-mentioned effects (for details see Section 4.1.2.6).

Additionally a gavage study was available, but the effects observed in that study were attributed to high peak concentrations. An oral exposure schedule, which does not result in high peak concentrations, (that is the drinking water study) is considered more appropriate for the dermal risk assessment. The formal NOAEL (40 mg/kg/day) is to be used for a calculation of the direct MOS and as a starting point for extrapolation, but a more relevant dose might be higher (perhaps 3-fold; range of 150 mg/kg/day). Metabolic rate scaling requires an extrapolation factor of 1/4 resulting in an anticipated human oral NAEL of 10 mg/kg/day (700 mg/person/day). For a route-to-route extrapolation specific differences in systemic availability should be considered due to differences in absorption. According to Section 4.1.2.1, the absorption after oral administration should be nearly quantitative. Dermal absorption is considered to be about 25%. A specific duration adjustment is not applied for acrylic acid. Based on the oral study a dermal NAEL (human, chronic) of 2,800 (700·4) mg/person/day is estimated.

The NOAEC/NOAEL of both the inhalation and the drinking water study have their limitations as starting point for an assessment of eventual systemic effects after dermal application. The formal NOAEC of the inhalation study (5 ppm) is derived from female mice, the NOAECs for male mice and rats were clearly higher. In addition it is not excluded, that the observed effect (lower body weight gain) is secondary to the predominant local effect. The formal NOAEL of the oral study (40 mg/kg/day) is based on effects that were related to the bad palatability and should not be relevant for the dermal route. More relevant doses might be about 3-fold higher, but a clear systemic target organ toxicity is not described in the drinking water studies. The toxicity profile of acrylic acid (oral and inhalation) is clearly influenced by the route of administration and the main weakness of both studies is the debatable appropriateness of the observed dose-response relationship to be extrapolated to the dermal route. A strong scientific argument to prefer the oral or inhalation study as a starting point is not available and erring possibly on the side of caution the inhalation study resulting in a lower NAEL is selected for the dermal risk assessment.

The NAEL used for the calculation of the direct MOS is 150 mg/p/d, for the adjusted MOS: > 600 mg/p/d.

For scenarios with repeated daily dermal exposures the highest exposure level is calculated to be 1-10.5 mg/p/d (industrial area: use of adhesives with <5% acrylic acid). The calculated MOS values are 14-150 (direct MOS), resp.>57-600 (adjusted MOS). Details see Table 4.7. Therefore systemic health risks by chronic dermal exposure are not considered of concern.

**Conclusion (ii)**  There is at present no need for further information or testing or risk reduction measures beyond those which are being applied already.
Combined exposure (systemic effects)

Systemic health effects due to combined exposure (inhalation and dermal contact) are to be assessed in addition to route-specific risk estimates.

The MOS values for combined exposure are calculated by the formula:

\[
\frac{1}{MOS_{comb.}} = \frac{1}{MOS_{inh.}} + \frac{1}{MOS_{derm.}}
\]

One scenario with daily repeated combined inhalation and dermal exposure is found in the industrial area.

The results of the calculations for combined exposure are presented for “Use of adhesives” as an example in Table 4.8.

Table 4.8 Combined exposure (repeated dose toxicity, systemic)

<table>
<thead>
<tr>
<th>Exposure scenario</th>
<th>MOS(_{inhalation}) *</th>
<th>Conclusion</th>
<th>MOS(_{dermal}) *</th>
<th>Conclusion</th>
<th>MOS(_{combined})</th>
<th>Conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Industrial area: use of adhesives</td>
<td>2 with LEV</td>
<td>ii</td>
<td>57</td>
<td>ii</td>
<td>1.9</td>
<td>ii</td>
</tr>
<tr>
<td>(&lt; 5% acrylic acid)</td>
<td>0.5 without LEV</td>
<td>iii</td>
<td>57</td>
<td>ii</td>
<td>0.5</td>
<td>iii</td>
</tr>
</tbody>
</table>

* Lowest MOS values of ranges are used

The MOS for combined exposure is mainly determined by the MOS for inhalation exposure. The conclusions are identical with those for isolated inhalation exposures. The conclusion for the scenario “use of adhesives (< 5% AA), ind. area, without LEV” is conclusion (iii). Thus no relevant additional risk by combined exposure is expected. This conclusion is also applicable to other scenarios where conclusion (iii) has been applied for repeated dose toxicity, systemic (see Table 4.9).

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

4.1.3.2.5 Mutagenicity

With reference to Section 4.1.2.7, acrylic acid is unlikely to be mutagenic in vivo. Corresponding risks at workplaces are not anticipated to occur.

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

4.1.3.2.6 Carcinogenicity

There is no evidence that acrylic acid administered orally to rats or applied dermally to mice is carcinogenic. Human data on carcinogenicity is not available. In conclusion, acrylic acid is not
suspected to be a carcinogenic agent. Based on these data carcinogenic effects are not anticipated to occur.

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

### 4.1.3.2.7 Toxicity for reproduction

Based on experimental data, acrylic acid is not considered to be a reproductive toxicant (fertility impairment and developmental toxicity). The oral rat NOAEL for reproductive function is 460 mg/kg/day. The rat NOAEC for developmental toxicity is reported to be 360 ppm (1,060 mg/m$^3$). Comparison of these data on reproductive toxicity with threshold levels for general local and systemic toxicity (anticipated NAEC for respiratory tract irritation slightly below 5 ppm; oral NOAEL of 40 mg/kg/d for systemic effects in the rat drinking water study) shows that toxicity for reproduction is not anticipated to occur.

Corresponding occupational risks are not anticipated to occur.

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

### 4.1.3.2.8 Conclusions of the occupational risk assessment

The conclusions of the occupational risk assessment are summarised in Table 4.9.
### Table 4.9  Conclusions of the occupational risk assessment of acrylic acid

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<tr>
<td>Production and further processing</td>
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<td>i</td>
<td>iii</td>
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<td>Manufacture of adhesives (1-10% acrylic acid)</td>
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<td>&lt; 5% acrylic acid (not labelled as corrosive)</td>
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<td>Decomposition during production of integrated circuits</td>
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<td><strong>Skilled trade</strong></td>
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<td>Use of adhesives ≥ 5 - 10% acrylic acid (labelled as corrosive)</td>
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<td>≤ 5% acrylic acid (not labelled as corrosive)</td>
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4.1.3.3 Consumers

4.1.3.3.1 Acute Toxicity

Following the exposure assessment, consumers are not expected to be exposed to acrylic acid in the range of doses which can be derived from acute oral or dermal toxicity figures based on animal LD$_{50}$ values (oral: 140 up to 1,400 mg/kg bw and dermal: 300-640 mg/kg bw). Therefore the substance is of no concern in relation to acute oral or dermal toxicity.

Consumer exposure may occur as a result of the inhalation exposure to adhesives (up to peak concentrations of 0.542 µg/l as calculated applying the SCIES model). Following inhalation exposure in rats, acrylic acid has demonstrated a LC$_{50}$ value of 3,600 µg/l/4h.

Acute toxicity is dominated by chemical interactions with water and/or biological material. Acrylic acid causes acute harmful effects by the oral, inhalation and dermal routes of exposure. Concerning an acute inhalation exposure scenario, the margin of safety is assumed to be sufficient.

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

4.1.3.3.2 Irritation/Corrosivity

Acrylic acid is known to cause skin corrosion and irritation of the respiratory tract in humans. The substance causes severe burns to skin and eyes of animals. Necrosis to rabbit skin is already caused by a 50% aqueous substance solution after 1 minute of exposure.

According to the severe local corrosive properties acrylic acid is classified as corrosive, C and labelled with the R phrase R 35, causes severe burns. Provided the safety advice in accordance with the classification is followed current risk reduction measures are considered sufficient.

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

4.1.3.3.3 Sensitisation

Pure acrylic acid does not show skin sensitising properties. The sensitising properties of previously available acrylic acid samples (commercial grade) was attributed to an impurity ($\alpha,\beta$-diacryloxypropionic acid). This impurity could not be detected in current commercial acrylic acid.

There is no information available on the potential of acrylic acid to produce respiratory sensitization in animals. However, respiratory sensitization has not been observed in humans.

**Conclusion (ii)** There is at present no need for further information or testing or risk reduction measures beyond those which are being applied already.
4.1.3.3.4 Repeated dose toxicity

Following the exposure assessment consumers may be exposed to an average concentration of 0.384 mg/m$^3$ (1 hour) with a possible peak value of 0.542 mg/m$^3$ using UV-hardening adhesives. This exposure does not reflect a realistic chronic exposure scenario. However, during use of nappy pants babies and smaller children may be exposed dermally to acrylic acid. This exposure results from the residual content of monomers in homopolymers of acrylic acid which are used as “superabsorbents” in such products.

Studies in experimental animals have shown that the toxicity of acrylic acid is dominated by its local irritation effects irrespective of the manner of application. Prolonged inhalation (14 and 90 days) of concentrations from 5 ppm or higher in mice (15 mg/m$^3$) and from 75 ppm or higher in rats (221 mg/m$^3$) induced degeneration of the olfactory mucosa. A NOAEC for local effects of 25 ppm (resp. 74 mg/m$^3$) was derived from the 90-day study on rats, whereas no data on NOAEL are available in mice. In this species a LOAEC of 5 ppm (15 mg/m$^3$) could be derived. Acrylic acid causes severe mucosal damage to the stomach after repeated gavage administration of >150 mg/kg bw/d, but not after application via drinking water at similar or higher doses (NOAEL 40 mg/kg bw/d (male rats), resp. 83 mg/kg bw/d (female rats)). Long-term dermal exposure of acrylic acid at concentrations of >1% resulted in skin irritation whereas no skin effect was evident at 1% (cf. dermal carcinogenicity studies in Section 4.1.2.8).

Following oral, dermal or inhalation administrations no other systemic toxic effects were detected except premature deaths and tubular degeneration/necrosis in the kidneys which were evident after gavage administration of dosages >150 mg/kg bw/d in a rat 3-month study. Effects did not occur in drinking water studies at similar or higher doses. After prolonged inhalation exposure a NOAEC for systemic effects of 5 ppm (15 mg/m$^3$) was derived for female mice and of 75 ppm (221 mg/m$^3$) for male mice as well as female and rats.

For the decision on the appropriateness of MOS, the following aspects regarding the critical effect as well as exposure have been considered and taken into account:

Overall confidence in the database

The data taken into account for performing the risk characterisation have been evaluated with regard to their reliability, relevance and completeness according to Section 3.2 of the TGD. The data were published in peer-reviewed journals or submitted to the Competent Authority in private reports being adequately detailed and in accordance with internationally recognised guidelines and to GLP.

The findings of all studies are not contradictory so that the judgement can be based on the database.

There are no reasons to assume limited confidence.

Uncertainty arising from the variability in the experimental data

The studies cited above allow a conclusion on the NOAEC/LOAEC of severe health effects during inhalation (degenerative lesions of the olfactory mucosa) from four studies. There is only one valid study to derive a NOAEC for local irritation on rats. Comparing the local effect concentrations, mice seem to be more sensitive than rats without sex preference. Thus, a LOAEC was derived from one single mice study, which was well performed and the results were in conformity with the findings of the other studies.
Three oral 90-day studies on rats allow the derivation of a NOAEL for systemic effects. The range varied from 40 mg/kg bw/d to 331 mg/kg bw/d. The NOAEL of 40 mg/kg bw/d (decreased body weight gain) was derived from the drinking water study (BASF, 1987) which was well performed and the results were in conformity with the findings of the other studies.

There are no reasons to assume a special extent of uncertainty which have to be taken into account.

**Intra- and interspecies variation**

Hitherto available data on kinetics of acrylic acid do not allow to calculate the intraspecies and interspecies variability by applying modern approaches. However, the present data give no hint on a particular high variability in kinetics. The variability of the data on the toxicodynamics has been described above and seems not to justify an increased MOS.

**Nature and severity of the effect**

The main effect considered as “critical effect” is degeneration of the olfactory mucosa (irreversible, serious health effect).

There are no reasons to assume that the effects shown in the animal experiments are limited to the species tested, thus being of no relevance for humans. Because of the seriousness of the effect there is concern, which has to be expressed in the magnitude of the MOS.

**Dose response relationship**

In rats as well as mice no steep dose-response relationship is observed for the irritation effects at the olfactorium. Due to the fact that the LOAEC from the most sensitive species is used for MOS considerations we are of the opinion that no further reasons are given for requiring a higher MOS.

**Differences in exposure (route, duration, frequency and pattern)**

**Inhalation and oral route**

Following the exposure assessment, the consumer may be exposed to acrylic acid via inhalation, oral exposure can be neglected. The described human exposure scenario (usage of an adhesive) does not represent a real chronic scenario. The LOAEC used for the discussion of the MOS regarding this application is derived from a 90-day inhalation study in mice. Because acrylic acid acts directly and locally at the nasal cavity, systemic effects have not to be considered.

**Dermal route**

The estimated dermal body burden with an assumed absorption of 100% is compared with an oral NOAEL from a 90-day study.

There are no reasons to assume that special concern can be derived neither from this procedure nor from the available toxicokinetic information; concerning different routes inasmuch as absorption was set at 100%.
Human population to which the quantitative and/or qualitative information on exposure applies

Following the inhalation exposure there is no reason to assume a special risk for elderly, children or other people suffering from special diseases like obesity or persons with high bronchial reactivity.

Regarding the dermal exposure special concern has to be directed to infants, in particular premature babies.

Other factors

There are no other factors known requiring a peculiar margin of safety.

MOS for inhalation exposure scenario

During application of an UV-hardening adhesive for one hour (4 times per year) the consumer may be exposed to an average concentration of 0.384 mg/m³ with a possible peak value of 0.542 mg/m³. This exposure does not reflect a realistic chronic exposure scenario. Therefore, the margin of safety between the estimated exposure level of 0.384 mg/m³ and the LOAEC for local irritation effects of 15 mg/m³ is about 40 which is judged to be sufficient because a worst-case exposure scenario and a LOAEC for mice as more sensitive species are taken into consideration.

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

MOS for dermal exposure scenario

The calculation of the dermal exposure of babies due to nappies leads to an internal exposure of 0.00018 mg/kg bw/d (uptake basis assuming the bioavailability via the dermal route is 100%). The margin of safety between the estimated exposure level of 0.00018 mg/kg bw/d and the oral NOAEL of 40 mg/kg bw/d is about 200,000 which is judged to be sufficient, even if the special considerations on premature babies as population at risk and route-to-route extrapolation are taken into consideration.

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

MOS for oral exposure scenario

The oral uptake is negligible.

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

Acrylic acid is non-mutagenic in Salmonella and CHO cells (HPRT locus) but clearly mutagenic in the mouse lymphoma assay and in the in vitro chromosomal aberration test. However, in vivo acrylic acid did not produce mutagenic effects in either rat bone marrow cells or mouse germ cells after oral administration. Based on the present results and taken into account data on structurally-related acrylic compounds, it is unlikely that acrylic acid is mutagenic in vivo.

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

4.1.3.3.6 Carcinogenicity

Studies on experimental animals indicate that acrylic acid is not carcinogenic in animals. There are no cancer data available with respect to human exposure.

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

4.1.3.3.7 Toxicity for reproduction

In oral reproductive toxicity studies (rats), no effects on reproductive function (fertility) were observed, but some signs of postnatal developmental toxicity (retarded body weight gain of the pups) were seen following exposure of the parental generation. No gross abnormalities were observed in the offsprings. No prenatal developmental toxicity was observed (rats and rabbits, inhalation).

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

4.1.3.4 Humans exposed via the environment

Indirect exposure via the environment is estimated using both a regional and a point source model.

The main route of exposure is via the drinking water for the regional exposures and stem/air for the point source approach.

Following these data, a total daily dose of 50 µg/kg bw/d is calculated for the local scenario and of 15.1 ng/kg bw/d for the regional one. In a repeated dose toxicity study (male rats, oral, 90-day study) the NOAEL was 40 mg/kg bw/d.

Comparison indirect exposure - Local scenario/NOAEL

\[
\frac{\text{Indirect exposure}}{\text{NOAEL}} = \frac{40 \text{ mg/kg bw/d}}{0.050 \text{ mg/kg bw/d}} = 800
\]

87
The margin of safety for systemic effects between the calculated local exposure and the NOAEL is considered to be sufficient. Thus, the substance is of no concern in relation to indirect exposure via the environment.

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

**Comparison indirect exposure - Regional scenario/NOAEL**

\[
\text{Indirect exposure} \approx \frac{\text{40 mg/kg bw/d}}{0.00015 \text{mg/kg bw/d}} = 2 \cdot 10^6
\]

On the basis of these data the margin of safety for the regional scenario is considered to be sufficient. The substance is of no concern in relation to indirect exposure via the environment.

**Conclusion (ii)** There is at present no need for further information or testing or risk reduction measures beyond those which are being applied already.
4.2 HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

4.2.1 Exposure assessment

4.2.1.1 Occupational exposure

See Section 4.1.1.2.

4.2.2 Effects assessment

4.2.2.1 Explosivity

Acrylic acid is not explosive.

4.2.2.2 Flammability

Acrylic acid is flammable.

4.2.2.3 Oxidising potential

In view of its chemical structure, acrylic acid is not expected to have an oxidising potential.

4.2.3 Risk characterisation

4.2.3.1 Workers

Acrylic acid is flammable. If it is heated above its flash point, an explosive atmosphere may be formed (lower explosion limit: 2.4% (vol.); upper explosion limit: 15.9% (vol.) according to manufacturer). In order to exclude any possible hazard to workers, the national regulations on handling flammable liquids and on the prevention of explosions must be observed.

Adequate worker protection measures must be observed. Risk reduction measures beyond those which are being applied already are not considered necessary.

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

5.1 ENVIRONMENT

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

Acrylic acid (AA) presents, based on the present data, a risk to the environment around point sources. A potential risk to municipal wastewater treatment plants is identified for the downstream use scenarios of super absorber polymers (SAP) production (based on default calculation and highest site-specific PEC_{wwtp}) and wet polymerisation (based on default calculation and known sites L, Q).

Since the PNEC_{microorganisms} is derived from single species tests with ciliated protozoa, there is a need for further data reflecting the integrity of the native ciliate population in sewage sludge as a whole. However, since risk reduction measures are necessary to remove concern for surface water (see below), these measures will also cover the protection of municipal wastewater treatment plants, and additional testing is not required.

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

This conclusion applies to effects on sediment, atmosphere, soil, and secondary poisoning. Conclusion (ii) applies also to the aquatic compartment regarding all production sites, the processing scenario (dry polymerisation), and the relevant use scenarios (leather finishing, textile finishing, formulation of paints and application of water treatment agents).

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

Acrylic acid (AA) presents, based on the present data, a risk to the environment around point sources.

A potential risk to the local aquatic environment is identified from wet polymerisation processes including wet production of SAP (super absorber polymers) by downstream users of monomeric AA (based on default calculations and known sites N, O, Q).

Although an improvement of the data (i.e. effluent measurements and/or site specific data on flow rates) may in principle be possible, it is judged to be unlikely that sufficiently complete representative monitoring data from the downstream users can be obtained with reasonable expenditure of time and money. For certain known SAP production sites and wet polymerisation sites, regular effluent concentrations up to 100 mg/l AA and significantly more have been reported. These data indicate that high effluent concentrations cannot be excluded, even if certain types of process engineering are applied. On the other hand, application of wastewater reutilization / recycling systems is known to result in zero emissions to the hydrosphere at a number of downstream user sites, processing about 50% of AA used externally for SAP production and about 12% of AA used externally in wet polymerisation processes. For sites applying this kind of technique, no further risk reduction measures are deemed necessary.
Measures applied for limiting the risk to the local aquatic environment are presumed to be also protective for municipal wastewater treatment plants.

During the use of a grouting agent containing magnesium diacrylate high concentrations of AA are released via the drainage water. The exposure assessment was based on measured effluent concentrations at a tunnel construction site. A quantitative extrapolation to other construction sites seems difficult, but similar conditions might be anticipated. Measures appropriate to local circumstances should be applied.

5.2 HUMAN HEALTH

5.2.1 Human health (toxicity)

5.2.1.1 Workers

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

This conclusion is reached because of:

- concerns for respiratory tract irritation and corrosivity as a consequence of single inhalation exposure arising from production and processing, production of adhesives containing the substance and use of adhesives containing the substance (industrial area and skilled trade),
- concerns for local effects as a consequence of repeated inhalation exposure arising from production and use of adhesives containing the substance,
- concerns for general systemic toxicity as a consequence of repeated inhalation exposure arising from production and use of adhesives containing the substance.

5.2.1.2 Consumers

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

5.2.1.3 Humans exposed via the environment

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

5.2.2 Human health (risks from physico-chemical properties)

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


Arbejdstilsynet (1996). Grænseværdier for stoffer og materialer. At-anvisning Nr.3.1.0.2. København Ø.


Black KA et al. (1994). The Toxicologist 14, 430.


Elf Atochem (1998). α-β-Diacycloxypropionic acid (DAPA) – Confirmation of absence of DAPA in commercial acrylic acid (complementary study), Carling, 08.06.1998.


Frantz SW et al. (1994). The Toxicologist, 14, 430.


Lomax LG, Brown DW, Frederick CB (1994). Regional histopathology of the mouse nasal cavity following two weeks of exposure to acrylic acid for either 6 or 22 hours per day. The Toxicologist 14, 312.


Miller RR, Ayres JA, Jersey GC, McKenna MJ (1981b). Results of a 90-day acrylic acid vapor inhalation study with rats and mice. The Toxicologist 1, 83.


Nyholm N (1999). Algal toxicity test reports on acrylic acid (AA), methacrylic acid (MAA), and methyl methacrylate (MMA) - a critical evaluation of selected test reports. Study contracted by the Danish EPA, Study report dated 27-02-1999, 7 pp.


ABBREVIATIONS

ADI  Acceptable Daily Intake
AF   Assessment Factor
ASTM American Society for Testing and Materials
ATP  Adaptation to Technical Progress
AUC  Area Under The Curve
B    Bioaccumulation
BBA  Biologische Bundesanstalt für Land- und Forstwirtschaft
BCF  Bioconcentration Factor
BMC  Benchmark Concentration
BMD  Benchmark Dose
BMF  Biomagnification Factor
BOD  Biochemical Oxygen Demand
bw   body weight / Bw, bw
C    Corrosive (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
CA   Chromosome Aberration
CA   Competent Authority
CAS  Chemical Abstract Services
CEC  Commission of the European Communities
CEN  European Standards Organisation / European Committee for Normalisation
CEPE European Committee for Paints and Inks
CMR  Carcinogenic, Mutagenic and toxic to Reproduction
CNS  Central Nervous System
COD  Chemical Oxygen Demand
CSTEE Scientific Committee for Toxicity, Ecotoxicity and the Environment (DG SANCO)
CT_{50} Clearance Time, elimination or depuration expressed as half-life
d.wt dry weight / dw
dfi  daily food intake
DG   Directorate General
DIN  Deutsche Industrie Norm (German norm)
DNA  DeoxyriboNucleic Acid
DOC  Dissolved Organic Carbon
DT_{50} Degradation half-life or period required for 50 percent dissipation / degradation
DT_{90} Period required for 90 percent dissipation / degradation
E    Explosive (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>EASE</td>
<td>Estimation and Assessment of Substance Exposure Physico-chemical properties [Model]</td>
</tr>
<tr>
<td>EbC50</td>
<td>Effect Concentration measured as 50% reduction in biomass growth in algae tests</td>
</tr>
<tr>
<td>EC</td>
<td>European Communities</td>
</tr>
<tr>
<td>EC10</td>
<td>Effect Concentration measured as 10% effect</td>
</tr>
<tr>
<td>EC50</td>
<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>
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<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>
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<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>
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<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>foc</td>
<td>Organic carbon factor (compartment depending)</td>
</tr>
<tr>
<td>GLP</td>
<td>Good Laboratory Practice</td>
</tr>
<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>
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<tr>
<td>HPLC</td>
<td>High Pressure Liquid Chromatography</td>
</tr>
<tr>
<td>HPVC</td>
<td>High Production Volume Chemical (&gt; 1000 t/a)</td>
</tr>
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<td>IARC</td>
<td>International Agency for Research on Cancer</td>
</tr>
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<td>IC</td>
<td>Industrial Category</td>
</tr>
<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>
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<tr>
<td>ISO</td>
<td>International Organisation for Standardisation</td>
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<td>IUCLID</td>
<td>International Uniform Chemical Information Database (existing substances)</td>
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<td>IUPAC</td>
<td>International Union for Pure and Applied Chemistry</td>
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<td>JECFA</td>
<td>Joint FAO/WHO Expert Committee on Food Additives</td>
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<tr>
<td>Acronym</td>
<td>Definition</td>
</tr>
<tr>
<td>---------</td>
<td>------------</td>
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<td>JMPR</td>
<td>Joint FAO/WHO Meeting on Pesticide Residues</td>
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<td>Koc</td>
<td>organic carbon normalised distribution coefficient</td>
</tr>
<tr>
<td>Kow</td>
<td>octanol/water partition coefficient</td>
</tr>
<tr>
<td>Kp</td>
<td>solids-water partition coefficient</td>
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<tr>
<td>L(E)C50</td>
<td>median Lethal (Effect) Concentration</td>
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<tr>
<td>LAEL</td>
<td>Lowest Adverse Effect Level</td>
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<tr>
<td>LC50</td>
<td>median Lethal Concentration</td>
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<tr>
<td>LD50</td>
<td>median Lethal Dose</td>
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<tr>
<td>LEV</td>
<td>Local Exhaust Ventilation</td>
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<tr>
<td>LLNA</td>
<td>Local Lymph Node Assay</td>
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<td>LOAEL</td>
<td>Lowest Observed Adverse Effect Level</td>
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<tr>
<td>LOEC</td>
<td>Lowest Observed Effect Concentration</td>
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<td>LOED</td>
<td>Lowest Observed Effect Dose</td>
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<tr>
<td>LOEL</td>
<td>Lowest Observed Effect Level</td>
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<td>MAC</td>
<td>Maximum Allowable Concentration</td>
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<tr>
<td>MATC</td>
<td>Maximum Acceptable Toxic Concentration</td>
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<td>MC</td>
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<td>MITI</td>
<td>Ministry of International Trade and Industry, Japan</td>
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<tr>
<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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<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>
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<tr>
<td>NAEL</td>
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<td>NOAEL</td>
<td>No Observed Adverse Effect Level</td>
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<td>NOEL</td>
<td>No Observed Effect Level</td>
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<td>NOEC</td>
<td>No Observed Effect Concentration</td>
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<td>NTP</td>
<td>National Toxicology Program (USA)</td>
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<td>O</td>
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<td>OECD</td>
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<td>OEL</td>
<td>Occupational Exposure Limit</td>
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<td>OJ</td>
<td>Official Journal</td>
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<td>OSPAR</td>
<td>Oslo and Paris Convention for the protection of the marine environment of the Northeast Atlantic</td>
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<td>P</td>
<td>Persistent</td>
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<td>PBT</td>
<td>Persistent, Bioaccumulative and Toxic</td>
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<td>Description</td>
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<td>--------------</td>
<td>-------------</td>
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<tr>
<td>PBPK</td>
<td>Physiologically Based PharmacoKinetic modelling</td>
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<tr>
<td>PBTK</td>
<td>Physiologically Based ToxicoKinetic modelling</td>
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<td>PEC</td>
<td>Predicted Environmental Concentration</td>
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<td>pH</td>
<td>logarithm (to the base 10) (of the hydrogen ion concentration ( \text{H}^+ ))</td>
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<tr>
<td>pKa</td>
<td>logarithm (to the base 10) of the acid dissociation constant</td>
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<tr>
<td>pKb</td>
<td>logarithm (to the base 10) of the base dissociation constant</td>
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<td>Predicted No Effect Concentration</td>
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<td>Persistent Organic Pollutant</td>
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<td>Personal Protective Equipment</td>
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<td>(Quantitative) Structure-Activity Relationship</td>
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<td>Risk phrases according to Annex III of Directive 67/548/EEC</td>
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<td>Risk Assessment Report</td>
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<td>RC</td>
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<td>Reference Concentration</td>
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<td>RfD</td>
<td>Reference Dose</td>
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<td>RNA</td>
<td>RiboNucleic Acid</td>
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<td>RPE</td>
<td>Respiratory Protective Equipment</td>
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<td>RWC</td>
<td>Reasonable Worst Case</td>
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<td>S phrases</td>
<td>Safety phrases according to Annex III of Directive 67/548/EEC</td>
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<tr>
<td>SAR</td>
<td>Structure-Activity Relationships</td>
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<td>SBR</td>
<td>Standardised birth ratio</td>
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<td>SCE</td>
<td>Sister Chromatic Exchange</td>
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<td>SDS</td>
<td>Safety Data Sheet</td>
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<td>Society of Environmental Toxicology And Chemistry</td>
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<td>Summary Notification Interchange Format (new substances)</td>
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<td>Species Sensitivity Distribution</td>
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<td>Sewage Treatment Plant</td>
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<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>
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<td>Tolerable Daily Intake</td>
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<td>Test Guideline</td>
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<td>Technical Guidance Document</td>
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<td>TNsG</td>
<td>Technical Notes for Guidance (for Biocides)</td>
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<td>TNO</td>
<td>The Netherlands Organisation for Applied Scientific Research</td>
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<td>ThOD</td>
<td>Theoritical Oxygen Demand</td>
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<td>UC</td>
<td>Use Category</td>
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<td>UDS</td>
<td>Unscheduled DNA Synthesis</td>
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<td>UN</td>
<td>United Nations</td>
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<td>Acronym</td>
<td>Description</td>
</tr>
<tr>
<td>---------</td>
<td>-------------</td>
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<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>
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<tr>
<td>UVCB</td>
<td>Unknown or Variable composition, Complex reaction products of Biological material</td>
</tr>
<tr>
<td>vB</td>
<td>very Bioaccumulative</td>
</tr>
<tr>
<td>VOC</td>
<td>Volatile Organic Compound</td>
</tr>
<tr>
<td>vP</td>
<td>very Persistent</td>
</tr>
<tr>
<td>vPvB</td>
<td>very Persistent and very Bioaccumulative</td>
</tr>
<tr>
<td>v/v</td>
<td>volume per volume ratio</td>
</tr>
<tr>
<td>w/w</td>
<td>weight per weight ratio</td>
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<tr>
<td>WHO</td>
<td>World Health Organization</td>
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<td>WWTP</td>
<td>Waste Water Treatment Plant</td>
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<tr>
<td>Xn</td>
<td>Harmful (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)</td>
</tr>
<tr>
<td>Xi</td>
<td>Irritant (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)</td>
</tr>
</tbody>
</table>
Appendix A1  Distribution and fate

Substance:  Acrylic Acid

- Melting point:  $MP = 286 \, K$
- Vapour pressure:  $VP = 380 \, Pa$
- Water solubility:  $SOL = 1000000 \, mg \cdot l^{-1}$
- Part. coefficient octanol/water:  $LOGP_{OW} = 0.46$
- Molecular weight:  $MOLW = 0.07206 \, kg \cdot mol^{-1}$
- Gas constant:  $R = 8.3143 \, J \cdot mol \cdot K^{-1}$
- Temperature:  $T = 293 \, K$
- Concentration of suspended matter in the river:  $SUSP_{water} = 15 \, mg \cdot l^{-1}$
- Density of the solid phase:  $RHO_{solid} = 2500 \, kg \cdot m^{-3}$
- Volume fraction water in susp. matter:  $F_{water\, susp} = 0.9$
- Volume fraction solids in susp. matter:  $F_{solid\, susp} = 0.1$
- Volume fraction of water in sediment:  $F_{water\, sed} = 0.8$
- Volume fraction of solids in sediment:  $F_{solid\, sed} = 0.2$
- Volume fraction of air in soil:  $F_{air\, soil} = 0.2$
- Volume fraction of water in soil:  $F_{water\, soil} = 0.2$
- Volume fraction of solids in soil:  $F_{solid\, soil} = 0.6$
- Aerobic fraction of the sediment comp.:  $F_{eaer\, sed} = 0.1$
- Product of $CONjunge$ and $SURFair$:  $K_{air\_water} = 10^{-5} \, Pa$

**Distribution air/water: Henry-constant**

$$HENRY = \frac{VP \cdot MOLW}{SOL}$$

$$\log \left( \frac{HENRY}{Pa \cdot m^{3} \cdot mol^{-1}} \right) = -1.563$$

$$K_{air\_water} = \frac{HENRY}{RT}$$

$K_{air\_water} = 1.12 \times 10^{-5}$
**solid/water-partition coefficient** $K_{p_{comp}}$ and **total compartment/water-partition coefficient** $K_{comp_{water}}$

\[ a \approx 0.52 \quad \text{(a,b from TGD, p. 539)} \]

\[ b \approx 1.02 \]

\[ K_{OC} = 10^{-a \cdot \text{LOGP}_{OW} + b} \cdot \text{kg}^{-1} \]

\[ K_{OC} = 18.16 \cdot \text{kg}^{-1} \]

**Suspended matter**

\[ K_{p_{susp}} := 1 \cdot \text{kg}^{-1} \]

\[ K_{susp_{water}} := F_{water_{susp}} + F_{solid_{susp}} \cdot K_{p_{susp}} \cdot RH_{solid} \quad K_{susp_{water}} = 1.15 \]

**factor for the calculation of** $C_{local_{water}}$:

\[ \text{factor} := 1 + K_{p_{susp}} \cdot SUSP_{water} \quad \text{factor} = 1 \]

**Sediment**

\[ K_{p_{sed}} := 1 \cdot \text{kg}^{-1} \]

\[ K_{sed_{water}} := F_{water_{sed}} + F_{solid_{sed}} \cdot K_{p_{sed}} \cdot RH_{solid} \quad K_{sed_{water}} = 1.3 \]

**Sludge**

\[ K_{p_{sludge}} := 1 \cdot \text{kg}^{-1} \]

**Soil**

\[ K_{p_{soil}} := 1 \cdot \text{kg}^{-1} \]

\[ K_{soil_{water}} := F_{air_{soil}} \cdot K_{air_{water}} + F_{water_{soil}} + F_{solid_{soil}} \cdot K_{p_{soil}} \cdot RH_{solid} \]

\[ K_{soil_{water}} = 1.7 \]
Elimination in STPs

rate constant in STP: \( k = 1 \text{ d}^{-1} \)

elimination \( P = f( k, \text{logpow}, \text{logH}) = 87.3 \)

fraction directed to surface water \( F_{\text{stpw}} = 12.7 \)

biodegradation in different compartments

**surface water**

\( k_{\text{bio water}} := 4.7 \times 10^{-2} \text{.d}^{-1} \) (cTGD, table 5)

**soil**

\( \text{DT}_{50\text{bio soil}} := 15 \text{d} \) (see RAR)

\( k_{\text{bio soil}} := \frac{\ln(2)}{\text{DT}_{50\text{bio soil}}} \approx 0.047 \text{d}^{-1} \)

**sediment**

\( k_{\text{bio sed}} := \frac{\ln(2)}{\text{DT}_{50\text{bio soil}}} \cdot F_{\text{aer sed}} \approx 0.005 \text{d}^{-1} \)

**degradation in surface waters**

\( k_{\text{hydr water}} := 1 \times 10^{-10} \text{.d}^{-1} \)

\( k_{\text{photo water}} := 1 \times 10^{-10} \text{.d}^{-1} \)

\( k_{\text{deg water}} := k_{\text{hydr water}} + k_{\text{photo water}} + k_{\text{bio water}} \)

\( k_{\text{deg water}} = 0.047 \text{d}^{-1} \)

**Atmosphere**

**calculation of CONjunge * SURFaer for the OPS-model**

\( \text{VPL} := \frac{VP}{\exp\left[6.79 \left(1 - \frac{\text{MP}}{285 \text{K}}\right)\right]} \quad \text{VP} := \text{wenn( MP > 285 K, VPL, VP)} \quad \text{VP} = 389.162 \text{Pa} \)

\( F_{\text{aer}} := \frac{\text{product}}{\text{VP} + \text{product}} \quad F_{\text{aer}} = 2.57 \times 10^{-7} \)

**degradation in the atmosphere**

\( k_{\text{deg air}} = 0.0175 \text{ h}^{-1} \) (see AOP-calculation)
Appendix A2  Calculation of $\text{PEC}_{\text{local}}$ for aquatic compartment during production and processing of chemicals at one site

Status: TGD, ESD, IC-3

**chemical: Acrylic Acid, generic scenario for production/processing**

Production volume: $T_1 := 330000 \text{t·a}^{-1}$

Processing volume: $T_2 := 330000 \text{t·a}^{-1}$

Emission factor for production (TGD, tab. A1.2): $f_1 := 0.3\%$

Emission factor for processing (TGD, tab. A3.3): $f_2 := 0.7\%$

Duration of emission for production (TGD, tab. B1.1): $T_{\text{emission 1}} := 300 \text{d·a}^{-1}$

Duration of emission for processing (TGD, tab. B3.2): $T_{\text{emission 2}} := 300 \text{d·a}^{-1}$

Fraction of emission directed to water: (SimpleTreat, $k: 1 \text{ h}^{-1}; \log H: -1.56; \log K_{ow}: 0.46$)

River flow rate (TGD): $V := 60 \text{ m}^3 \text{ s}^{-1}$

Factor $(1 + K_p \times \text{SUSPwater})$: $\text{FACTOR} := 1$

**Emission per day:**

\[
E_{\text{local water}} := \frac{T_1 f_1}{T_{\text{emission 1}}} + \frac{T_2 f_2}{T_{\text{emission 2}}} \quad \text{E}_{\text{local water}} = 1.1 \times 10^4 \text{ kg·d}^{-1}
\]

**Concentration in surface water:**

\[
C_{\text{local water}} := \frac{E_{\text{local water}} F_{\text{stp water}}}{V \times \text{FACTOR}} \quad C_{\text{local water}} = 269.5 \mu g\text{·L}^{-1}
\]
Appendix A3  Default exposure estimation of Clocal\textsubscript{water}

Status: TGD, Tables A and B

stage of life cycle: default processing (polymerisation), wet process
IC/UC/MC:11/33/III

Total annual tonnage of chemical: 

\[ \text{TONNAGE} := 10000 \text{t·a}^{-1} \]


\[ f_{\text{emission}} := 0.01 \]

Fraction of main source (B-table: B3.9): 

\[ F_{\text{main source}} := 1 \]

Waste water flow of wwtp: 

\[ \text{EFFLUENT}_{\text{stp}} := 2000 \text{m}^3 \cdot \text{d}^{-1} \]

Duration of emission (B-table: B3.9): 

\[ T_{\text{emission}} := 300 \text{d·a}^{-1} \]

Fraction of emission directed to water: 

\[ F_{\text{stp water}} := 12.7\% \]

Duration of emission (B-table: B3.9): 

\[ T_{\text{emission}} := 300 \text{d·a}^{-1} \]

Fraction of emission directed to water: 

\[ F_{\text{stp water}} := 12.7\% \]

Dilution factor (TGD): 

\[ \text{DILUTION} := 10 \]

Factor (1+Kp * SUSPwater): 

\[ \text{FACTOR} := 1 \]

\textbf{Emission per day:}

\[ \text{E}_{\text{local water}} := \frac{\text{TONNAGE} F_{\text{main source}} f_{\text{emission}}}{T_{\text{emission}}} \]

\[ \text{E}_{\text{local water}} = 333.33 \text{kg·d}^{-1} \]

\textbf{Influent concentration:}

\[ \text{C}_{\text{local inf}} := \frac{\text{E}_{\text{local water}}}{\text{EFFLUENT}_{\text{stp}}} \]

\[ \text{C}_{\text{local inf}} = 1.6 \times 10^6 \text{ µg·l}^{-1} \]

\textbf{Effluent concentration:}

\[ \text{C}_{\text{local eff}} := \text{C}_{\text{local inf}} F_{\text{stp water}} \]

\[ \text{C}_{\text{local eff}} = 2.12 \times 10^4 \text{ µg·l}^{-1} \]

\textbf{Concentration in surface water:}

\[ \text{C}_{\text{local water}} := \frac{\text{C}_{\text{local eff}}}{\text{FACTOR} \cdot \text{DILUTION}} \]

\[ \text{C}_{\text{local water}} = 2.12 \times 10^3 \text{ µg·l}^{-1} \]

\textbf{Annual average local concentration in water:}

\[ \text{C}_{\text{local water ann}} := \frac{T_{\text{emission}}}{365 \text{d·a}^{-1}} \]

\[ \text{C}_{\text{local water ann}} = 1.74 \times 10^3 \text{ µg·l}^{-1} \]
Appendix A4  Calculation of $\text{PEC}_{\text{local}}$ for the aquatic compartment - leather production industry

Status: TGD, ESD, IC-7

Mass of processed goods per day: $W_1 := 15 \, \text{t/d}$

Mass of $\text{AA}$-monomer used per mass of good: (see RAR) $W_2 := 0.2 \, \text{kg/t}$

Degree of 'fixation': $F := 95\%$

Participation factor on production per day $A := 100\%$

Waste water flow of wwtp: $\text{EFFLUENT}_{\text{stp}} := 2000 \, \text{m}^3\text{d}^{-1}$

Fraction of emission directed to water: $F_{\text{stp water}} := 12.7\%$

Dilution factor (TGD): $\text{DILUTION} := 10$

Factor $(1+K_p*\text{SUSPwater})$: $\text{FACTOR} := 1$

\section*{Emission per day:}

$E_{\text{local water}} := W_1 \cdot W_2 \cdot (1 - F) \cdot A$

$E_{\text{local water}} = 0.15 \, \text{kg/d}$

\section*{Influent concentration:}

$C_{\text{local inf}} := \frac{E_{\text{local water}}}{\text{EFFLUENT}_{\text{stp}}}$

$C_{\text{local inf}} = 0.07 \, \text{mg/L}$

\section*{Effluent concentration:}

$C_{\text{local eff}} := C_{\text{local inf}} \cdot F_{\text{stp water}}$

$C_{\text{local eff}} = 0.00952 \, \text{mg/L}$

\section*{Concentration in surface water:}

$C_{\text{local water}} := \frac{C_{\text{local eff}}}{\text{DILUTIONFACTOR}}$

$C_{\text{local water}} = 0.952 \, \text{µg/L}$

$C_{\text{local water}} = \text{PEC}_{\text{local}}$ for $\text{PEC}_{\text{regional}} = 0$
Appendix A5  Calculation of PEC_{local} for aquatic compartment for textile finishing industry

Status: TGD, ESD, IC-13

Mass of good processed per day: \( W_1 := 3 \cdot t \cdot d^{-1} \)  \( \mu g := 10^{-9} \cdot kg \)
Mass of substance used per mass of good: \( W_2 := 0.2 \cdot kg \cdot t^{-1} \)
Degree of fixation: \( F := 95 \% \)
Waste water flow of wwtp: \( \text{EFFLUENT}_{stp} := 2000 \cdot m^3 \cdot d^{-1} \)
Fraction of emission directed to water: \( F_{stp \ water} := 12.7 \% \)
Factor \((1+Kp*\text{SUSP}_{water})\): \( \text{FACTOR} := 1 \)
Dilution factor \((\text{TGD})\): \( \text{DILUTION} := 10 \)

Emission per day:
\[
E_{local \ water} := W_1 \cdot W_2 \cdot (1 - F)
\]
\[
E_{local \ water} = 0.03 \cdot kg \cdot d^{-1}
\]

Influent concentration:
\[
C_{local \ inf} := \frac{E_{local \ water}}{\text{EFFLUENT}_{stp}}
\]
\[
C_{local \ inf} = 0.015 \cdot mg \cdot l^{-1}
\]

Effluent concentration:
\[
C_{local \ eff} := C_{local \ inf} \cdot F_{stp \ water}
\]
\[
C_{local \ eff} = 0.002 \cdot mg \cdot l^{-1}
\]

Concentration in surface water:
\[
C_{local \ water} := C_{local \ eff} \cdot \text{DILUTION}^{-1} \cdot \text{FACTOR}^{-1}
\]
\[
C_{local \ water} = 0.19 \cdot \mu g \cdot l^{-1}
\]
Appendix A6  Default calculation of PEC_{\text{local}} for the hydrosphere

Status: TGD for Existing Substances / EUSES

AA

formulation of paints

tables: A2.1,  B2.3

tonnage: $T := 8 \cdot \text{tonne} \cdot \text{a}^{-1}$
release factor (A2.1): $r := 0.003$
fraction of main source (B2.3): $f := 1$
waste water flow of the WWTP: $Q := 2000 \cdot \text{m}^3 \cdot \text{d}^{-1}$
number of days for releases: $d := 300 \cdot \text{d} \cdot \text{a}^{-1}$

\[
C_{\text{inf}} = \frac{T \cdot r \cdot f}{Q \cdot d}
\]

Elimination in WWTP related to SIMPLETREAT:

$P := 87.3\% \quad P = f(\text{biodegradation, log pow, log H})$

\[
C_{\text{eff}} = C_{\text{inf}} (1 - P) \quad C_{\text{eff}} = 5.08 \cdot 10^{-3} \cdot \text{mg} \cdot \text{l}^{-1}
\]

Calculation of C-local:

partition coefficient for susp.matter: $K_{p_{\text{susp}}} := 1 \cdot \text{kg} \cdot \text{l}^{-1}$
concentration of suspended matter: $c_{\text{susp}} := 15 \cdot \text{mg} \cdot \text{l}^{-1}$
dilution factor for receiving surface water: $D := 10$

\[
C_{\text{local}} := \frac{C_{\text{eff}}}{(1 + K_{p_{\text{susp}}} \cdot c_{\text{susp}}) \cdot D}
\]

$C_{\text{local}} = 0.508 \cdot \mu \text{g} \cdot \text{l}^{-1}$
Appendix A7  Atmosphere (OPS model) - generic approach

Calculation of $C_{\text{local air}}$ and $\text{PEC}_{\text{local air}}$

(generic approach)

d := 24 h

concentration in air at source

strength of 1 kg/d.

$C_{\text{std air}} := 2.78 \times 10^{-4} \text{ mg m}^{-3} \text{ kg}^{-1} \text{ d}$

fraction of release at the local main source at life cycle stage:

$F_{\text{main source}} := 1$

number of days per year for the emission:

$T_{\text{emission}} := 300 \text{ d a}^{-1}$

release to air during life cycle stage:

RELEASE := 36.3 t a$^{-1}$

local emission to air:

$E_{\text{local air}} := \frac{F_{\text{main source}} \cdot \text{RELEASE}}{T_{\text{emission}}}$

$E_{\text{local air}} = 121 \text{ kg d}^{-1}$

fraction of the emission to air from STP:

$F_{\text{stp air}} := 0$

local emission rate to STP during emission episode:

$E_{\text{local water}} := 11 \text{ t d}^{-1}$

local emission to air from STP during emission episode:

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

$E_{\text{stp air}} = 0 \text{ kg d}^{-1}$

local concentration in air during emission episode:

$C_{\text{local air}} := \text{wenn } (E_{\text{local air}} \cdot E_{\text{stp air}} \cdot E_{\text{local air}} \cdot C_{\text{std air}} \cdot E_{\text{stp air}} \cdot C_{\text{std air}}) \\nonumber$

$C_{\text{local air}} = 0.034 \text{ mg m}^{-3}$

annual average concentration in air, 100m from point source:

$C_{\text{local air, ann}} := \frac{C_{\text{local air}}}{365 \text{ d a}^{-1}}$

$C_{\text{local air, ann}} = 0.028 \text{ mg m}^{-3}$
regional concentration in air:

annual average predicted environmental concentration in air.

\[ \text{PEC}_{\text{regional air}} := 0 \text{ mg m}^{-3} \]

\[ \text{PEC}_{\text{local air ann}} + \text{PEC}_{\text{regional air}} \]

\[ \text{PEC}_{\text{local air ann}} = 0.028 \text{ mg m}^{-3} \]

**Calculation of the deposition rate**

\[ \text{DEP}_{\text{std aer}} := 1 \cdot 10^{-2} \text{ mg m}^{-2} \text{ d}^{-1} \text{ kg}^{-1} \text{ d}^{-1} \]

fraction of the chemical bound to aerosol

\[ F_{\text{aer}} := 2.6 \times 10^{-7} \]

deposition flux of gaseous compounds as a function of Henry’s Law coefficient, at a source strength of 1 kg/d

\[
\begin{align*}
\log H < -2 & \quad 5 \times 10^{-4} \text{ mg m}^{-2} \text{ d}^{-1} \\
-2 < \log H < 2 & \quad 4 \times 10^{-4} \text{ mg m}^{-2} \text{ d}^{-1} \\
\log H > 2 & \quad 3 \times 10^{-4} \text{ mg m}^{-2} \text{ d}^{-1}
\end{align*}
\]

\[ \text{DEP}_{\text{std gas}} := 4 \times 10^{-4} \text{ mg m}^{-2} \text{ d}^{-1} \text{ kg}^{-1} \text{ d}^{-1} \]

total deposition flux during emission episode:

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

\[ \text{DEP}_{\text{total}} = 0.048 \text{ mg m}^{-2} \text{ d}^{-1} \]

annual average total deposition flux:

\[ \text{DEP}_{\text{total ann}} := \frac{\text{DEP}_{\text{total}} \cdot T_{\text{emission}}}{365 \text{ d} \text{ a}^{-1}} \]

\[ \text{DEP}_{\text{total ann}} = 0.04 \text{ mg m}^{-2} \text{ d}^{-1} \]
Appendix A8  Atmosphere (OPS-model) - external processing (wet and dry polymerisation)

Calculation of C\text{local \ air} and PEC local air

External processing (wet and dry polymerisation)

c\text{local \ air} \text{ : concentration in air at source strength of 1kg/d.} \text{:\quad d := 24 h}

F\text{mainsource} := 1

T\text{emission} := 300 d \cdot a^{-1}

\text{RELEASE := 10 \ tonne \cdot a^{-1}}

C\text{std \ air} := 2.78 \times 10^{-4} \text{mg m}^{-3} \cdot \text{kg}^{-1} \cdot \text{d}

\text{Clocal \ air} \text{ := wenn (Elocal \ air > Estp \ air \cdot Elocal \ water \cdot Cstd \ air \cdot Estp \ air \cdot Cstd \ air)}

\text{Clocal \ air} = 9.267 \times 10^{-3} \text{mg m}^{-3}

\text{Elocal \ air} = 33.333 \text{kg \cdot d}^{-1}

\text{Elocal \ water} := 3300 \text{tonne \cdot d}^{-1}

\text{Estp \ air} := 0 \text{kg \cdot d}^{-1}

\text{Clocal \ air}_{\text{ann}} := \frac{\text{Clocal \ air} \cdot \text{T\text{emission}}}{365 \text{d \cdot a}^{-1}}

\text{Clocal \ air}_{\text{ann}} = 7.616 \times 10^{-3} \text{mg m}^{-3}
regional concentration in air:

annual average predicted environmental concentration in air.

\[ \text{PEC}_{\text{regional \ air}} := 0 \text{ mg m}^{-3} \]

\[ \text{PEC}_{\text{local \ air \ ann}} := \text{local \ air \ ann} + \text{PEC}_{\text{regional \ air}} \]

\[ \text{PEC}_{\text{local \ air \ ann}} = 7.616 \times 10^{-3} \text{ mg m}^{-3} \]

**Calculation of the deposition rate**

\[ \text{DEP}_{\text{std \ aer}} := 1 \times 10^{-2} \text{ mg m}^{-2} \text{ d}^{-1} \text{ kg}^{-1} \text{ d} \]

fraction of the chemical bound to aerosol

\[ \text{Fass aer} := 2.6 \times 10^{-7} \]

deposition flux of gaseous compounds as a function of Henry’s Law coefficient, at a source strength of 1 kg/d

\[
\begin{array}{c|c}
\text{logH} < -2 & 5 \times 10^{-4} \text{ mg m}^{-2} \text{ d}^{-1} \\
-2 < \text{logH} < 2 & 4 \times 10^{-4} \text{ mg m}^{-2} \text{ d}^{-1} \\
\text{logH} > 2 & 3 \times 10^{-4} \text{ mg m}^{-2} \text{ d}^{-1}
\end{array}
\]

\[ \text{DEP}_{\text{std \ gas}} := 4 \times 10^{-4} \text{ mg m}^{-2} \text{ d}^{-1} \text{ kg}^{-1} \text{ d} \]

total deposition flux during emission episode:

\[ \text{DEP}_{\text{total}} := \left( \text{Elocal air} + \text{Estp air} \right) \left[ \text{Fass aer} \text{DEP}_{\text{std aer}} + \left( 1 - \text{Fass aer} \right) \text{DEP}_{\text{std gas}} \right] \]

\[ \text{DEP}_{\text{total}} = 0.013 \text{ mg m}^{-2} \text{ d}^{-1} \]

annual average total deposition flux:

\[ \text{DEP}_{\text{total \ ann}} := \frac{\text{DEP}_{\text{total}}}{365 \text{ d a}^{-1}} \]

\[ \text{DEP}_{\text{total \ ann}} = 0.011 \text{ mg m}^{-2} \text{ d}^{-1} \]
Appendix A9   Exposure of soil

Chemical: Acrylic acid, generic production and processing

Input:
annual average total deposition flux: \( \text{DEP}_{\text{total,ann}} = 0.040 \text{ mg m}^{-2} \text{ d}^{-1} \)
soil-water partitioning coefficient: \( K_{\text{soil,water}} = 1.7 \)
concentration in dry sewage sludge: \( C_{\text{sludge}} = 0 \text{ mg kg}^{-1} \)
air-water partitioning coefficient: \( K_{\text{air,water}} = 1.1 \times 10^5 \)
rate constant for for removal from top soil: \( k_{\text{bio,soil}} = 0.047 \text{ d}^{-1} \)
PECregional:
PECregional natural_soil = 0 \text{ mg kg}^{-1}

Defaults:
mixing depth of soil:

bulk density of soil:

average time for exposure:

partial mass transfer coefficient at air-side of the air-soil interface:

partial mass transfer coefficient at soilair-side of the air-soil interface:

partial mass transfer coefficient at soilwater-side of the air-soil interface:

fraction of rain water that infiltrates into soil:

rate of wet precipitation:

\( \text{DEP}_{\text{total,ann}} = 0.040 \text{ mg m}^{-2} \text{ d}^{-1} \)
\( K_{\text{soil,water}} = 1.7 \)
\( C_{\text{sludge}} = 0 \text{ mg kg}^{-1} \)
\( K_{\text{air,water}} = 1.1 \times 10^5 \)
\( k_{\text{bio,soil}} = 0.047 \text{ d}^{-1} \)
\( \text{DEP}_{\text{total,ann}} = 0.040 \text{ mg m}^{-2} \text{ d}^{-1} \)
\( K_{\text{soil,water}} = 1.7 \)
\( C_{\text{sludge}} = 0 \text{ mg kg}^{-1} \)
\( K_{\text{air,water}} = 1.1 \times 10^5 \)
\( k_{\text{bio,soil}} = 0.047 \text{ d}^{-1} \)
\( \text{DEP}_{\text{total,ann}} = 0.040 \text{ mg m}^{-2} \text{ d}^{-1} \)
\( K_{\text{soil,water}} = 1.7 \)
\( C_{\text{sludge}} = 0 \text{ mg kg}^{-1} \)
\( K_{\text{air,water}} = 1.1 \times 10^5 \)
\( k_{\text{bio,soil}} = 0.047 \text{ d}^{-1} \)
dry sludge application rate: \[ \text{APPL}_{\text{sludge}i} := \begin{array}{c} 0.5 \text{ kg m}^{-2} \text{ a}^{-1} \\ 0.5 \text{ kg m}^{-2} \text{ a}^{-1} \\ 0.1 \text{ kg m}^{-2} \text{ a}^{-1} \end{array} \]

**Calculation:**

aerial deposition flux per kg of soil:
\[ D_{\text{air}i} := \frac{\text{DEP}_{\text{total ann}}}{\text{DEPTH}_{\text{soil}i} \cdot \text{RHO}_{\text{soil}}} \]

rate constant for volatilisation from soil:
\[ k_{\text{volat}i} := \left[ \frac{1}{\text{kasl}_{\text{air}i} \cdot \text{K}_{\text{air-water}}} + \frac{1}{\text{kasl}_{\text{soil}air} \cdot \text{K}_{\text{air-water}} + \text{kasl}_{\text{soil-water}}} \right] \cdot \text{K}_{\text{soil-water}} \cdot \text{DEPTH}_{\text{soil}i} \]

rate constant for leaching from soil layer:
\[ k_{\text{leach}i} := \frac{\text{Finf}_{\text{soil}} \cdot \text{RAINrate}}{\text{K}_{\text{soil-water}} \cdot \text{DEPTH}_{\text{soil}i}} \]

removal from top soil:
\[ k_{i} := k_{\text{volat}i} + k_{\text{leach}i} + k_{\text{bio soil}} \]

**concentration in soil**

concentration in soil due to 10 years of continuous deposition:
\[ \text{C}_{\text{dep soil 10}i} := \frac{D_{\text{air}i}}{k_{i}} \cdot \left(1 - \exp(-365 \cdot 10 \cdot k_{j}^i)\right) \]

concentration just after the first year of sludge application:
\[ \text{C}_{\text{sludge soil 1}i} := \frac{\text{C}_{\text{sludge}} \cdot \text{APPL}_{\text{sludge}i} \cdot a}{\text{DEPTH}_{\text{soil}i} \cdot \text{RHO}_{\text{soil}}} \]

initial concentration in soil after 10 applications of sludge:
\[ \text{C}_{\text{sludge soil 10}i} := \text{C}_{\text{sludge soil 1}i} \left[ 1 + \sum_{n=1}^{9} \left(\exp(-365 \cdot d \cdot k_{j}^i)^n\right) \right] \]
sum of the concentrations due to both processes:

\[ C_{\text{soil}_{10i}} := C_{\text{dep soi}l_{10i}} + C_{\text{sludge soil}_{10i}} \]

average concentration in soil over T days:

\[ C_{\text{local soil}_i} := \frac{D_{\text{air}_i}}{k_i} + \frac{1}{k_i T_i} C_{\text{soil}_{10i}} - \frac{D_{\text{air}_i}}{k_i} \left(1 - \exp(-k_i T_i)\right) \]

\[ \text{PEC}_{\text{local soil}_i} := C_{\text{local soil}_i} + \text{PEC}_{\text{regional natural soil}} \]

<table>
<thead>
<tr>
<th>C_{\text{local soil}_i}</th>
<th>ppm</th>
<th>PEC_{\text{local soil}_i}</th>
<th>ppm</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0024</td>
<td></td>
<td>0.00243</td>
<td></td>
</tr>
<tr>
<td>0.0024</td>
<td></td>
<td>0.00243</td>
<td></td>
</tr>
<tr>
<td>0.0047</td>
<td></td>
<td>0.00469</td>
<td></td>
</tr>
</tbody>
</table>

Indicating persistency of the substance in soil

initial concentration after 10 years:

\[ C_{\text{soil}_{10i}} \]

\[ \begin{array}{c|c|c|c}
  & \text{C}_{\text{soil}_{10i}} & \text{PEC}_{\text{local soil}_i} \\
  \hline
  \text{C}_{\text{local soil}_i} & 0.0024 & 0.00243 \\
  \text{C}_{\text{local agr.soil}_i} & 0.0024 & 0.00243 \\
  \text{C}_{\text{local grassland}} & 0.0047 & 0.00469 \\
\end{array} \]

initial concentration in steady-state situation:

\[ C_{\text{soil}_{ss i}} := \frac{D_{\text{air}_i}}{k_i} + \text{C_{sludge soil}_{1i}} \left(\frac{1}{1 - \exp(-365 \cdot k_i)}\right) \]

\[ \begin{array}{c|c|c|c}
  & \text{C}_{\text{soil}_{10i}} & \text{C}_{\text{soil}_{ss i}} \\
  \hline
  \text{C}_{\text{soil}_{ss i}} & 0.0024 & 0.00243 \\
  \text{C}_{\text{soil}_{ss i}} & 0.0024 & 0.00243 \\
  \text{C}_{\text{soil}_{ss i}} & 0.0047 & 0.00469 \\
\end{array} \]

fraction of steady-state in soil achieved:

\[ F_{st \_st \_i} := \frac{C_{\text{soil}_{10i}}}{C_{\text{soil}_{ss i}}} \]

\[ F_{st \_st \_i} \]
**concentration in pore water**

\[
C_{\text{local soil porew}} = \frac{C_{\text{local soil}} \cdot \rho_{\text{soil}}}{K_{\text{soil water}}} \\
C_{\text{local soil porew}} = 0.00242 \\
C_{\text{local agr soil porew}} = 0.00242 \\
C_{\text{local grassland porew}} = 0.00469
\]

\[
P_{\text{EClocal soil porew}} = \frac{P_{\text{EClocal soil}} \cdot \rho_{\text{soil}}}{K_{\text{soil water}}} \\
P_{\text{EClocal soil porew}} = 0.00242 \\
P_{\text{EClocal agr soil porew}} = 0.00242 \\
P_{\text{EClocal grassland porew}} = 0.00469
\]

**concentration in ground water**

\[
P_{\text{EClocal grw}} = P_{\text{EClocal agr soil porew}}
\]
## Appendix A10 Calculation of continental and regional PECs

**SimpleBox2.0a - calculation of continental and regional PECs**

- adaptation to TGD (1996) / Umweltbundesamt (06/98)

### Physicochemical properties

<table>
<thead>
<tr>
<th>Parameter names acc. SimpleBox20</th>
<th>Unit</th>
<th>Input</th>
<th>Parameter names according Euses</th>
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<tr>
<td>COMPOUND NAME</td>
<td>[-]</td>
<td>AA</td>
<td>Substance</td>
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<td>MOL WEIGHT</td>
<td>[g.mol(^{-1})]</td>
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<td>Molecular weight</td>
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<td>MELTING POINT</td>
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<td>Melting Point</td>
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<td>VAPOUR PRESSURE(25)</td>
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<td>Vapour pressure at 25°C</td>
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<td>log Kow</td>
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<td>SOLUBILITY(25)</td>
<td>[mg.l(^{-1})]</td>
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<td>Water solubility</td>
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</table>

### Distribution - Partition coefficients

- **Solids water partitioning (derived from K\(_{\infty}\))**
  - Kp(soil) \([l.kg^{-1}]\) 1 Solids-water partitioning in soil
  - Kp(sed) \([l.kg^{-1}]\) 1 Solids-water partitioning in sediment
  - Kp(susp) \([l.kg^{-1}]\) 1 Solids-water partitioning in suspended matter

- **Biota-water**
  - BCF(fish) \([l.kg^{-1}]\) 0,49 Bioconcentration factor for aquatic biota

### Degradation and Transformation rates

- **Characterisation and STP**
  - PASSreadytest \([y / n]\) y Characterisation of biodegradability

- **Environmental Total Degradation**
  - kdeg(air) \([d^{-1}]\) 4,20 \(\cdot\) 10\(^{-01}\) Rate constant for degradation in air
  - kdeg(water) \([d^{-1}]\) 4,70 \(\cdot\) 10\(^{-02}\) Rate constant for degradation in bulk surface water
  - kdeg(soil) \([d^{-1}]\) 4,70 \(\cdot\) 10\(^{-02}\) Rate constant for degradation in bulk soil
  - kdeg(sed) \([d^{-1}]\) 2,30 \(\cdot\) 10\(^{-03}\) Rate constant for degradation in bulk sediment

### Sewage treatment (e.g. calculated by SimpleTreat)

- **Continental**
  - FR(volatstp) \([C]\) [-] 0,00 \(\cdot\) 10\(^{-00}\) Fraction of emission directed to air (STPcont)
  - FR(effstt) \([C]\) [-] 1,27 \(\cdot\) 10\(^{-01}\) Fraction of emission directed to water (STPcont)
  - FR(sludgestp) \([C]\) [-] 0,00 \(\cdot\) 10\(^{-00}\) Fraction of emission directed to sludge (STPcont)

- **Regional**
  - FR(volatstp) \([R]\) [-] 0,00 \(\cdot\) 10\(^{-00}\) Fraction of emission directed to air (STPreg)
  - FR(effstt) \([R]\) [-] 1,27 \(\cdot\) 10\(^{-01}\) Fraction of emission directed to water (STPreg)
  - FR(sludgestp) \([R]\) [-] 0,00 \(\cdot\) 10\(^{-00}\) Fraction of emission directed to sludge (STPreg)
Release estimation

- Continental

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Unit</th>
<th>Output</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Edirect(air) [C]</td>
<td>[t.y⁻¹]</td>
<td>277</td>
<td>Total continental emission to air</td>
</tr>
<tr>
<td>STPload [C]</td>
<td>[t.y⁻¹]</td>
<td>973</td>
<td>Total continental emission to wastewater</td>
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<tr>
<td>Edirect(water1) [C]</td>
<td>[t.y⁻¹]</td>
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<td>Edirect(soil3) [C]</td>
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<td>Total continental emission to industrial soil</td>
</tr>
<tr>
<td>Edirect(soil2) [C]</td>
<td>[t.y⁻¹]</td>
<td>0</td>
<td>Total continental emission to agricultural soil</td>
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- Regional

<table>
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<th>Parameter</th>
<th>Unit</th>
<th>Output</th>
<th>Description</th>
</tr>
</thead>
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<td>Edirect(air) [R]</td>
<td>[t.y⁻¹]</td>
<td>54</td>
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<tr>
<td>STPload [R]</td>
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<td>Total continental emission to wastewater</td>
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<tr>
<td>Edirect(water1) [R]</td>
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<tr>
<td>Edirect(soil3) [R]</td>
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<td>Total continental emission to industrial soil</td>
</tr>
<tr>
<td>Edirect(soil2) [R]</td>
<td>[t.y⁻¹]</td>
<td>0</td>
<td>Total continental emission to agricultural soil</td>
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</table>

### Physicochemical properties

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<th>Parameter</th>
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<td>COMPOUND NAME</td>
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<td>Substance</td>
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### Output

- Continental

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<td>PECsurfacewater (total)</td>
<td>[mg.l⁻¹]</td>
<td>3.39·10⁻⁵</td>
<td>Continental PEC in surface water (total)</td>
</tr>
<tr>
<td>PECsurfacewater (dissolved)</td>
<td>[mg.l⁻¹]</td>
<td>3.39·10⁻⁵</td>
<td>Continental PEC in surface water (dissolved)</td>
</tr>
<tr>
<td>PECair</td>
<td>[mg.m⁻³]</td>
<td>3.28·10⁻⁷</td>
<td>Continental PEC in air (total)</td>
</tr>
<tr>
<td>PECagr.soil</td>
<td>[mg.kg⁻¹]</td>
<td>3.29·10⁻⁶</td>
<td>Continental PEC in agricultural soil (total)</td>
</tr>
<tr>
<td>PECporewater agr.soil</td>
<td>[mg.l⁻¹]</td>
<td>3.29·10⁻⁶</td>
<td>Continental PEC in pore water of agricultural soils</td>
</tr>
<tr>
<td>PECnat.soil</td>
<td>[mg.kg⁻¹]</td>
<td>1.12·10⁻⁵</td>
<td>Continental PEC in natural soil (total)</td>
</tr>
<tr>
<td>PECind.soil</td>
<td>[mg.kg⁻¹]</td>
<td>1.12·10⁻⁵</td>
<td>Continental PEC in industrial soil (total)</td>
</tr>
<tr>
<td>PECsediment</td>
<td>[mg.kg⁻¹]</td>
<td>3.15·10⁻⁵</td>
<td>Continental PEC in sediment (total)</td>
</tr>
</tbody>
</table>

- Regional

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Unit</th>
<th>Output</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>PECsurfacewater (total)</td>
<td>[mg.l⁻¹]</td>
<td>3.96·10⁻⁴</td>
<td>Regional PEC in surface water (total)</td>
</tr>
<tr>
<td>PECsurfacewater (dissolved)</td>
<td>[mg.l⁻¹]</td>
<td>3.96·10⁻⁴</td>
<td>Regional PEC in surface water (dissolved)</td>
</tr>
<tr>
<td>PECair</td>
<td>[mg.m⁻³]</td>
<td>2.04·10⁻⁶</td>
<td>Regional PEC in air (total)</td>
</tr>
<tr>
<td>PECagr.soil</td>
<td>[mg.kg⁻¹]</td>
<td>2.05·10⁻⁵</td>
<td>Regional PEC in agricultural soil (total)</td>
</tr>
<tr>
<td>PECporewater agr.soil</td>
<td>[mg.l⁻¹]</td>
<td>2.05·10⁻⁵</td>
<td>Regional PEC in pore water of agricultural soils</td>
</tr>
<tr>
<td>PECnat.soil</td>
<td>[mg.kg⁻¹]</td>
<td>6.95·10⁻⁵</td>
<td>Regional PEC in natural soil (total)</td>
</tr>
<tr>
<td>PECind.soil</td>
<td>[mg.kg⁻¹]</td>
<td>6.95·10⁻⁵</td>
<td>Regional PEC in industrial soil (total)</td>
</tr>
<tr>
<td>PECsediment</td>
<td>[mg.kg⁻¹]</td>
<td>3.73·10⁻⁴</td>
<td>Regional PEC in sediment (total)</td>
</tr>
</tbody>
</table>
Appendix A11  Indirect exposure via the environment

(TGD, Chapter 2)

**Input**

**chemical properties**

- octanol-water partitioning coefficient \( K_{\text{OW}} \)
  - log \( K_{\text{OW}} \) := 0.46
  - \( K_{\text{OW}} := 10^{\log K_{\text{OW}}} \)
- Henry - partitioning coefficient \( K_{\text{HENRY}} \)
  - log \( K_{\text{HENRY}} \) := -0.027 \( \text{Pa} \cdot \text{m}^3 \cdot \text{mol}^{-1} \)
- air-water partitioning coefficient \( K_{\text{air\_water}} \)
  - \( K_{\text{air\_water}} := 1.12 \times 10^{-5} \) \( \text{Pa} \cdot \text{m}^3 \cdot \text{mol}^{-1} \)
- fraction of the chemical associated with aerosol particles \( F_{\text{ass\_aer}} \)
  - \( F_{\text{ass\_aer}} := 2.6 \times 10^{-7} \)
- half-life for biodegration in surface water \( D_{\text{T}_{50\_bio\_water}} \)
  - \( D_{\text{T}_{50\_bio\_water}} := 15 \text{d} \)

**environmental concentrations**

- annual average local PEC in surface water (dissolved) \( \text{PEC}_{\text{local\_water\_ann}} \)
  - \( \text{PEC}_{\text{local\_water\_ann}} := 270 \mu g \cdot l^{-1} \cdot \frac{300}{365} \)
- annual average local PEC in air (total) \( \text{PEC}_{\text{local\_air\_ann}} \)
  - \( \text{PEC}_{\text{local\_air\_ann}} := 28 \mu g \cdot m^{-3} \)
- local PEC in grassland (total), averaged over 180 days \( \text{PEC}_{\text{local\_grassland}} \)
  - \( \text{PEC}_{\text{local\_grassland}} := 0.00469 \mu g \cdot kg^{-1} \)
- local PEC in porewater of agriculture soil \( \text{PEC}_{\text{local\_agr\_soil\_porew}} \)
  - \( \text{PEC}_{\text{local\_agr\_soil\_porew}} := 0.00242 \mu g \cdot l^{-1} \)
- local PEC in porewater of grassland \( \text{PEC}_{\text{local\_grassland\_porew}} \)
  - \( \text{PEC}_{\text{local\_grassland\_porew}} := 0.00469 \mu g \cdot l^{-1} \)
- local PEC in groundwater under agriculture soil \( \text{PEC}_{\text{local\_grw}} \)
  - \( \text{PEC}_{\text{local\_grw}} := 0.00242 \mu g \cdot l^{-1} \)
- regional PEC in surface water (dissolved) \( \text{PEC}_{\text{regional\_water}} \)
  - \( \text{PEC}_{\text{regional\_water}} := 0.396 \mu g \cdot l^{-1} \)
- regional PEC in air (total) \( \text{PEC}_{\text{regional\_air}} \)
  - \( \text{PEC}_{\text{regional\_air}} := 2 \times 10^{-3} \mu g \cdot m^{-3} \)
- regional PEC in agriculture soil (total) \( \text{PEC}_{\text{regional\_agr\_soil}} \)
  - \( \text{PEC}_{\text{regional\_agr\_soil}} := 0.02 \mu g \cdot kg^{-1} \)
- regional PEC in porewater of agriculture soils \( \text{PEC}_{\text{regional\_agr\_soil\_porew}} \)
  - \( \text{PEC}_{\text{regional\_agr\_soil\_porew}} := 0.02 \mu g \cdot l^{-1} \)
Definition of the concentrations used for indirect exposure

\[ C_{\text{water,local}} := PEC_{\text{local,water}} \]
\[ C_{\text{air,local}} := PEC_{\text{local,air}} \]
\[ C_{\text{grassland,local}} := PEC_{\text{local,grassland}} \]
\[ C_{\text{agr_porew,local}} := PEC_{\text{local,agr_soil_porew}} \]
\[ C_{\text{grass_porew,local}} := PEC_{\text{local,grassland_porew}} \]
\[ C_{\text{grw,local}} := PEC_{\text{local,grw}} \]
\[ C_{\text{water,regional}} := PEC_{\text{regional,water}} \]
\[ C_{\text{air,regional}} := PEC_{\text{regional,air}} \]
\[ C_{\text{grassland,regional}} := PEC_{\text{regional,agr_soil}} \]
\[ C_{\text{agr_porew,regional}} := PEC_{\text{regional,agr_soil_porew}} \]
\[ C_{\text{grass_porew,regional}} := PEC_{\text{regional,agr_soil_porew}} \]
\[ C_{\text{grw,regional}} := PEC_{\text{regional,agr_soil_porew}} \]

Results of calculation

\[ \text{DOSE}_{\text{tot,local}} = 0.05 \text{ mg kg bw d} \]
\[ \text{DOSE}_{\text{tot,regional}} = 1.5 \times 10^{-5} \text{ mg kg bw d} \]

\[ \text{RDOSE}_{\text{drw,local}} = 11.83 \% \]
\[ \text{RDOSE}_{\text{drw,regional}} = 74.78 \% \]
\[ \text{RDOSE}_{\text{air,local}} = 11.19 \% \]
\[ \text{RDOSE}_{\text{air,regional}} = 2.83 \% \]
\[ \text{RDOSE}_{\text{stem,local}} = 76.6 \% \]
\[ \text{RDOSE}_{\text{stem,regional}} = 19.56 \% \]
\[ \text{RDOSE}_{\text{root,local}} = 0.02 \% \]
\[ \text{RDOSE}_{\text{root,regional}} = 0.7 \% \]
\[ \text{RDOSE}_{\text{meat,local}} = 1.13 \times 10^{-3} \% \]
\[ \text{RDOSE}_{\text{meat,regional}} = 7.61 \times 10^{-4} \% \]
\[ \text{RDOSE}_{\text{milk,local}} = 0.02 \% \]
\[ \text{RDOSE}_{\text{milk,regional}} = 0.01 \% \]
\[ \text{RDOSE}_{\text{fish,local}} = 0.33 \% \]
\[ \text{RDOSE}_{\text{fish,regional}} = 2.11 \% \]
Appendix B1  Simulation of consumer exposure to UV-hardening adhesives

Substance:  
Acrylic acid

CAS:  
79-10-7

Computer model used:  
EPA-model: SCIES  
(Screening-Level Consumer Inhalation Exposure Software)

Category of consumer products:  
UV-hardening adhesives

Results:  
User potential dose rate from inhalation:  
\[ 7.70 \text{ Milligramm/yr} \]
\[ = 13.0 \text{ Mikrogramm/day} \]
\[ = 216 \text{ Nanogramm/kg b.w. and day} \]

Generic Product

Annual Frequency of Use:  
4 Events/Year
Mass of Product:  
1.000 grams
Duration of Use:  
1.000 Hours
Zone 1 Volume:  
40.000 cubic meters
Whole House Volume:  
292.000 cubic meters
House Air Exchange Rate:  
0.200 room air exchanges/hr
User Inhalation Rate:  
1.300 cubic meters/hr (during use)
Non-User Inhalation Rate:  
1.100 cubic meters/hr (& User after use)
Molecular Weight:  
72.060 g/mole
Vapour Pressure:  
7.725E+00 torr
Weight Fraction:  
0.060
Starting Time:  
12:00 NOON

OUTPUT SUMMARY

Evaporation Time:  
0.347 Hours
Release Time:  
1.000 Hours (Duration of Exposure)
Duration Following Each Use:  
2189.000 Hours
Interval Between Uses:  
2190.000 Hours

User Potential Dose Rate From Inhalation:  
4.706 mg/yr
Non-User Potential Dose Rate From Inhalation:  
2.937 mg/yr

-------------------------------------------------------------------------------------
### Concentration in zone of release

<table>
<thead>
<tr>
<th></th>
<th>Average (mg/m³)</th>
<th>Peak (mg/m³)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>During period of use</strong></td>
<td>0.384</td>
<td>0.542</td>
</tr>
<tr>
<td><strong>During period after use</strong></td>
<td>0.000</td>
<td>0.448</td>
</tr>
</tbody>
</table>

### Concentration in Zone 2

<table>
<thead>
<tr>
<th></th>
<th>Average (mg/m³)</th>
<th>Peak (mg/m³)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>During period of use</strong></td>
<td>0.052</td>
<td>0.119</td>
</tr>
<tr>
<td><strong>During period after use</strong></td>
<td>0.000</td>
<td>0.154</td>
</tr>
</tbody>
</table>

### Concentration to which User and Non-User are exposed

<table>
<thead>
<tr>
<th></th>
<th>Person Using Product (user)</th>
<th>Person Not Using Product (non-user)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>During period of use</strong></td>
<td>0.000</td>
<td>0.542</td>
</tr>
<tr>
<td><strong>During period after use</strong></td>
<td>0.000</td>
<td>0.154</td>
</tr>
</tbody>
</table>

### HOURLY ACTIVITY PATTERN

**User:**

```
11111123454267422744411
```

**Non-User:**

```
111111132442476422644411
```

**Hour:**

```
03 06 09 15 18 21 24
```
The report provides the comprehensive risk assessment of the substance acrylic acid. It has been prepared by Germany in the frame of Council Regulation (EEC) No. 793/93 on the evaluation and control of the risks of existing substances, following the principles for assessment of the risks to 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 human health risk assessment for acrylic acid concludes that there is a concern for workers. For consumers and human exposed via the environment the risk assessment concludes that there is at present no concern.

The risk assessment for the environment concludes that there is concern for the aquatic ecosystem, and a need for further information on risks for microorganisms in the sewage treatment plant. The information requirement is postponed since risk reduction measures to protect surface water will also protect municipal wastewater treatment plants.

The conclusions of this report will lead to risk reduction measures to be proposed by the Commission's Committee on risk reduction strategies set up in support of Council Regulation (EEC) 793/93.
The mission of the JRC is to provide customer-driven scientific and technical support for the conception, development, implementation and monitoring of EU policies. As a service of the European Commission, the JRC functions as a reference centre of science and technology for the Union. Close to the policy-making process, it serves the common interest of the Member States, while being independent of special interests, private or national.