European Chemicals Bureau

Institute for Health and Consumer Protection

European Chemicals Bureau

Existing Substances

European Union Risk Assessment Report

CAS No: 1310-73-2

EINECS No: 215-185-5

sodium hydroxide

$NaOH$

4th Priority List

Volume: 73

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

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

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

Contact information:

Institute of Health and Consumer Protection (IHCP)
Address: Via E. Fermi – 21027 Ispra (Varese) – Italy
E-mail: ihcp-contact@jrc.it
Tel.: +39 0332 785959
Fax: +39 0332 785730
http://ihcp.jrc.ec.europa.eu

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

Directorate-General Joint Research Centre
http://www.jrc.ec.europa.eu/dgs/jrc/index.cfm

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SODIUM HYDROXIDE

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TARGETED RISK ASSESSMENT

Cataloguing data can be found at the end of this publication

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TARGETED RISK ASSESSMENT

Final Report 2007

Portugal

Rapporteur for the risk assessment of Sodium Hydroxide is Portugal. Responsible for the risk evaluation and subsequently for the contents of this report, is the rapporteur.

The scientific work on this report has been prepared by the Netherlands Organisation for Applied Scientific Research (TNO) and the National Institute for Public Health and the Environment (RIVM), by order of the rapporteur.

Contact point:

Environment : Institute for the Environment
Rua da Murgueira, 9/9A
Apartado 7585
2611-865 Amadora
Portugal

Human Health: General Directorate for Health
Alameda D. Afonso Henriques, 45
1049-005 Lisboa
Portugal

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Final report:	**2007**

Foreword

We are pleased to present this Risk Assessment Report which is the result of in-depth work carried out by experts in one Member State, working in co-operation with their counterparts in the other Member States, the Commission Services, Industry and public interest groups.
The Risk Assessment was carried out in accordance with Council Regulation (EEC) 793/93[1] on the evaluation and control of the risks of "existing" substances. "Existing" substances are chemical substances in use within the European Community before September 1981 and listed in the European Inventory of Existing Commercial Chemical Substances. Regulation 793/93 provides a systematic framework for the evaluation of the risks to human health and the environment of these substances if they are produced or imported into the Community in volumes above 10 tonnes per year.
There are four overall stages in the Regulation for reducing the risks: data collection, priority setting, risk assessment and risk reduction. Data provided by Industry are used by Member States and the Commission services to determine the priority of the substances which need to be assessed. For each substance on a priority list, a Member State volunteers to act as "Rapporteur", undertaking the in-depth Risk Assessment and recommending a strategy to limit the risks of exposure to the substance, if necessary.
The methods for carrying out an in-depth Risk Assessment at Community level are laid down in Commission Regulation (EC) 1488/94[2], which is supported by a technical guidance document[3]. 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 Health and Environmental Risks (SCHER) 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 and confirmed in the Johannesburg Declaration on Sustainable Development at the World Summit on Sustainable Development, held in Johannesburg, South Africa in 2002.
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.

Roland Schenkel
Director General
DG Joint Research Centre

Mogens Peter Carl
Director General
DG Environment

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[1] O.J. No L 084, 05/04/199 p.0001 – 0075

[2] O.J. No L 161, 29/06/1994 p. 0003 – 0011

[3] Technical Guidance Document, Part I – V, ISBN 92-827-801 [1234]

0 OVERALL RESULTS OF THE RISK ASSESSMENT

CAS Number: 1310-73-2
EINECS Number: 215-185-5
IUPAC Name: Sodium hydroxide

Environment

Conclusions to the risk assessment for the aquatic compartment:

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

Conclusion (ii) applies to production and use of NaOH. Based on the results from a questionnaire among producers and users, it is concluded that discharges of NaOH from production are well controlled and that discharges of NaOH from the various downstream applications rarely occur. The available data clearly indicate that neutralisation of NaOH containing waste waters and effluents is common practice, either from a legal point of view (legislation for surface waters) or from a practical point of view (protection of the functioning of biological STPs/WWTPs). Regarding surface water, the enforcement of the (EU) legislation is an important issue for the validity of conclusion (ii).

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.

Conclusion (iii) is reached because it cannot be excluded that respiratory tract irritation may occur in the production when bagging NaOH, and when using NaOH in aluminium and textile industry and in the de-inking of waste paper in pulp and paper industry.

In relation to all other potential adverse effects and the worker population it is concluded that based on the available information at present there is no concern and no further information/testing on the substance is needed.

Consumers

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

Conclusion (iii) applies to the high number of accidents (foreseeable misuse) that occur with sodium hydroxide, which points out that consumer protection against improper use of sodium hydroxide is insufficient. Because sodium hydroxide has local effects the conclusion (iii) is applicable for the endpoint “irritation and corrosivity” for all routes of exposure (oral, dermal, ocular and inhalatory exposure).

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

Conclusion (ii) applies to the normal use of corrosive and irritating concentrations of sodium hydroxide if the required protection is used.

Humans exposed via the environment

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

Production and use of sodium hydroxide is normally not expected to increase the pH of the environment. Even after an accidental release the substance will be neutralised finally and therefore the human exposure to sodium hydroxide via the environment is expected to be negligible. Therefore, no direct or systemic exposure via the environment is expected from sodium hydroxide.

Human health (physico-chemical properties)

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

This conclusion is reached because the risk assessment shows that risks are not expected. Risk reduction measures already being applied are considered sufficient.

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TABLES

0.1 GENERAL INTRODUCTION: 'TARGETED RISK ASSESSMENT'

In order to accelerate the EU risk assessment process for existing substances, the European Commission has clearly expressed a wish to perform so-called 'targeted risk assessments' (TRA) for the 4th Priority-list substances, which include NaOH. In this context 'targeted' means that not all endpoints, as defined in the Technical Guidance Documents (TGD), are addressed thoroughly in the risk assessment. In a TRA one deviates therefore from the standard comprehensive risk assessment that covers all possible exposure routes of the chemical and all protection goals. Arguments and requirements for performing a TRA within EC Regulation 793/93 are discussed in the CA discussion paper 'Use of Targeted Risk Assessments in the EU' (CA, 2001).

Based on this and taken into account the data and conclusions in the recent SIDS Initial Assessment Report on NaOH (OECD, 2002), the Rapporteur proposed to perform a targeted risk assessment for NaOH (Portugal, 2003). This proposal was approved by the EU member states at the Technical Meeting in December 2003 (ECB, 2003). For the environmental risk assessment it was agreed to focus solely on the aquatic environment, as the emissions of NaOH in the different life-cycle stages (production and use) mainly apply to (waste) water. The aquatic risk assessment will only deal with the effect on organisms/ecosystems due to possible pH changes related to OH^- discharges, as the toxicity of the Na^+ ion is expected to be insignificant compared to the (potential) pH effect. Only the local scale (thus not the regional scale) will be addressed, when applicable including sewage treatment plants (STPs) or waste water treatment plants (WWTPs), both for production and industrial use.

For the human health risk assessment it was agreed to focus on the risks from acute exposure (local effects, thus not systemic effects), both for workers and consumers. Regarding the environmental and human health effects assessments (hazard identifications) it was concluded that these subjects are covered adequately by the OECD report, thus the effects assessments data from the OECD report will be used without a re-evaluation or update. The same applies to the IUCLID that is included in the OECD evaluation[4]. Thus, the key-issues in this EU Risk Assessment Report are the exposure assessments and the risk characterisations for the aquatic environment (STPs/WWTPs included), workers and consumers.

[4] A number of additional references were used, however, to add essential information, for example on pH tolerance of aquatic species.

1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTIFICATION OF THE SUBSTANCE

CAS Number: 1310-73-2
EINECS Number: 215-185-5
IUPAC Name: Sodium hydroxide
Molecular Formula: NaOH
Structural Formula: NaOH
Molecular Weight: 40
Synonyms: Caustic soda (liquid or solid)
Liquid caustic
Lye (liquid)
Caustic flake (solid)
Sodium hydrate

1.2 PURITY/IMPURITIES, ADDITIVES

Sodium hydroxide (NaOH) is a white and deliquescent solid. Impurities are sodium chloride (≤ 2%), sodium carbonate (≤ 1.0%) and sulfate (≤ 0.2%). The concentration of other impurities is less than 0.1%.

1.3 PHYSICO-CHEMICAL PROPERTIES

Table 1.1 Summary of physico-chemical properties

Physico-chemical properties	Results
Melting point	318°C (solid, 100%)
	140°C (solution of 80%)
	42°C (solution of 60%)
	16°C (solution of 40%)
	-26°C (solution of 20%)
Boiling point	1388°C at 1013 hPa (solid, 100%)
	216°C at 1013 hPa (solution of 80%)
	160°C at 1013 hPa (solution of 60%)
	128°C at 1013 hPa (solution of 40%)
	118°C at 1013 hPa (solution of 20%)
Density	2.13 at 20°C (solid, 100%)
	1.43 at 20°C (solution of 40%)
	1.22 at 20°C (solution of 20%)
Water solubility	52% (by weight) at 20°C
	42% (by weight) at 0°C
Vapour pressure	$< 10^{-5}$ hPa at 25°C (calculated)

Table 1.1 continued overleaf

Table 1.1 continued Summary of physico-chemical properties

Physico-chemical properties	Results
Partition coeffient (log K_{ow})	Not applicable
Oxidising/explosive properties	Not applicable
Autoflammability	Not applicable

NaOH is miscible with water at all proportions but solidifies at 20°C if the concentration is higher than 52% (by weight), which can be considered the maximum water solubility at 20°C. NaOH has a very low vapour pressure (< 10^{-5} hPa at 25°C). The octanol water partition coefficient is not relevant for an inorganic substance such as NaOH.

NaOH is a strong alkaline substance that dissociates completely in water into the sodium ion (Na^+) and hydroxyl ion (OH^-). The dissolution/dissociation in water is strongly exothermic, so a vigorous reaction occurs when NaOH is added to water.

1.4 CLASSIFICATION

1.4.1 Current classification

Sodium hydroxide is included in Annex I of Directive 67/548/EEC.

Classification: C; R35

Label: C

R- phrases: 35

S- phrases: (1/2)-26-37/39-45

Specific concentration limits:

C ≥ 5%: C, R35
2% ≤ C < 5%: C, R34
0.5% ≤ C < 2%: Xi, R36/38

This has remained unchanged since the 19th ATP (1 September, 2003).

1.4.2 Proposed classification

No changes to the classification are proposed.

2 GENERAL INFORMATION ON EXPOSURE

2.1 PRODUCTION

2.1.1 Production processes

The production of sodium hydroxide (NaOH) is based on the electrolysis of NaCl, which can be done via the mercury, diaphragm or membrane process. In most electrolysis processes in Europe NaOH is formed in the electrolysis liquid, simultaneously with chlorine at the anode and hydrogen at the cathode. An illustration of these processes can be found in Euro Chlor (2004a). An overview of the production sites of chlorine/NaOH in Europe is provided in **Table 2.1**, representing the situation at the end 2003 (Euro Chlor, 2004b). The distribution of the production routes to chlorine/NaOH in Western Europe is mercury process 46%, diaphragm process 18%, membrane process 33% and other processes 3%. NaOH is mainly commercialised as a solution in water at different concentrations (lye), or as solid (cast, flakes, pearls). Solid NaOH was produced at 27% of the production sites, but covers only a small percentage (4%) of the total market; the remaining amount (96%) is NaOH in solution. The most important industrial concentration is 50% (Euro Chlor, 2004c), NaOH solidifies at a concentration of higher than 52% (by weight) at 20°C (OECD, 2002).

2.1.1.1 Mercury process

In the mercury electrolyser, mercury flows concurrently with a solution of salt (brine) along the base of an electrolytic cell. The mercury acts as the cathode and forms an amalgam with sodium. Chlorine is formed at the coated titanium anodes, which are suspended in the brine. The amalgam flows to a reactor (denuder or decomposer) where the amalgam reacts with water in the presence of carbon (graphite) to form caustic soda and hydrogen. The free mercury is returned to the electrolytic cell. The resulting caustic soda solution is then stored in tanks as a 50% solution. Under normal operating conditions the mercury content is 40-60 µg/kg, but in certain cases values higher than 200 µg/kg have been measured (Euro Chlor, 2004a).

2.1.1.2 Diaphragm process

Diaphragm cells can have a monopolar (cells in parallel) or in some cases a bipolar (cells in series) configuration and there are different types of construction. In the diaphragm electrolyser an asbestos or synthetic fibers diaphragm separates the anolyte and catholyte chambers. In some cases polymer modified asbestos is used as the diaphragm. The anode is titanium with a suitable rare metal oxide coating and the cathode is steel or nickel coated steel. Differential hydraulic pressure causes the anolyte to flow through the diaphragm from the anolyte compartment to the catholyte compartment. Chlorine is removed from the gas space above the anolyte normally under suction. Diaphragm cell liquor containing 9-12% caustic soda and 15-17% sodium chloride overflows from the catholyte chamber to intermediate storage. This liquor can be used directly for other processes or sent to an additional evaporation unit, with separation from the precipitated NaCl to reach the commercial concentration of 50% caustic soda. The sodium chloride concentration in 50% caustic soda liquor from this process is about 1-1.5% (EC, 2001).

2.1.1.3 Membrane process

Membrane electrolysers can also have a monopolar or more modern bipolar configuration. In the membrane electrolysers an ion selective membrane separates the anolyte and catholyte chambers. In comparison with the diaphragm electrolyser there is no physical flow from the anolyte to the catholyte chamber. Instead, sodium ions pass through the membrane and form caustic soda and hydrogen in the catholyte. Caustic soda and hydrogen are produced in the catholyte compartment by the addition of water. The anodes are made from titanium with a suitable rare metal oxide coating. The cathodes are constructed in steel or nickel and possibly have a coating. The strength of the caustic soda in the membrane process is up to 33%. The solution is then usually sent to evaporators, which concentrate it to a 50% solution by removing the water

2.1.2 Production capacity

In the European Union (EU), 50 companies at 84 sites in 20 countries produce chlorine and the vast majority of them simultaneously produce NaOH. **Table 2.1** presents the chlorine production within the European Union, including the used processes and production capacity. Based on **Table 2.1** the total EU production capacity of chlorine was 12.4 million tonnes in 2003. Sodium hydroxide is produced in a fixed ratio of 1.128 tonnes (as 100% NaOH) per tonne chlorine produced and therefore the EU production capacity of NaOH was 14 million tonnes in 2003. The Western European consumption of sodium hydroxide was 9.7 million tonnes in 2003 (see **Table 2.2**). No information was reported about the import or export from the EU. Of the total amount of NaOH produced and used, about 96% is in liquid form (NaOH solutions in water) and 4% in solid form (Dr. D. van Wijk, Euro Chlor, personal communication, 2004).

Table 2.1 Production sites of NaOH in the EU (Euro Chlor, 2004b)

Country	Number on Map*	Company	Site	Basin	Process	Capacity (000 tonnes)
AUSTRIA	1	Donau Chemie	Brückl	D	M	60
BELGIUM	3	Solvin	Antwerp (Lillo)	A	Hg	330
	4	Solvin	Jemeppe	A	M	174
	5	Tessenderlo Chemie	Tessenderlo	A	Hg	250
CZECH REPUBLIC	6	Spolana	Neratovice	A	Hg	135
	7	Spolchemie	Usti	A	Hg	62
FINLAND	8	Akzo Nobel	Oulu	C	Hg	43
	9	Finnish Chemicals	Joutseno	C	M	75
FRANCE	10	Albemarle	Thann	A	Hg	72
	11	ChlorAlp	Pont de Claix	B	D	240
	12	Atofina	Fos	B	D, M	270
	13	Atofina	Jarrie	B	Hg	170
	14	Atofina	Lavera	B	Hg, D	341
	15	Atofina	Saint Auban	B	Hg	184
	16	MSSA	Pomblières	B	Na	42
	17	Prod. Chim. D'Harbonnières	Harbonnières	A	Hg	22.5
	18	Solvay	Tavaux	B	Hg, M	375
	19	Tessenderlo Chemie	Loos	A	Hg	18
GERMANY	20	BASF	Ludwigshafen	A	Hg, M	370
	21	Bayer	Dormagen	A	M, HCl	450
	22	Bayer	Leverkusen	A	M, HCl	330
	23	Bayer	Uerdingen	A	Hg, M	220
	24	Bayer	Brunsbuttel	A	HCl	190
	25	Dow	Schkopau	A	M	215
	26	Vinnolit	Knapsack	A	Hg, M	280
	27	Clariant	Gersthofen	D	M	40
	28	Dow	Stade	A	D, M	1444
	29	Akzo Nobel	Ibbenbüren	A	Hg	125
	30	Akzo Nobel	Bitterfeld	A	M	75
	31	Degussa	Lulsdorf	A	Hg	138
	32	Ineos Chlor	Wilhemshafen	A	Hg	149
	33	LII Europe	Frankfurt	A	Hg	167
	34	Solvay	Rheinberg	A	D	200
	35	Vestolit	Marl	A	Hg, M	216
	36	Vinnolit	Gendorf	D	Hg	82
	37	Wacker	Burghausen	D	M	50
GREECE	38	Hellenic Petroleum	Thessaloniki	B	Hg	40
HUNGARY	39	BorsodChem	Kazinbarcika	D	Hg	137
IRELAND	40	MicroBio	Fermoy	A	M	6
ITALY	41	Altair Chimica	Volterra	B	Hg	27
	42	Solvay	Bussi	B	Hg	89
	43	Caffarro	Toreviscosa	B	Hg	68
	44	Syndial	Assemini/Cagliari	B	M	170
	45	Syndial	Porto Marghera	B	Hg	200
	47	Syndial	Priolo	B	Hg	204
	48	Eredi Zarelli	Picinisco	B	Hg	6
	49	Solvay	Rosignano	B	Hg	127
	50	Tessenderlo Chemie	Pieve Vergonte	B	Hg	42
THE NETHERLANDS	51	Akzo Nobel	Botlek	A	M	424
	52	Akzo Nobel	Delfzijl	A	D	125
	53	Akzo Nobel	Hengelo	A	Hg	74
	54	General Electric Plastics	Bergen-op-Zoom	A	M	87
NORWAY	55	Borregaard	Sarpsborg	A	M	45
	56	Elkem	Bremanger	A	M	10
	57	Norsk Hydro	Rafnes	A	D	136
POLAND	58	Rokita	Brzeg Dolny	C	Hg	127
	59	Zachem	Bydgoszcz	C	D	60
	60	Anwil	Wloclawek	C	D	197
	86	Dwory	Oswiecim	C	Hg	39
	87	Tarnow	Tarnow	C	Hg	32
PORTUGAL	61	Solvay	Povoa	A	M	29
	62	Quimigal	Estarreja	A	M	68
SLOVAK REPUBLIC	63	Novacke Chemicke	Novaky	D	Hg	76
SLOVENIA	88	TKI Hrastnik	Hrastnik	D	M	15
SPAIN	64	EIASA (Aragonesas)	Huelva	A	Hg	101
	65	EIASA (Aragonesas)	Sabinanigo	B	Hg	25
	66	EIASA (Aragonesas)	Villaseca	B	Hg, M	190
	67	Electroq. de Hernani	Hernani	A	M	15
	68	Elnosa	Lourizan	A	Hg	34
	69	Ercros	Flix	B	Hg	150
	70	Quimica del Cinca	Monzon	B	Hg	31
	71	Solvay	Martorell	B	Hg	223
	72	Solvay	Torrelavega	A	Hg	63
SWEDEN	73	Akzo Nobel	Bohus	A	Hg	100
	74	Akzo Nobel	Skoghall	A	M	85
	75	Norsk Hydro	Stenungsund	A	Hg	120
SWITZERLAND	76	Syngenta	Monthey	B	Hg	27
	77	SF-Chem	Pratteln	A	Hg	27
	89	Borregaard	Atisholtz	A	M	9
UK	80	Albion Chemicals	Sandbach	A	Hg	90
	82	Ineos Chlor	Runcorn	A	Hg, M	767
	84	Rhodia	Staveley	A	Hg	30
	85	Albion Chemicals	Thetford	A	M	6

BASIN **A**: North Sea – Atlantic **B**: Mediterranean Sea **C**: Baltic Sea **D**: Black Sea
PROCESS **Hg**: Mercury **M**: Membrane **Na**: Sodium **D**: Diaphragm **HCl**: Electrolysis of HCl to Cl_2

2.2 USES

2.2.1 Introduction

NaOH is used for different purposes in a variety of industrial sectors. The sector with the largest use of NaOH is the production of other chemicals, both organics (28%) and inorganics (16%). Other uses are in the sectors pulp and paper industry (12%), aluminium and metal industry (6%), food industry (3%), water treatment (3%) and textile (3%). The remainder is used in the production of soaps, mineral oils, bleach, phosphates, cellulose, rubber and others (Euro Chlor, 2004b). **Table 2.2** presents the major sectors where NaOH is applied. An indication of the 'industrial category' is also included in the table. The actual use or purpose of NaOH can be quite similar across different sectors; a summary of the main uses will be given.

- One main application of NaOH in chemical production is as a process aid, when certain pH conditions are needed to optimise reactions, for example in reactions where acid is produced. Examples are in the production of organics and inorganics, in the pulp and paper industry and in the metal industry, for example when a metal ore is first agressively treated with acid and the obtained product needs to be neutralised for further processing. Also in waste water treatment NaOH is used for its neutralisation property.
- Another major use of NaOH is because of its aggressive properties, for example in the aluminium industry to treat bauxit to solubilise the Al, or in other metal industries or in the food industry to peel peaches or tomatoes.
- A third major application is as cleaning agent, among others because it effectively dissolves grease. Examples of sectors using NaOH for cleaning are the food industry, cleaning recycled bottles and in households as oven cleaner or drain deblocker.
- Finally the use of the Na in NaOH can be the actual application, such as in the production of soap where Na is the counter ion or in the production of phosphate, sodium percarbonate and sodium perborate.

The most feasible use category for NaOH is 'pH regulators' (UC=40), which is probably applied in all sectors mentioned in **Table 2.1**, but most prominently in the waste water treatment sector. Other use categories are less applicable, for example 'cleaning/washing agent' as applied in consumer products and food industry (UC=9) and 'conductive agent' as applied in fuel cells (UC= 12).

Table 2.2 Main use categories of NaOH in Western European countries (Euro Chlor, 2004b)

Application	IC	UC	Quantity used (x1000 tonnes/year)	Percentage of total use
Production of chemicals (organic)	2/3		2,72	28%
Production of chemicals (inorganic)	2/3		1.48	16%
Pulp and paper industry	12		1.17	12%
Aluminium and metal industry	8		0.58	6%
Food industry	0		0.29	3%
Water treatment	-	40	0.29	3%

Table 2.2 continued overleaf

Table 2.2 continued Main use categories of NaOH in Western European countries (Euro Chlor, 2004b)

Application	IC	UC	Quantity used (x1000 tonnes/year)	Percentage of total use
Textiles	13		0.29	3%
Consumer products (soap, detergents)	5		1.07	11%
Other	0		1.65	17%
Total			9.72	100%

2.2.1.1 Production of chemicals

NaOH is used for the production of organic and inorganic chemicals which end up in a broad variety of end products (Euro Chlor, 2004b).

2.2.1.2 Production and whitening of paper pulp

The major applications of NaOH in the paper and pulp industry are pH regulation, pulping, bleaching reactant, cleaning agent, water treatment for steam production and demineralisation (Euro Chlor, 2005). Paper and pulp mills produce acid effluents and NaOH is used in waste water treatment for neutralisation, for example of strongly acidic condensate from evaporation of spent liquor. No surplus NaOH is discharged to the WWTP and/or in the receiving water (Euro Chlor, 2005). Other examples of pulp and paper processes using NaOH are:

- Kraft pulping, which is full chemical pulping with NaOH and Na_2S, pH above 12, 160-180^{o}C, 800 kPa (120 psi), 0.5-3 hours.
- The so-called extended delignification, which are techniques to remove more lignin prior to bleaching. NaOH and heat act to break complex bonds in the lignin to make them soluble in water or volatile. NaOH and heat also break bonds in the cellulose reducing strength and yield.
- The bleaching process in the so-called alkali extraction where the organic acids and alcohols react with the NaOH to form organic sodium compounds and water. These organic substances dissolve in water. Here NaOH is used to create a high pH to optimise the bleaching process. NaOH is not the bleaching agent. The purpose of the bleaching is to remove lignin without damaging the cellulose.
- Waste paper recycling: adding water, NaOH, and heat repulps recycled material. The pulp is then used to make a finished paper product on a paper machine in the same manner as in a virgin paper mill.

2.2.1.3 Production of aluminium and other metals

Caustic soda is used in the treatment of bauxite, from which alumina, the basis of aluminium, is extracted. Aluminium has become the second largest metal used in the world, which has led to a large increase in caustic soda consumption in this segment. In the stage of surface

treatment of aluminium finished products, caustic soda is used for pickling (Euro Chlor, 2000a).

2.2.1.4 Food industry

Caustic soda can be used for a large number of applications in the food industry.

In the food production sector, caustic soda is regularly used for (Euro Chlor, 2000a):

- washing and cleaning of bottles, processes and equipment;
- chemical peeling/shelling of fruits and vegetables;
- modification of starch;
- preparation of carboxyl methyl cellulose;
- preparation of salts such as sodium citrate and sodium acetate.

2.2.1.5 Water treatment

Caustic soda is widely used in the treatment of water. In sewage treatment stations, caustic soda allows the neutralisation of effluent and a reduction in the hardness of water. In industry, caustic soda allows the regeneration of ion exchange resins. NaOH is currently used in water treatment with various objectives (Euro Chlor, 2004a, 2005):

- control of the water hardness;
- regulation of the pH of water;
- neutralisation of effluent before the water is discharged;
- regeneration of ion exchange resins;
- elimination of heavy metal ions by precipitation.

NaOH is also used for the cleaning of combustion or incineration flues. Among the technologies used, the washing of gases in a scrubber using alkaline solutions is a process offered by a large number of engineering companies. The concentrations of NaOH solutions used vary according to the application, the level of performance to be achieved, financial situation, etc. The level of scrubbing performance of this technology allows reductions in acid components (HCl, SO_2, etc.) and in heavy metals (Hg, Cd, etc.) to comply with the requirements of international and national standards (Euro Chlor, 2004a, 2005).

2.2.1.6 Textiles

Besides natural materials such as wool, cotton or linen, synthetic fibres are extensively used by the textile industry. Cellulose textiles, obtained by the viscose process (rayon, spun rayon) have a significant market share. At present (2004) annual world production of cellulose textiles easily exceeds 3 million tonnes. Their manufacture consumes considerable tonnages of caustic soda, were 600 kg of caustic soda is needed to produce a tonne of cellulose fibres.

The function of NaOH in the production of cellulose is unknown. NaOH is also used as general processing aid such as neutralisation (Euro Chlor, 2004a, 2005).

2.2.1.7 Other uses

NaOH is further applied in various other industrial sectors such as in production of soaps, mineral oils, bleach, phosphates, cellulose and rubber (Euro Chlor, 2004a, 2005). In most of these applications NaOH also serves as a process aid, such as neutralisation. In the public domain the major applications of NaOH are as oven cleaner and as drain deblocker.

2.3 TRENDS

The global demand for NaOH is expected to grow with 3.1% per year (CMAI, 2000).

2.4 LEGISLATIVE CONTROLS

2.4.1 EU Legislation

There are several EU Directives with quality standards for surface waters, aimed at the protection of human health or aquatic wildlife, depending on the function of the surface water. These EU Directives include a quality standard for the pH, see below:

- Council Directive 98/83/EC of 3 November 1998 concerning the quality of surface water intended for human consumption: $6.5 \leq pH \leq 9.5$;
- Council Directive 75/440/EEC of 16 June 1975 concerning the quality of surface water intended for the abstraction of drinking water: $5.5 \leq pH \leq 9.0$;
- Council Directive (76/160/EEC) of 8 December 1975 concerning the quality of bathing water: $6.0 \leq pH \leq 9.0$;
- Council Directive (78/659/EEC) of 18 July 1978 concerning the quality of fresh waters needing protection or improvement in order to support fish life: $6.0 \leq pH \leq 9.0$;
- Council Directive (79/923/EEC) of 30 October 1979 on the quality required of shellfish waters: $7.0 \leq pH \leq 9.0$.

These EU Directives should have been implemented in national legislation of the ‘old’ EU Member States or be implemented in the near future in the ‘new’ EU Member States.

2.4.2 National Legislation

The Member States were asked to provide (additional) data on national legislation for pH control of waste waters that are discharged to STPs or receiving waters. Information on legislation was received from 16 EU Member States (Sweden, Italy, Estonia, Finland, Greece, Hungary, Lithuania, Slovakia, Slovenia, United Kingdom, Poland, Spain, Belgium-Flanders region, France, Portugal and the Netherlands) which can be considered to be representative for the whole region. The findings are summarised below.

Sweden

In Sweden each public STP sets up its own pH-limits for the waste water it will accept, typically limits are set to between 6.5 and 11. This limit is primarily to avoid damage to materials. Some set a longer-term value of less than 10.5 to avoid a decrease in efficiency of the plant. Generally the pH of the waste water is not an issue. For industrial waste water treatment plants only the pH of the discharged water is regulated by authorities (unless it can be a worker protection or similar issue). The pH limits of the water discharged is set in the environmental permits for each plant (public and industrial), and is set on a case-by-case basis, taking into account quantities, local conditions etc.

Italy

In Italy the following Decree is in place: Decreto Legislativo 18 agosto 2000, n. 258 "Disposizioni correttive ed integrative del decreto legislativo 11 maggio 1999, n. 152, in materia di tutela delle acque dall'inquinamento, a norma dell'articolo 1, comma 4, della legge 24 aprile 1998, n. 128" published in the “Gazzetta Ufficiale n. 218 del 18 settembre 2000 - Supplemento ordinario n. 153”. In Table 3 of Annex 5 of this Decree emission limit values for discharge in surface water and in sewage for pH is reported in the range of 5.5-9.5. In Table 4 of the same Annex are reported the emission limit values for waste waters to be used in soil (i.e. agriculture) and the range for pH is 6-8.

Estonia

The pH control of waste waters discharged to the STP in Estonia is regulated by regulations of the city governments. According to the Water Act (Passed 11 May 1994; RT1 I 1996, 40, 655; consolidated text RT I 1998, 13, 241), which entered into force on the 16th of June 1994: Effluent may be discharged into a water body pursuant to the procedure established by the Government of the Republic, which shall contain the requirements for discharging effluent into water bodies and the measures for checking that these requirements are met. That regulation is "Heitvee veekogusse või pinnasesse juhtimise kord" and there is limit value for pH 6.0-9.0, but local city governments can impose more strict requirements for waste waters discharged to STP. According to the regulation of the Tallinn City Government, the pH limit values are 6.5-8.5.

Finland

In Finland there are no general legislative norms for the pH of waste waters discharged to STP or receiving waters. However, paragraphs 3, 4 and 42 of the Finnish Environmental Protection Act sets some general rules to be followed in environmental permits (free form translation): Harmful environmental effects must be prevented in advance or unavoidable harmful effects are to be limited to a minimum (paragraph 3). Best available techniques should be applied (paragraph 4). An environmental permit can only be granted if significant environmental changes in water bodies are prevented (paragraph 42).

Greece

In Greece there is no consolidated national legislation on pH control of waste waters that are discharged to STP or receiving waters. The relevant legislation implemented is based on regional administrative decisions.

Hungary

According to the Decree No 28/2004 (December 25) of KvVM (Hungarian Ministry of Environment and Water) the lower and upper pH limit of sewage water released indirectly to living water (channel) are 6.5 and 10. The appropriate values in case of direct release are 6.0 and 9.5.

Lithuania

Environmental Requirements for the Waste Water Control were adopted on 5 October 2001 by the order No 495 of the Minister of Environment, transposing provisions of Council Directive 91/271/EEC concerning urban waste water treatment and Commission Directive 98/15/EC amending Council Directive 91/271/EEC.

Slovakia

In Slovakia pH of waste waters discharged to STPs or receiving waters must be in the range of 6 to 9. This applies for discharge from all industrial plants including chemical industry. The above pH range is stipulated in the Government Ordinance no. 491/2002 which specifies pH limits for waste waters in the Slovak Republic.

Slovenia

Slovenia has a national legislation on pH control of waste waters that are discharged to STP or receiving waters. There is an obligation to measure pH according to method

SIST ISO 10520. Special regulations are in order for specific sectors (municipal STP, metal industry, textile industry and about 45 other different sectors). The pH limit value for discharge in water is 6.5-9.0 and for STP 6.5-9.5.

United Kingdom

In the UK, environmental quality standards are set for receiving waters, which are used to calculate the permitted pH of any discharges. Most of the quality standards are driven by the EU legislation mentioned in Section 2.4.1 of this risk assessment. In the UK, each individual water company is responsible for regulating the quality of discharges to their STPs. There is no national legislation which governs this.

Poland

In Poland there is a Regulation from the Ministry of Infrastructure that regulates parameters of industrial wastewaters, one of them is the pH. Industrial waste supplied to the sewer should have a pH between 6.5 and 9.5.

Spain

Data on pH from authorisations of discharges issued by the ‘Confederaciones Hidrograficas’ show that the pH in these authorisations range from 5 to 9-9.5. The control stations from ICA (Red Integrada de Calidad de las Aquas) has mapped pH values of discharges and concluded that values are mainly between 6.5 and 8.5, fulfilling the legislation on water quality. Only some point sources are identified that have pH values lower than 5.5, for which the basin of river Odiel and river Guadiana are mentioned.

Belgium – Region of Flanders

In Belgium the pH of wastewater discharges is a competence of the 3 regions Flanders, Brussels and Wallonia. Only information from Flanders was received. Companies located in the Flanders region are liable to the dispositions of the so-called sectoral environmental conditions for classified establishments prescribed by the Vlarem (Flemish environmental regulation). Dependent on the type of industrial activity (e.g. chemical industry in the case of NaOH production), this regulation requires that the pH in the effluent corresponds to a certain range (in general pH between 6 and 9.5 prior to discharge into the sewer or river system (VMM, personal communication, 2005).

France

Arr. 02/02/1998, art. 31: the pH of wastewater coming from ICPE (French industrial installation classified for the protection of the environment) that are emitted to the aquatic environment should range between 5.5 and 8.5 (9.5 in case of alkaline neutralisation with lime). This is the general case but exceptions exist.

A specific pH range should be maintained in specific receiving waters:

- For water bodies where fish farming exists and for water where swimming is authorised: between 6 and 9;
- For water bodies that are used for potable water production: between 6.5 and 8.5;
- For water bodies where shellfish farming occurs: between 7 and 9.

Portugal

The Portuguese legislation for pH control of waste water discharged to STPs or its receiving water establishes that the pH Emission Limit Value must be in the range of 6.0 – 9.0.

This pH range is stipulated in Annex XVIII of Decree-law nr. 236/98 published in "Diário da República" nr. 176/98 of 1998/08/01.

The Netherlands

All EU Directives mentioned in Section 2.4.1 have been implemented in national legislation and published in ‘het Staatblad van het Koninkrijk der Nederlanden’ (Bulletion of Acts and Decrees) (VROM, 2001). In the Netherlands the pH limits for surface water for freshwater fish and shellfish have been set at $6.5 \leq pH \leq 9.0$ and $7.5 \leq pH \leq 9.0$, respectively. For (industrial) point sources there are no specific regulations for the discharge of NaOH, but there are regulations for pH limits and the total salt content of waste water (effluent) discharges. Both for direct emissions to surface waters and indirect emissions to municipal waste water treatment plants the pH of the effluents should normally be in the range of 6.5-9.0. In individual cases a pH value up to 10 is accepted, depending on the receiving surface water or municipal waste water treatment plant. These regulations are included in the licences that are needed in the framework of the ‘Wet Milieubeheer’ (Environmental Management Act) and the ‘Wet Verontreiniging Oppervlaktewater’ (Surface Water Pollution Act). The above data were provided by the Dutch Institute for Inland Water Management and Waste Water Treatment (RIZA), Ministry of Transport, Public Works and Watermanagement.

2.4.3 Conclusions

Taking into account the existing EU Directives for pH control for surface water (see Section 2.4.1) and the data of many Member States on additional national regulations to control the pH of waste waters (STP influents) and surface waters it is concluded that STPs and surface waters are sufficiently protected with regard to pH changes.

3 ENVIRONMENT

3.1 ENVIRONMENTAL EXPOSURE

3.1.1 Introduction

As stated in the general introduction of this report, the targeted risk assessment for the environment will focus solely on the aquatic environment, when applicable including STPs/WWTPs, as the emissions of NaOH in the different life-cycle stages (production and use) mainly apply to (waste) water. The aquatic effect and risk assessment will only deal with the effect on organisms/ecosystems due to possible pH changes related to OH^- discharges, as the toxicity of the Na^+ ion is expected to be insignificant compared to the (potential) pH effect. Only the local scale will be addressed, including sewage treatment plants (STPs) or waste water treatment plants (WWTPs) when applicable, both for production and industrial use. Any effects that might occur would be expected to take place on a local scale and therefore it was decided as not meaningful to include the regional or continental scale in this targeted risk assessment. Furthermore, the high water solubility and very low vapour pressure indicate that NaOH will be found predominantly in water. Significant emissions to air are not expected due to the very low vapour pressure of NaOH. Significant emissions to the terrestrial environment are not expected either. The sludge application route is not relevant for the emission to agricultural soil, as no sorption of NaOH to particulate matter will occur in STPs/WWTPs. Additionally it should be realised that any waste water from NaOH production sites is coming from the salt electrolyses and is an inorganic wastewater stream and for this reason it is not feasible to treat it biologically. Therefore wastewater streams from NaOH production sites will normally not be treated in biological waste water treatment plants (WWTPs). NaOH may be used, however, for pH control of acid wastewater streams that are treated in biological WWTP's (Dr. D. van Wijk, Euro Chlor, personal communication, 2004).

The exposure assessment for the aquatic environment will only deal with the possible pH changes in STP effluent and surface water related to the OH^- discharges at the local scale.

3.1.2 Environmental releases

The emissions of NaOH during production and use mainly apply to the aquatic environment. For sodium, other anthropogenic sources are for instance mining and the use of road salt (sodium chloride). In water (including pore water of sediment and soil), NaOH dissociates into the sodium ion (Na^+) and hydroxyl ion (OH^-), both having a wide natural occurrence. See Section 3.1.4.2 for data on the measured concentrations of the hydroxyl ion, i.e. pH measurements.

3.1.2.1 Releases from production

The production of NaOH can potentially result in an aquatic emission and locally increase the sodium concentration and pH in the aquatic environment. When the pH is not neutralised, the discharge of effluent from NaOH production sites may cause an increase in pH in the

receiving water. The pH of effluents is normally measured very frequently and can be neutralised easily. The production capacity and the applied processes for all sites in the EU are presented in **Table 2.1**.

Since the exposure assessment focussed on possible pH changes in the local aquatic environment, industry submitted actual data on pH values in effluent and receiving surface waters at NaOH production sites, based on the results of a questionnaire that was sent to a broad cross section of NaOH producers in the EU via Euro Chlor, representing 97% of NaOH production capacity in the enlarged Europe (Euro Chlor, 2004b). The results of this questionnaire (Euro Chlor, 2004c) provided effluent and receiving water data for 43 out of 84 production sites. The sites are anonymised by numbers which do not correspond to the numbers in **Table 2.1**. The 43 responding production sites are from 15 different EU countries with a wide geographical spread. The respondents include 34 sites in the old EU member states, 6 sites in the new EU member states, 2 in Norway and 1 in Switzerland (Euro Chlor, 2004c). The three major NaOH production processes, i.e. membrane, diaphragm and mercury process, were well represented among the respondents to the questionnaire. The production capacities of the sites that responded represented a very broad range from several tens of ktonne/year up to several hundreds of ktonne/year (Dr. D. van Wijk, Euro Chlor, personal communication, 2004).

The questionnaire revealed that 11 sites do not have effluents which are discharged to the environment. On these specific sites the waste water can be completely recycled due to the specific on-site process conditions. The results also showed that out of 43 sites reporting, 31 sites neutralize their effluent before discharging into the receiving water. A total of 32 sites reported to be legally obliged to neutralize their effluent and 6 sites, which do not have effluents, did not respond to this question. However, 5 sites reported not to have such legal requirements, while 2 of these 5 sites reported actually to neutralize their effluents. One site (site 30) reports a legal obligation to neutralize but they do not adapt their pH, because the pH range of their effluent is already within a narrow range close to neutral.

According to Euro Chlor (2004c), many sites reported pH values for wastewater sub-streams instead of final effluents, even though they are included as ‘effluent data’ This was concluded from the fact that many sites reported broad ranges of pH values, but also indicated that final effluents were neutralised before discharging and from some subsequent checks with respondents. Substreams are normally combined with other wastewater sub-streams on the site before they are finally discharged into the receiving water. Therefore, the first two columns of **Table 3.1** on effluents are the most important with respect to the potential pH effect on receiving waters.

A total number of 36 production sites, including 2 sites (no. 17 and 30) that do not discharge their effluent into the environment, i.e. sewer or receiving water, reported measured pH values of the effluent. Of these 36 sites, 19 sites reported pH values within the range of 6-9 (range of lowest pH to highest pH), 7 sites reported pH values within the range of 5-10 and 10 sites reported pH values outside the range of 5-10. Most importantly, all but one of the sites that discharge effluent into the environment reported to neutralise their effluent before discharge. Only one site (no. 15) reporting a very wide effluent pH rang of 3.0-11.6 and stated not to neutralise its effluent before discharge. After contacting this site it became clear that the reported pH values for this site represented measurements in the wastewater sub-stream, immediately after leaving the production unit. Depending on the process conditions this can have the listed extreme values, which reportedly only last for 10-15 minutes because the wastewater sub-stream is then combined with other wastewater sub-streams at the site and the pH becomes circumneutral (Dr. D. van Wijk, Euro Chlor, personal communication).

Thereafter the final effluent (i.e. the combined wastewater sub-streams) enters a municipal sewage treatment plant before it is discharged into the receiving water. This site is not legally obliged to neutralise its effluent before discharge (Euro Chlor, 2004c). As all further sites that reported a high difference between the lowest and highest pH of the effluent also reported to neutralise their effluent, it can be assumed that for these sites the pH values are also for wastewater sub-streams (that are combined with other sub-streams before neutralisation of the final effluent) and not for the final effluents that are discharged into the environment.

The results from the questionnaire, reported for 43 out of 84 production sites, demonstrate that the pH of waste water discharges is controlled and that almost always proper regulations are in place.

Table 3.1 Effluent and receiving water data for NaOH producers in the EU (Euro Chlor, 2004c)

	Effluent data										Receiving water data						
No.	Effluent discharged in the Environment	Neutra-lization before Discharge	Obligation of Neutra-lization	Continuous Measurement of the pH	pH (avg.)	Lowest pH	Highest pH	Alka-linity (meq/l)	Flow rate avg. (m^3/d)	Flow rate range (m^3/d)	Type of receiving water	Continuous Measurement of the pH	Lowest pH	Highest pH	Alka-linity (meq/l)	Flow rate avg. (m^3/d)	Flow rate range (m^3/d)
2	Yes	Yes	Yes	Yes	11.8	3.8	13.9		78		River	Yes	7	8.2			
3	Yes	Yes	Yes	Yes	7.3	6.9	7.9	NA	6,500	5,500 – 8,000	River	Yes	7.6	8.4	NA	1,000,000	260,000 – 5,000,000
15	Yes	No	No	Yes	7.62	3.01	11.55	2.22	10,240	6,010 – 17,280	River	No	7.1	7.96	NA	25,532,064	4,855,680-?
16	Yes	Yes	Yes	Yes	7.3	7	7.9	1.87	30,606	18,000-41,096	River	No	7.3	7.8	2.6	5,356,800	1,468,800-12,700,800
17	No	Yes	Yes	Yes	7.25	7	7.5	NA	26,300	NA	river	No	7.7	7.7	NA	10,972,800	NA
18	Yes	Yes	Yes	Yes	7.9	3.9	13.2		1,800	1,000	River	No				1,978,584	15,000,000
20	Yes	Yes	Yes	Yes	7.5	7	8.5	NA	173,000	150,000-200,000	River	No	6.5	8.2	NA	8,208,000	483,840-65,577,600
21	Yes	Yes	No	Yes	12	10	13	NA	10	8 - 15	River	No	7.0	7.8	3 - 4	172,800,000	60,480,000-864,000,000
22	Yes	Yes	No	Yes	3	2	4	NA	4,560	3,240-5,640	Sea						
25	No	No	No	No							Estuary	No					
26	Yes	Yes	Yes	Yes	7 - 7.5	6	8.5	3.5	9,600	9,600-12,000	River	No	8	8.2	2.8	400	400-600
29	Yes	Yes	Yes	Yes	7.2	6.1	9.4		178	67 - 602	River	No					
30	No	No	Yes	Yes	7.9	7.5	8.2	NA	5,842	max. 6,000	River	No	6.9	8	NA	3,456,000	NA
32	Yes	Yes	Yes	Yes	7.2	7	7.8		48,000	45,000 – 55,000	River	No	7.1	7.5		100,000,000	60,000,000 – 150,000,000
33	Yes	Yes	Yes	Yes	7.8	6.5	8.5	1.004	17,461	12,692-21,928	River	No	7.5	8.1	3.567	475,200	95,040-1,080,000
34	Yes	Yes	Yes	Yes	6.7	5	10	NA	3,600	2,400-6,000	Sea	No	6.7	6.7	NA		
35	Yes	Yes	Yes	Yes	5	3	11	NA	114	46-520	Sea	No	7.8	7.8	NA		
37	Yes	Yes	Yes	Yes	7.7	6.7	8.5		600	300	River	No	8	8		2,500,000	?-5,200,000
39	Yes	Yes	Yes	Yes	12	4	13	NA	300	150-400	Sea	Yes	6.5	8.0	NA	25,920,000	12,960,000-34,560,000
40	Yes	Yes	Yes	Yes	7.4	6.6	8.2		25,000	20,000-30,000	River	No					
41	Yes	Yes	Yes	Yes	8	7	9	NA	4,800	4,600-4,900	Sea	No	NA	NA	NA	NA	NA
46	Yes	Yes	Yes	Yes	7.5	6.6	8.5	NA	134	NA	Other	Yes	4.5	10	NA	301	
49	Yes	Yes	Yes	Yes	7.28	7.09	7.48	NA	853	634-1,170	Estuary	No	6.8	8	NA	1,000,000	

Table 3.1 continued overleaf

Table 3.1 continued Effluent and receiving water data for NaOH producers in the EU (Euro Chlor, 2004c)

	Effluent data										Receiving water data						
No.	Effluent discharged in the Environment	Neutra-lization before Discharge	Obligation of Neutra-lization	Continuous Measurement of the pH	pH (avg.)	Lowest pH	Highest pH	Alka-linity (meq/l)	Flow rate avg. (m^3/d)	Flow rate range (m^3/d)	Type of receiving water	Continuous Measurement of the pH	Lowest pH	Highest pH	Alka-linity (meq/l)	Flow rate avg. (m^3/d)	Flow rate range (m^3/d)
51	Yes	Yes	Yes	Yes	8.2	6.9	8.9	6	728	660-790	River	Yes	7.6	7.9	3	51,000,000	25,000,000-70,000,000
52	Yes	Yes	Yes	Yes	8	4	10		9.4	0-55	River	Yes	6.5	9		14,077	14,965-20,612
53	No																
54	No																
58	Yes	Yes	Yes	Yes	11.5	11	12	3.10^{-3}	4,000	3,500-4,500	River	Yes	7.5	8.5		174,744	127,744-221,744
60	Yes	Yes	Yes	Yes	7.9	7	8.4	1.3	14,097	11,000-17,000	River	No	7.63	8.19	4.05	1,309,589	140,832 – 27,734,400
61	Yes	Yes	Yes	Yes	6-8	6	8	NA	16,344	NA	River	Yes	6.9	7.2	NA	17,460	8,000-36,000
64	No			Yes													
65	No			Yes													
66	No			Yes													
68	Yes	Yes	Yes	Yes	7	6.9	7.3	NA	374,000	245,000 – 500,000	River	Yes	7.7	8.1	NA	96,768,000	30,240,000-259,200,000
69	Yes	Yes	Yes	Yes	7.5	5.5	8.5	92	3,500	5,000	Sea	No	NA	NA	75		
70	No			Yes	7.4/7.8	6.2 / 6.8	8.4 / 9.4		48,312 / 4,032	25,320 / 4,368	River	No	7.5	8.1		3,456,000	?-7,948,800
71	Yes	Yes	Yes	Yes	7.5	6	9		4,500	4,000-6,000	Sea	Yes	NA	NA	NA		
72	Yes	Yes	Yes	Yes	7.3	3	9.2	NA	23,000	15-35 000	River	No	NA	NA	NA	450,000	300,000-?
79	Yes	Yes	Yes	Yes	7	6	9		330	180 - 460	Other	No	7.2	7.4			
80	No	No	No														
83	No	Yes	Yes	Yes	7.8	6.4	9.4		2,112	1,183-7,966	Other	Yes	7.2	8.7			
84	Yes	Yes	Yes	Yes	10	6.5	11	30	1,300	600 – 2,000	Other	Yes	6.9	7.7	5		
85	Yes	Yes	Yes	Yes	6.6	5.4	9.7	NA	1,900		Lake	No	4.2	9.2	NA		

NA Not available

3.1.2.2 Releases from use

To estimate the environmental releases from the uses of NaOH a questionnaire was organised by Euro Chlor, in cooperation with the Portuguese and Dutch authorities, focussing on the major downstream uses. Because the exposure assessment focussed on possible pH changes in the local aquatic environment, data were requested on the pH control at user sites. Based on the experience with the results from the questionnaire to producers (see Section 3.1.2.1) it was envisaged that the pH of discharges would also be strictly controlled by the industry involved, often in response to local requirements. Therefore, the environmental part of the questionnaire was simplified in agreement with the rapporteur, asking the following two questions: 'does your final waste water which is discharged to the receiving water still contain NaOH ?' and 'if yes: what do you do to prevent an impact from NaOH discharge'? The results of the user questionnaire have been reported in detail in Euro Chlor (2005).

The paper and pulp industry was addressed via CEPI, the Confederation of the European Paper Industries and received 34 replies. For the paper and pulp industry one questionnaire was received from Germany (National Federation), which represented the common practice in this country.

Other industries were approached via five large producers of NaOH who each sent out a questionnaire to 20 of their customers, in nearly all cases end users of NaOH. A reply had been received from 24 customers, representing a response of 24%. From these 24 customers, 8 responses were received from Spain. The other customers were located in Belgium, France, Germany, The Netherlands and United Kingdom. The majority originated from the chemical industry (17 replies). One reply was received from the steel industry, textile industry, rubber production, distribution, food industry, metal industry and aluminium industry. In one case a distributor completed the questionnaire, which is not an end user of NaOH.

For the pulp and paper industry the average amount of NaOH used per day was 14 tonnes (range from 0.005 – 160 tonnes), while the remaining end users used an average amount of 24 tonnes/day (range from 1.5 – 110 tonnes). For the paper and pulp sector 32 respondents answered that the final wastewater did not contain NaOH, but in two cases it did. For these cases it was stated that the impact was controlled. For the 23 other end users questioned (excluding the distributor), 21 indicated to have no NaOH in the final effluent. For two sites, from the chemical industry, the final effluent contained NaOH. For these sites it is not specifically known if they neutralised their effluent. Normally, local procedures are in place to prevent discharges outside the range required by authorities, such as recycling, mixing with other streams for neutralisation or discharge to a WWTP when that is considered favourable.

The results from the questionnaires for the use sites demonstrate that in most cases the final effluents did not contain NaOH anymore. Usually, the pH of waste water discharges is controlled and almost always proper regulations are in place. Nevertheless, for some use sites, emitting their effluents to the environment, it cannot be excluded that they do not neutralise their effluents and have no legal obligation to neutralise.

3.1.3 Environmental fate and distribution

As stated in Section 3.1, the emissions of NaOH mainly apply to (waste) water. Furthermore, the high water solubility and very low vapour pressure indicate that NaOH will be found predominantly in water. In water (including soil or sediment pore water), NaOH is present as the sodium ion (Na^+) and hydroxyl ion (OH^-), as solid NaOH rapidly dissolves and subsequently dissociates in water.

3.1.3.1 Sewage treatment plants (STPs)

If emitted to waste water that is treated in a biological STP or WWTP, virtually the total amount will end up in the effluent, as sorption to the STP sludge and volatilisation will be negligible.

3.1.3.2 Aquatic compartment

If emitted to surface water, sorption to particulate matter and sediment will be negligible. An addition of NaOH to surface water may increase the pH, depending on the buffer capacity of the water. The higher the buffer capacity of the water, the lower the effect on pH will be. In general the buffer capacity preventing shifts in acidity or alkalinity in natural waters is regulated by the equilibrium between carbon dioxide (CO_2), the bicarbonate ion (HCO_3^-) and the carbonate ion (CO_3^{2-}):

$$CO_2 + H_2O \leftrightarrow HCO_3^- + H^+ \qquad (pK_{a1} = 6.35)$$
$$HCO_3^- \leftrightarrow CO_3^{2-} + H^+ \qquad (pK_{a2} = 10.33)$$

If the pH is < 6, un-ionised CO_2 is the predominant species and the first equilibrium reaction is most important for the buffer capacity. At pH values of 6-10 the bicarbonate ion (HCO_3^-) is the predominant species and at pH values > 10 the carbonate ion (CO_3^{2-}) is the predominant species. In the majority of natural waters the pH values are between 6 and 10, thus the bicarbonate concentration and the second equilibrium reaction are most important for the buffer capacity (Rand, 1995; De Groot and Van Dijk, 2002; OECD, 2002). UNEP (1995) reported the bicarbonate concentration for a total number of 77 rivers in North-America, South-America, Asia, Africa, Europe and Oceania. The 10^{th}–percentile, mean and 90^{th}-percentile concentrations were 20, 106 and 195 mg/l, respectively (OECD, 2002).

To underline the importance of the bicarbonate concentration for the buffer capacity in natural waters, **Table 3.2** summarises the concentration of NaOH needed to increase the pH from an initial pH of 8.25-8.35 to a value of 9.0, 10.0, 11.0 and 12.0 at different bicarbonate concentrations. The data of **Table 3.2** are based on calculations but were confirmed by experimental titrations of bicarbonate (HCO_3^-) concentrations of 20, 106 and 195 mg/l, respectively, in purified water. The difference between the calculated and measured NaOH concentration needed to obtain a certain pH value was always $\leq$ 30% (De Groot and Van Dijk, 2002; OECD, 2002). The data in **Table 3.2** for distilled water are from OECD (2002).

The alkalinity, defined as the acid-neutralising (i.e. proton accepting) capacity of the water, thus the quality and quantity of constituents in water that result in a shift in the pH toward the alkaline site of neutrality, is determined for > 99% by the concentrations of bicarbonate (HCO_3^-), carbonate (CO_3^{2-}) and hydroxide (OH^-) (Rand, 1995), with bicarbonate being the predominant species at pH values in the range of 6-10 (see also above). Hydroxide is only relevant in alkaline waters. Thus, the data in **Table 3.2** are useful to estimate pH increases in natural waters (most of them having a pH value of 7-8), if data on NaOH additions and bicarbonate concentrations are available. The alkalinity is determined from acid/base titration or can be calculated from the calcium concentration, as follows (De Schampelaere et al., 2003; Heijerick et al., 2003):

$$\text{Log (alkalinity in eq/l)} = - 0.2877 + 0.8038 \text{ Log (Ca in eq/l)}$$

Table 3.2 Concentration of NaOH (mg/l) needed to increase the pH to values of 9.0, 10.0, 11.0 and 12.0 (De Groot and Van Dijk, 2002; OECD, 2002)

Buffer capacity [1]	Final pH			
	9.0	10.0	11.0	12.0
0 mg/l HCO_3^- (distilled water)	0.4	4.0	40	400
20 mg/l HCO_3^- (10^{th} percentile of 77 rivers)	1.0	8.2	51	413
106 mg/l HCO_3^- (mean value of 77 rivers)	3.5	26	97	468
195 mg/l HCO_3^- (90^{th} percentile of 77 rivers)	6.1	45	145	525

1) The initial pH of a bicarbonate solution with a concentration of 20-195 mg/l was 8.25-8.35

3.1.3.3 Terrestrial compartment

The terrestrial compartment is not included in this targeted risk assessment, because it is not considered relevant for NaOH. With respect to the fate of NaOH in soil the following information is available. If emitted to soil, sorption to soil particles will be negligible. Depending on the buffer capacity of the soil, OH^- will be neutralised in the soil pore water or the pH may increase.

3.1.3.4 Atmospheric compartment

The air compartment is not included in this targeted risk assessment because it is considered not relevant for NaOH. With respect to the fate of NaOH in air the the following information is available. If emitted to air as an aerosol in water, NaOH will be rapidly neutralised as a result of its reaction with CO_2 (or other acids), as follows:

$$NaOH + CO_2 \rightarrow HCO_3^- + Na^+$$

Subsequently, the salts (e.g. sodium(bi)carbonate) will be washed out from the air (US EPA, 1989; OECD, 2002). Thus, atmospheric emissions of neutralised NaOH will largely end up in soil and water. Based on a NaOH concentration of 50% in the aerosol droplets, the atmospheric half-life of NaOH was estimated at 13 seconds. Based on model calculations, this degradation rate results in only 0.4% of the NaOH emitted to air remaining in the air at a point 200 metres from the emission point (U.S. EPA, 1988; 1989).

3.1.3.5 Accumulation

Bioaccumulation in organisms is not relevant for NaOH. Based on this, there is no need to perform a risk assessment for secondary poisoning.

3.1.4 Aquatic compartment (effluent and surface water)

3.1.4.1 Predicted environmental concentrations

Not applicable. The risk assessment is based on measured pH changes at the local scale from discharges from production and use, see Sections 3.1.2.1 and 3.1.2.2.

3.1.4.2 Measured levels in water

3.1.4.2.1 Freshwater (surface waters)

The concentration of hydroxyl ions (OH^-) in the environment has been determined very extensively via pH measurements. Geochemical, hydrological and/or biological processes mainly determine the pH of an aquatic ecosystem. The pH is an important parameter of aquatic ecosystems and it is a standard parameter of water quality monitoring programs. The most important freshwater aquatic ecosystems of the world revealed average annual pH values between 6.5 and 8.3 but lower and higher values have been measured in other aquatic ecosystems. In aquatic ecosystems with dissolved organic acids a pH of less than 4.0 has been measured, while in waters with a high chlorophyll content the bicarbonate assimilation can result in pH values of higher than 9.0 at midday (OECD, 2002, from UNEP 1995).

Also sodium (Na^+) has been measured extensively in freshwater aquatic ecosystems. For example, the 10^{th}-percentile, mean and 90^{th}-percentile concentrations for a total number of 75 rivers in North-America, South-America, Asia, Africa, Europe and Oceania were 1.5, 28 and 68 mg/l, respectively (OECD, 2002, from UNEP, 1995).

For European freshwaters, there are extensive databases on physico-chemical properties, including pH, hardness (calculated from the measured calcium and magnesium concentration), alkalinity (determined by acid/base titration or calculated from the calcium concentration, see further Section 3.1.3.2) and sodium concentration. In the framework of the EU Risk Asessment Report on Zn Metal (The Netherlands, 2004), data on physico-chemical properties of freshwaters in individual European countries and the combined data for freshwaters in European countries were collected and reported by De Schampelaere et al. (2003) and Heijerick et al. (2003). The combined European data for the above physico-chemical properties, all relevant for pH changes, are summarised in **Table 3.3**. The data in this table are based on 1991-1996 data for 411 European locations, extracted from the 'GEMS/Water database' (Global Environmental Monitoring System) that is mainly aimed on the large river systems. A correlation analysis on the data from all 411 locations indicate that all parameters listed in **Table 3.3** are positively correlated, i.e. an increased pH is associated with increased concentrations of Ca, Mg and Na and increased hardness and alkalinity (De Schampelaere et al., 2003; Heijerick et al., 2003).

The variation in the above physico-chemical properties of the large river systems in different European countries is rather small, with exception of some areas in the Nordic countries (Denmark, Sweden, Norway and Finland) which are characterised by 'soft water' conditions, i.e. a hardness <24 mg $CaCO_3$/l and low pH. For example, in Sweden the 50^{th} percentile value for hardness is 15 mg $CaCO_3$/l, which is 10-times lower than that for whole Europe. In Sweden the 50^{th} percentile value for pH is just below 7, which is about 1 pH unit lower than that for whole Europe (De Schampelaere et al., 2003; Heijerick et al., 2003; The Netherlands, 2004).

Data on pH (and for some sites data on alkalinity) in surface waters, receiving effluent of NaOH producers, are given in **Table 3.1**. In all but 3 of the receiving waters for which pH values are available, the pH values are within the range of 6.5-8.5. These waters include freshwater (rivers) and seawater; each of these waters have a more narrow range of pH values, usually within one pH unit (most waters: pH range of 7.0 to 8.0). Thus, in most receiving waters the pH values are in the range that is expected in most EU waters (see **Table 3.3**). In one river the pH ranged from 6.5-9.0 and in two waters there was an even wider range of pH

values, viz. 4.2-9.2 in a lake and 4.5-10.0 in another, unspecified water type. There is no data on sodium concentrations in the receiving waters at the NaOH production sites (a question on the sodium content was not included in the questionnaire).

Table 3.3 Physico-chemical properties of European freshwaters (De Schampelaere et al., 2003; Heijerick et al., 2003)

Percentile value	pH	Hardness [1] (mg/l, as $CaCO_3$)	Alkalinity (mg/l, as $CaCO_3$)	Ca (mg/l)	Mg (mg/l)	Na (mg/l)
5th percentile	6.9	26	3	8	1.5	3
10th percentile	7.0	41	6	13	2	5
20th percentile	7.2	70	15	23	3	7
30th percentile	7.5	97	31	32	4	10
40th percentile	7.7	126	53	42	5	13
50th percentile	7.8	153	82	51	6	17
60th percentile	7.9	184	119	62	7	22
70th percentile	7.9	216	165	73	8	29
80th percentile	8.0	257	225	86	10	40
90th percentile	8.1	308	306	103	12	63
95th percentile	8.2	353	362	116	15	90

1) Hardness: total hardness, calculated from the Ca and Mg concentration

3.1.4.2.2 Seawater

In over 97% of the seawater in the world, the salinity (the amount of dissolved inorganic constituents), is 35% (promille, in g/kg), but can be lower[5]. The major constituents of seawater at 35 $^0/_{00}$ are Cl^- (19.35 g/kg), Na^+ (10.77 g/kg), SO_4^{2-} (2.71 g/kg), Mg^{2+} (1.29 g/kg), Ca^{2+} (0.41 g/kg), K^+ (0.40 g/kg) and HCO_3^- (0.142 g/kg, being the carbonate alkalinity expressed as though it were all HCO_3^-, as this is the dominant species in seawater; the concentrations of CO_2 and CO_3^{2-} in seawater are very low compared to that of HCO_3^-) (Stumm and Morgan, 1981).

The pH of seawater (ocean water) is normally 8.0-8.3, which is very similar to the 80th to 95th percentile values in European freshwaters (8.0-8.2, **Table 3.3).** The total range of pH values reported for seawater is 7.5-9.5 (Caldeira and Berner (1999) and data from several sources on the internet). The sodium (Na) concentration in seawater (10,770 mg/kg, equivalent to 10,450 mg/l) is 115-times higher than the 95th percentile value in European freshwaters (90 mg/l). The bicarbonate (HCO_3^-) concentration in seawater (142 mg/kg, equivalent to 137 mg/l) is between the mean HCO_3^- concentration (106 mg/l) and the 90th percentile HCO_3^- concentration (195 mg/l) in European freshwaters, indicating a relatively high buffer capacity in seawater. The total hardness of seawater (6,100 mg/l, as $CaCO_3$, calculated from the Ca and Mg concentration) is 17-times higher than the 95th percentile value in EU freshwaters, due to the much higher Ca and especially Mg concentration in seawater compared to freshwater.

5 Commonly used classification of watertypes based on salinity:
seawater: salinity >20 $^0/_{00}$
brackish water: salinity 5-20 $^0/_{00}$
freshwater: salinity <5 $^0/_{00}$

3.1.4.2.3 Effluent of NaOH production sites

The data on pH (and alkalinity) in effluent at NaOH production sites is given in **Table 3.1**. There is no data on sodium concentrations in these effluents (a question on the sodium content was not included in the questionnaire).

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

3.2.1 Aquatic compartment

3.2.1.1 Toxicity to aquatic organisms

3.2.1.1.1 Acute toxicity

The results of single-species acute toxicity tests with NaOH are summarised in **Table 3.4**, based on the data reported in OECD (2002). The data include tests with fish and invertebrates; all but one test were performed with freshwater species. The tests with fish resulted in acute LC_{50} values and toxic/lethal concentrations ranging from 35 to 189 mg/l. The results for invertebrates are very similar, with a range of 33 to 450 mg/l. There is no data for algae and higher aquatic plant species (OECD, 2002). An algal growth test is a 'base set' requirement, but industry (Euro Chlor) submitted a derogation statement that was accepted by the rapporteur.

Additional data on acute toxicity (not listed in Table 3.4)

Concentrations of 20-180 mg/l and 70-180 mg/l were reported to be lethal to various species of fish and invertebrates (crabs, oysters), respectively, after an exposure time varying from 2-10 minutes to 120 hours. Concentrations of 125 to 1,000 mg/l were reported to be lethal to various species of insect larvae (McKee and Wolf, 1963).

The toxicity of NaOH can be ascribed to the pH increase due to the addition of OH^-, as the sodium concentrations are too low to explain the effects. For example, acute toxicity tests with fish *Leuciscus idus melanotus* (golden orfe) resulted in a LC_{50} of 189 mg/l for NaOH (included in **Table 3.4**), while the same test system resulted in a LC_{50} of >10,000 mg/l for NaBr (Juhnke and Lüdemann, 1978). As NaOH, NaBr is highly soluble in water, but aqueous solutions of NaBr have a near neutral pH of 6.5-8.0 (Windholz, 1983). The toxicity of NaOH is depending on the composition of the test waters, especially the buffer capacity of the water, and is further depending on species sensitivity and species life-stage.

3.2.1.1.2 Chronic toxicity

For chronic toxicity of NaOH only one study is available, with fish (guppy) *Lebistes reticulatus* (Rustamova, 1977). Two tests were performed, in which the NaOH solutions were changed daily to maintain a constant pH. The controls contained 'pure' water (no NaOH; no

further data on water characteristics). The data obtained were subjected to statistical analyses, but the data on these analyses were not reported.

In the first test, 1-day to 2-day-old fry were exposed for up to 5 months to NaOH concentrations of 0-25-50-75-100 mg/l. At all concentrations tested, survival, growth, the onset of sexual differentiation, sexual maturation and fecundity were adversely and dose-related affected. Effects were first observed only at 75 and 100 mg/l, but with increasing exposure time effects were also observed at 50 and 25 mg/l.

In the second test (a 3-generation test), mature females of the same age, reared in pure water, were transferred to NaOH concentratons of 25-50-100 mg/l (25 females/treatment) in which they were exposed together with males. The control group remained in the pure water. At all concentrations tested, survival, maturation, fecundity and the quality of the progeny were adversely affected. At 25 mg/l, the percent of females attaining sexual maturity and the numbers of young in the first generation were similar to that in the control, but decreased sharply in the second and third generation. At 25 mg/l, also the quality of the progeny (measured by deformities and early dead) was affected especially in the second and third generation. Data on the pH values in the control and NaOH treatments were not reported.

Although the reported data on this study (Rustamova, 1977) are limited, especially regarding the results of the 3-generation test, the study clearly showed effects on survival, growth and reproduction of fish at long-term exposure to NaOH concentrations of 25 mg/l and higher.

3.2.1.1.3 Conclusion on toxicity to aquatic organisms (single-species tests)

The available data indicate that NaOH concentrations of 20 to 40 mg/l may be acutely toxic to fish and invertebrates. Data on pH increases due to the addition of these amounts of NaOH in the used test waters are lacking. In waters with a relatively low buffering capacity, NaOH concentrations of 20-40 mg//l may result in a pH increase with one to several pH units (see **Table 3.2**).

3.2.1.1.4 Reliability of the aquatic toxicity tests and the need for further testing

OECD (2002) assigned a low code of reliability (‘invalid’ or ‘not assignable’) to all available tests, as in general the tests were not conducted according to the current test guidelines. Furthermore, in many tests reports there were no data on pH, buffer capacity and/or test medium composition, although this is essential information for toxicity tests with NaOH. This is the most important reason why most of the tests were considered ‘invalid’. Despite of this, there is no need for additional aquatic toxicity testing with NaOH, as all available tests resulted in a rather small range of toxicity values (acute toxicity tests: 20 to 450 mg/l; chronic toxicity test: $\geq$ 25 mg/l) and there are sufficient data on the pH ranges that are tolerated by major taxonomic groups.

Moreover, a generic PNEC cannot be derived from single-species toxicity data for NaOH, as the pH of natural waters as well as the buffer capacity of natural waters show considerable differences and aquatic organisms/ecosystems are adapted to these specific natural conditions, resulting in different pH optima and pH ranges that are tolerated (see Section 3.1.4.2). According to OECD (2002) a lot of information is available about the relationship between pH and ecosystem structure and also natural variations in pH of aquatic ecosystems have been quantified and reported extensively in ecological publications and handbooks.

Table 3.4 Acute toxicity of NaOH to aquatic organisms (OECD, 2002)

Species	Toxicological endpoint	Result (mg/l)	CoR [1]	Remark	Reference
Freshwater fish					
Carassius auratus (goldfish)	Non-lethal concentration 24-hour LC_{50}	100 160	3	pH 9.8 at 100 mg/l	Jensen (1978)
Leuciscus idus melanotus (golden orfe)	48-hour LC_{50}	189	4		Juhnke and Lüdemann (1978)
Gambusia affinis (mosquitofish)	Non-lethal concentration 96-hour LC_{50}	84 125	3	pH 9 at 100 mg/l	Wallen et al. (1957)
Poecilia reticulata (guppy)	24-hour LC_{50}	145	3		Yarzhombek et al. (1991)
Lucioperca lucioperca L. (pike perch) – fry	Toxic concentration	$\geq$ 35	3		Stangenberg (1975)
Freshwater invertebrates					
Dapnia magna (water flea)	Toxicity threshold concentration	40 – 240	4		McKee and Wolf (1963)
Ceriodaphnia dubia (water flea)	48-hour LC_{50}	40	2		Warne and Schifko (1999)
Biomphalaria a. alexandrina (snail)	96-hour Lethal concentration	450	3		Gohar et al. (1961)
Bulinus truncatus (snail)	96-hour Lethal concentration	150	3		Gohar et al. (1961)
Lymnaea caillaudi (snail)	96-hour Lethal concentration	150	3		Gohar et al. (1961)
Marine invertebrates					
Ophryotrocha diadema (polychaete worm)	48-hour LC_{50}	33 – 100	3		Parker (1984)

1 Code of Reliability (CoR): 1 = valid without restrictions,
2 = valid with restrictions,
3 = invalid,
4 = not assignable

3.2.1.2 pH tolerance of (freshwater) aquatic organisms

Based on the OECD guidelines for aquatic toxicity tests with major taxonomic groups, i.e. algae, crustaceans (daphnids) and fish, a pH range of 6-9 is well tolerated by a variety of aquatic organisms. It is noted, however, that the tolerance to relatively low and high pH values depends on the composition of the water and acclimation of the organisms.

Algae and other plants

Some plants tolerate pH values below 3 (Alabaster and Lloyd, 1980).

Invertebrates

Some invertebrates tolerate pH values below 3 (Alabaster and Lloyd, 1980).

Fish

Fish usually tolerate a pH range of 6-9. Most data are available on the tolerance of fish to acid pH values. A pH range of 5-6 may become lethal, as an acid discharge may liberate sufficient carbon dioxide from bicarbonate in the water either to be directly toxic, or to cause the pH range of 5-6 to become lethal. Below a pH value of 5, mortalities may be expected for many species, although some species may be acclimated to pH values as low as around 4 (Alabaster and Lloyd, 1980). The fish *Umbra pygmaea,* which is indigenous in North-America, can tolerate a pH value as low as 3. This fish species has been introduced in the Netherlands in the past and is the only fish species that lives in acid bogs (OVB, 2002).

Data on the tolerance of fish to alkaline pH values is more limited. Relative high pH values of 9-10 may be toxic or lethal to some fish species and above a pH value of 10 mortalities may be expected for many species exposed for a prolonged period. However, where high pH values are caused by vigorous photosynthetic activity of aquatic algae and macrophytes, other factors including a high temperature, supersaturation of dissolved gases and toxins produced by certain algal blooms, obscure the pH effect (Alabaster and Lloyd, 1980). One of the studies reviewed by Alabaster and Lloyd (1980) is described in detail below, based on the original publication (Jordan and Lloyd, 1964).

A test with rainbow trout (*Oncorhynchus mykiss*, formerly known as *Salmo gairdnerii*), acclimatised for 5 days to pH values of 6.55, 7.50 or 8.40 and subsequently exposed to pH values of 9.5 to 11.5, resulted in 1-day LC_{50} values of 9.86, 9.91 and 10.13, respectively. The fish that were acclimatised to the pH value of 8.4 showed a small, but statistically significant higher tolerance to a high pH value than the fish that were acclimatised to the lower pH values, based on the 1-d results. The acclimation did not result in an increased tolerance when the fish were exposed to pH values that were lethal within a few hours, i.e pH values of 10.5-11.5. In a second test with rainbow trout, the fish were acclimatised for 1 day to a pH value of 8.3 and subsequently exposed for 15 days to pH values of 9.5-11.0; this test resulted in a 15-day LC_{50} of 9.5.

In a test with roach (*Rutilus rutilus*), the fish were acclimatised for 1 day to a pH value of 8.3 and subsequently exposed for 10 days to pH values of 10.2-11.7; this test resulted in a 10-day LC_{50} of 10.2. In the above 15-day and 10-day test, the relation between the pH value and the log median survival period showed no threshold value, as appears also to be the case with acids. From the trends of the curves, however, the authors of the study concluded that rainbow trout and roach can tolerate several months of exposure to pH values of 9.0 and 9.8, respectively, which is in good agreement with earlier reported minimum lethal pH values for rainbow trout and roach i.e. 9.2 and 10.4, respectively. The tests were performed in hard borehole water (total hardness 320 mg/l, as $CaCO_3$) to which hydrochloric acid was added to decrease the pH and NaOH was added to increase the pH. In the test with exposure times of more than 1 day, the fish were transferred daily to fresh solutions and fed on alternative days before transfer (Jordan and Lloyd, 1964).

Note that the above data from Alabaster and Lloyd (1980) with respect to fish tolerance to acid and alkaline pH values are based on laboratory and field data for a variety of fish species (salmonids and non-salmonids), with an emphasis on European species.

3.2.1.2.1 Conclusion on tolerance of aquatic organisms to alkaline pH values

The above data on the pH tolerance of fish show that an increase in pH value from around 8.5 to 9.5-10.5. i.e. an increase with 1 to 2 pH units result in acute lethality in fish that were not acclimatised to intermediate values. The data further show that pH values of 9-10 may be toxic or lethal to some fish species and above a pH value of 10 mortalities may be expected for many species exposed for a prolonged period. Data on tolerance of aquatic species other than fish are not included in this report.

Note: Besides a 'direct' effect, i.e. pH increase, NaOH can also have an 'indirect' effect, as the pH change can affect the chemical speciation and thus the toxicity of other substances in water. It is emphasised that these 'indirect' effects are beyond the scope of this risk assessment report for NaOH, but two examples are given in **Appendix A**, for illustration.

3.2.1.3 Aquatic field studies

There are no aquatic field studies available for NaOH. According to OECD (2002) there is, however, a lot of information about the relationship between pH and ecosystem structure and also natural variations in pH of aquatic ecosystems have been quantified and reported extensively in ecological publications and handbooks (OECD, 2002)

3.2.1.4 Toxicity to aquatic micro-organisms

In a test system with the freshwater ciliated protozoan *Tetrahymena thermophila,* NaOH is used as a positive control. The endpoint studied in this test system is the motility pattern, determined by microscopic examination of samples of 25-50 organisms, approximately 2 minutes after the start of the exposure. For a 1% NaOH solution the historical mean HTD ('highest tolerated dose', being the minimal dilution allowing at least 90% normal cell motility compared to the control) is 60 (Silverman and Pennisi, 1987). This is equivalent to a NaOH concentration of 167 mg/l. Based on the TGD criteria, the HTD is considered to be the NOEC or EC10, as $\leq$ 10% effect is observed at the HTD compared to the control. At the NOEC (EC10) of 167 mg/l the calculated pH value was 11.6, assuming that the dilution medium has no buffer capacity. The dilution medium was filtered MM2 medium (0.1% liver powder, 0.1% *Saccaromyces cervisiae*, and 0.001% soy lecithin in distilled water).

The inhibition of the bioluminescence of the marine bacterium *Photobacterium phosphoreum* by NaOH has been measured with the Microtox test system, resulting in a 15-minutes EC_{50} of 22 mg/l. The test medium was a 2% NaCl solution i.e. saltwater (Bulich, Tung and Schreibner, 1990). At the EC_{50} value the calculated pH value was 10.7.

3.2.1.5 Calculation of Predicted No Effect Concentration (PNEC)

3.2.1.5.1 Surface water

No generic PNEC for surface water or STP effluent could be calculated, see Section 3.2.1.1. The risk assessment will only deal with the (potential) pH changes related to local OH^- discharges.

3.3 RISK CHARACTERISATION

3.3.1 Aquatic compartment (STPs and surface waters)

3.3.1.1 Production

Based on the results from a questionnaire among producers, it is concluded that discharges of NaOH from production to STPs/WWTPs and receiving waters are well controlled in all investigated cases (see Section 3.1.2.1). Taking into account the existing EU Directives for pH control for surface water (see Section 2.4) and the data of many Member States on (additional) national regulations to control the pH of waste waters (STP influents) and surface waters it is concluded that STPs and surface waters are sufficiently protected with regard to pH changes.

Conclusion (ii).

3.3.1.2 Use

The results from a questionnaire among users indicate that in most cases the final effluent did not contain NaOH anymore, so it is concluded that discharges of NaOH from the various downstream applications rarely occur. If discharges do occur they are well controlled in all investigated cases (see Section 3.1.2.2) and are often covered by EU and/or national regulations (see Section 2.4).

Conclusion (ii).

Regarding conclusion (ii) for the aquatic compartment it is emphasised that it cannot be excluded that there are (some) sites with NaOH discharges to the aquatic environment, resulting in significant pH changes and effects on biological STPs/WWTPs or receiving surface waters. However, the available data clearly indicate that neutralisation of NaOH containing waste waters and effluents is common practice, either from a legal point of view (legislation for surface waters) or from a practicl point of view (protection of the functioning of biological STPs/WWTPs). Regarding surface water, the enforcement of the (EU) legislation is an important issue for the validity of conclusion (ii).

PBT assessment

The PBT assessment is conducted according to the TGD (EC, 2003).

Persistence

NaOH will rapidly dissolve and dissociate in water. Therefore, NaOH does not fulfil the P criterion.

Bioaccumulation

Bioaccumulation is not relevant for NaOH, therefore, NaOH does not meet the B criterion of the PBT criteria.

Toxicity

The lowest LC_{50} for freshwater and marine organisms were found to be 40 and 33 mg/l, respectively. This is clearly above the cut-off value of 0.1 mg/l. Therefore, NaOH does not meet the T criterion in the PBT assessment.

Conclusion

NaOH, does not fulfil the criteria for persistency, bioaccumulation and toxicity as laid down in the TGD (EC, 2003). Therefore, this substance is not considered a PBT or vPvB substance.

4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1 General discussion

Sodium hydroxide is a white and deliquescent solid, which is generally produced as a 50% solution. It has a melting point and boiling point of 318 and 1388°C, respectively. NaOH solidifies at 20°C if the concentration is higher than 52% (by weight), which can be considered the maximum water solubility at 20°C. NaOH has a very low vapour pressure ($< 10^{-5}$ hPa at 25°C). The octanol water partition coefficient is not relevant for an inorganic substance such as NaOH (OECD, 2002).

NaOH is a strong alkaline substance that dissociates completely in water to sodium and hydroxyl ions. The dissolution/dissociation in water is strongly exothermic, so a vigorous reaction occurs when NaOH is added to water (OECD, 2002).

4.1.1.2 Occupational exposure

The Western European consumption of sodium hydroxide was 9.7 million tonnes in 2003 (see **Table 2.2**). NaOH is used for different purposes in a variety of industrial sectors. The sector with the largest use of NaOH is the production of other chemicals, both organics (28%) and inorganics (16%). Other uses are in the sectors pulp and paper industry (12%), aluminium and metal industry (6%), food industry (3%), water treatment (3%) and textile (3%). The remainder is used in the production of soaps, mineral oils, bleach, phosphates, cellulose, rubber and others (Euro Chlor, 2004b). **Table 2.2** presents the major sectors where NaOH is applied.

NaOH is also used by the drink and beer industry to clean non-disposable bottles. Although the main quantities are used by the industry (large enterprises) it is also widely used by small and medium sized enterprises. It is also used for example for straightening of hair, disinfection and cleaning purposes.

NaOH (up to 100%) is also used by consumers. It is used at home for drain and pipe cleaning, wood treatment and it also used to make soap at home (Keskin et al., 1991; Hansen et al., 1991; Kavin et al., 1996). NaOH is also used in batteries and in oven-cleaner pads (Vilogi et al, 1985).

The previously mentioned uses are only examples of uses but probably many other uses do occur because NaOH is widely available. However, significant differences in uses between countries can be expected.

Dermal contact is the most obvious route of exposure to workers because of the low vapour pressure of NaOH. Ocular exposure is possible due to hand-eye contact or splashes. NaOH is a corrosive substance. For the handling of corrosive substances and formulations, immediate

dermal contacts occur only occasionally and it is assumed that repeated daily dermal exposure can be neglected. Therefore according to the TGD (EC, 2003; Section 2.2.5.3) dermal exposure to pure NaOH will not be assessed. Dermal exposure to dilutions of NaOH that result in a substance or formulation which has no corrosive labelling (dilutions containing < 2% NaOH, according to EU classification and labelling), will be taken into account. Repeated dermal exposure cannot be neglected for these substances and formulations.

Relevant populations potentially exposed to generally corrosive products are workers in the chemical industry, aluminium industry and paper industry. Also textile workers and cleaners may have more or less direct contact with (diluted) NaOH.

The exposure is assessed using the available information on substance, processes and work tasks. Industry provided information on the process and measured data for production of NaOH. Down stream users provided information about the use of NaOH as end product.

More detailed information on the various exposure scenarios may lead to a more accurate exposure assessment.

In this part of the assessment, external (potential) exposure is assessed using relevant models and other available methods in accordance with the Technical Guidance Documents and agreements made at official Meetings of the Competent Authorities. Internal dose depends on external exposure and the percentage of the substance that is absorbed (through the skin and via the respiratory system).

The exposure is generally assessed without taking account of the possible influence of personal protective equipment (PPE). If the assessment as based on potential exposure indicates that risks are to be expected, the use of personal protective equipment may be one of the methods to decrease actual risks, although other methods (technical and organisational) are to be preferred. This is in fact obligatory following harmonised European legislation.

Knowledge of effectiveness of PPE in practical situations is very limited. Furthermore, the effectiveness is largely dependent on site-specific aspects of management, procedures and training of workers. A reasonably effective use of proper PPE for skin exposure may reduce the external exposure with 90%. For respiratory protection the efficiency depends largely on the type of protection used. Without specific information, a tentative reduction efficiency of 90% may be assumed, equivalent to the assigned protection factors for supplied-air respirators with a half mask in negative pressure mode (NIOSH, 1987). Better protection devices will lead to higher protection. Imperfect use of the respiratory protection will lower the practical protection factor compared to the assigned factor. These estimations of reduction are not generally applicable "reasonable worst case" estimations, but indicative values based on very limited data. They will not be used directly in the exposure and risk assessment. Furthermore, the reduction of external exposure does not necessarily reflect the reduction of absorbed dose. It has to be noted, that the use of PPE can result in a relatively increased absorption through the skin (effect of occlusion), even if the skin exposure is decreased. This effect is very substance-specific. Therefore, in risk assessment it is not possible to use default factors for reduction of exposure as a result of the use of PPE. In the assessment of NaOH, the use of effective PPE is always assumed when corrosive concentrations are handled.

For occupational exposure assessment, the following data (if available) are used:

- physico-chemical data of NaOH and products containing the substance: physical appearance, vapour pressure at room temperature, percentage of NaOH in products;

- data regarding methods of use and use pattern of the substance and products potentially containing NaOH and exposure control pattern in the relevant industries;
- exposure data for NaOH from the IUCLID and other sources (literature, exposure databases);
- results from exposure models if applicable (EASE model); in the exposure models the above mentioned types of data are used.

For the occupational exposure assessment the exposure situations can be clustered into 4 scenarios based on the occurrence of NaOH, which are subdivided in different subscenarios. In the first scenario, production of NaOH is considered. The second scenario (formulation) assesses NaOH in products (soap, bleach, etc.) and the third scenario assesses the use of formulated products. Finally, the fourth scenario considers the industrial end use of NaOH. In this scenario four subscenarios are described: use of NaOH in organic and inorganic industry, use of NaOH in the pulp and paper industry, use of NaOH in the aluminium industry and the use of NaOH in the textile industry.

Scenario 1 Occupational exposure from production
Scenario 2 Occupational exposure from formulation
Scenario 3 Occupational exposure from end use of formulated products
Scenario 4 Occupational exposure from industrial end use of NaOH

In this report for each occupational exposure scenario first a general description of dermal and inhalation exposure will be presented. Subsequently, measured data (if available), and results from similar substances in comparable exposure scenarios will be presented. This will be followed by data derived using suitable inhalation models. The methods of estimation for inhalation exposure will be compared using expert judgement and a choice for the best applicable estimates will be made. Dermal exposure will be described and assessed by means of the EASE model and will be compared to measured data (if available) using expert judgement.

4.1.1.2.1 Occupational exposure from production

NaOH is produced commercially by an electrolytic process. Brine, prepared from sodium chloride, is electrolyzed in either a mercury cell, diaphragm cell or membrane cell. The coproducts are chlorine and hydrogen. In the mercury cell process, a sodium-mercury amalgam is formed in the cell. The amalgam is sent to a decomposer where it is reacted with water to form liquid NaOH, hydrogen and free mercury. The free mercury is returned to the electrolytic cell. The resulting NaOH solution is then stored in storage tanks as a 50% solution. The solution is shipped in tank trucks, tank cars or barges. In the membrane process, a solution of approximately 30% in strength is formed. The solution is then sent to evaporators, which concentrate it to a strength of 50% by removing the appropriate amount of water. The resulting NaOH solution is stored in storage tanks prior to shipment. The diaphragm process is very similar to the membrane process except that a solution of only 10-12% is formed in the cell. Therefore, additional evaporation is required to reach the commercialised concentration of 50%. The anhydrous forms of NaOH are obtained through further concentration of 50% NaOH. Solid NaOH results when molten NaOH, from which all the water has been evaporated, is allowed to cool and solidify. Flake NaOH is made by passing molten NaOH over cooled flaking rolls to form flakes of uniform thickness. The flakes can be milled and screened into several crystalline products with controlled particle size. The manufacture of NaOH beads involves feeding molten liquor into a prilling tower

under carefully controlled operating conditions, producing a spherical bead (OxyChem, 2000).

To collect the required information related with occupational exposure at the production sites, a questionnaire has been developed by Euro Chlor in cooperation with the Rapporteur Member State. In the questionnaire the following issues were addressed: type of products (solid/liquid), number of workers, estimation of exposure based on tasks, exposure measurements and accidental exposure. The questionnaires were sent by Euro Chlor to 97% of the European chlorine production sites (a total of 86). A total number of 36 production sites (42%) responded to the questionnaire and based on these data a detailed report has been prepared (Euro Chlor, 2004c). The main results of this report will be presented below.

It can be concluded that nearly all production sites manufacture liquid NaOH with a concentration of about 50%. For 36% of the sites also other liquid products (between 10 and 75%) are manufactured with concentrations which were in general lower than 50%. Solid NaOH (flakes, pearls or cast) was produced at 23% of the production sites. Flakes can be packed in bags (25 or 50 kg). Micro pearls are packed in bags, bulk bags (500 or 1,000 kg) but it is also delivered in bulk (by road). Cast is delivered in metallic drums (e.g. 400 kg). However, it should be realised that other packaging forms could exist.

Although realising that accidental exposure is normally excluded from an EU risk assessment, based on Council Regulation 793/93, accidental exposure was included in the questionnaire because all the NaOH products, which are handled at the production sites, are corrosive. Based on the questionnaires, the average number of accidental exposures was about 6 per NaOH production site for a period of 5 years. Based on the completed accident files, the average number of accidental exposures was about 4 per NaOH production site for a period of 5 years. Overall, the available data indicate a frequency of about one accident per year per NaOH production site. However, the number could be higher due to underreporting.

Inhalation exposure

Atmospheric exposure measurements are available for 6 production sites from 4 different countries (Czech Republic, Poland, Spain and United Kingdom). In all cases the concentrations were lower than 2 mg/m^3 (see **Table 4.1**). Most NaOH production sites replied that the OEL was 2 mg/m^3 in their country. One operation with the possibility of exposure is sampling. The temperature of the liquid product during sampling ranges between 10 and 150°C and the average temperature is 68°C. The amount of product sampled ranged between 0.1 and 15 litres. The responses with the highest quantities were “15”, “2.2”, “2”, “3x1” and “few litres per day”. The remaining respondents replied that an amount of less than 1 kg was sampled. The confinement was in general “semi closed” (18 sites). In the remaining cases the confinement was “open” (6 sites) or “totally closed” (9 sites). General ventilation was present for 26 sites, while 5 sites had no “general ventilation” during sampling. Only five sites had “local exhaust ventilation”. Four sites had neither “general ventilation” nor “local exhaust ventilation”. For sampling the “task duration in minutes per day” ranged between 1 and 600 minutes and the average duration was 71 minutes.

In nearly all cases no PPE was used to protect against inhalation, but in all cases the skin and eyes were protected (e.g. safety glasses, full face mask, gloves, special clothes). Because different NaOH exposures can occur between producing liquid or solid NaOH, different estimations for these scenario’s will be given. Highest exposure will be expected at the drumming/bagging place, depending on the concentration of NaOH. The average number of workers per site appeared to be 30.

Due to the low vapour pressure of NaOH, the atmospheric concentration of NaOH based on vaporisation from the liquid will be very low. Although the exposure to a vapour of NaOH is estimated to be very low, the task related data can not be used to predict the exposure to aerosols (mists).

Measured data

In **Table 4.1** the results of the available atmospheric exposure measurements from 6 NaOH production sites are presented.

Table 4.1 Results of atmospheric exposure measurements at 6 NaOH production sites

Product(s) manufactured at site	Type of measurement	Tasks performed during measurement	Year	N	AM (mg/m^3)	Range (mg/m^3)
Liquid, Cast, Pellets	PAS	Drumming/Bagging	2003	10	0.84	0.1 – 1.8
Liquid	STAT	Truck loading	2002; 2003	17	0.14	0.02 – 0.5
Liquid	Spot	Other	2003	5	0.33	0.29 – 0.37
Liquid	STAT	Other	2002	20		< 0.26
Liquid, Pearls	STAT	Close to installation	2002	109	0.01*	0.05 - 0.18*
Liquid, Cast, Pearls	PAS	Drumming/ Bagging	2003	12	0.09	0.01 – 0.27
Liquid, Cast, Pearls	STAT	Bagging	2003	20	0.05	0.01 – 0.1

PAS Personal Air Sample
STAT Stationary Air Sample
Spot Short term stationary sample
N Amount of measurements
AM Arithmetic mean
* These values are considered not to be correct. A mean value can't be lower than the range.

The data of the production site in Spain are based on measurements of the sodium content, which were performed according to a norm of the National Institute for Worker Safety and Hygiene (NTP-63 of 1983). For this production site the sampling duration was 6-8 hours. Other sites reported that the measurements were based on a Polish standard method, a colorimetric method or on atomic absorption spectroscopy. The sampling duration was unknown for these sites.

Highest exposures are measured at the drumming/bagging place and therefore taken to the risk characterisation. Assuming all measurements were done at drumming/bagging with solid NaOH, the mean values will give an AM of 0.33 mg/m^3 with the range 0.01 – 1.8 mg/m^3.

Three sets of measurements are available during drumming liquid NaOH with a range of 0.02–0.5 mg/m^3. These measurements are stationary samples and no information is given about the conditions. Therefore the highest mean value 0.33 mg/m^3 is taken to the risk characterisation as reasonable worst case and 0.14 mg/m^3 is taken to the risk characterisation as typical exposure.

Modelled data

Drumming liquid NaOH

Inhalation exposure to vapour due to drumming is estimated with EASE 2.0. The exposure range is estimated 0 – 0.17 mg/m^3 (0 – 0.1 ppm, 20^{o}C), assuming very low vapour pressure, no aerosol formation and non-dispersive use. Typical exposure is estimated as 0.085 mg/m^3 (middle value of range). The reasonable worst-case exposure is estimated as 0.17 mg/m^3 (upper value of range) assuming no aerosol formation and non-dispersive use with dilution ventilation. Following the questionnaire, it is assumed that in the present industry LEV is not generally available. Presence of LEV will not influence the exposure range in this estimation. Assuming a NaOH concentration of 50% the typical exposure is estimated to be 0.04 mg/m^3 and the reasonable worst case exposure is estimated to 0.085 mg/m^3. Frequency of exposure for drumming is estimated to be up to 200 days per year with a duration of up to 4 hours/day, while the number of workers involved is estimated to be up to 50 (expert judgement). Assuming 4 hours of handling and zero exposure during the remainder of the working day, 8-hour TWA typical exposure is estimated as 0.02 mg/m^3 and an 8-hour TWA reasonable worst case exposure is estimated as 0.04 mg/m^3.

Bagging/drumming solid NaOH

Considering the particle size distribution (more than 90% larger than 100 μm) of the substance other assumptions than the default assumptions mentioned in the TGD (EC, 2003; Appendix I D), "production and processing of powders" will be used to estimate inhalation exposure to dust with EASE 2.0. Typical exposure is estimated to be 0-1 mg/m^3, assuming low dust technique in the presence of LEV. The reasonable worst case exposure is estimated to be 0-5 mg/m^3, assuming the absence of LEV. Frequency of exposure for drumming is estimated to be up to 200 days per year with a duration of up to 4 hours/day, while the number of workers involved is estimated to be up to 50 (expert judgement). Assuming 4 hours of handling and zero exposure during the remainder of the working day, 8-hour TWA typical exposure is estimated as 0 – 0.5 mg/m^3 and the 8-hour TWA reasonable worst case exposure is estimated as 0 – 2.5 mg/m^3.

Summary of the inhalation exposure level

Modelled data for bagging/drumming solid NaOH are in accordance with the measured data. Because there is a relatively large number of measured data, these will be used for risk characterisation. The value 0.33 mg/m^3 for drumming/bagging solid NaOH (mean value of the measured data) is taken to the risk characterisation as typical exposure level. The value 1.8 mg/m^3 (highest measured value) is taken to the risk characterisation as reasonable worst case exposure level for drumming/bagging solid NaOH. For drumming liquid NaOH the modelled data are underestimated by EASE in comparison with the measured data. Because there is a relatively large number of measured data, these will be used for risk characterisation. The value 0.33 mg/m^3 is taken as reasonable worst case level and 0.14 mg/m^3 is taken as typical exposure level.

Dermal exposure

NaOH products with a concentration > 2% are corrosive, therefore effective control measures are expected to be in place to prevent dermal exposure. Furthermore protective clothing and gloves are considered to be used consistently when handling corrosive substances. Production companies report the use of protective gloves, suits and boots while handling pure NaOH.

Repeated daily dermal exposure to commercial product is therefore considered negligible. Dilutions of NaOH containing < 2% are not produced at the production sites.

4.1.1.2.2 Occupational exposure from formulation

In this scenario NaOH is used for producing products containing NaOH. Occupational exposure can occur during production of these products. Especially during loading and mixing a higher exposure can be expected. No measured data is available in this scenario. An example from the formulation of products containing NaOH, the formulation of certain cleaning products, is presented here. It is assumed that the formulation of other products is done in similar ways and leads to similar exposure levels.

Scenario formulating certain cleaning products with NaOH

Following the "fact sheet cleaning products" of the RIVM, no NaOH is present in all purpose cleaners (Prud'homme et al., 2004). Only in floor strippers (typical concentration ammonia/NaOH 3 – 10%), oven cleaners (liquid, typical concentration 1 – 15% and aerosol, spray can 1 – 5%) and drain openers (liquid, 15 - 30% and solid, pellets 100%) NaOH is present. High exposures can occur during the production process of these cleaning products, when loading concentrated NaOH, which typically involves pumping or pouring a fluid from a barrel or a drum into a process vessel. Inhalation exposure during loading may take place due to vapours or aerosols formed when the barrel or drum is opened and when adding the product to the process. NaOH will be diluted after loading into a tank.

Inhalation exposure

Measured data

No measured data is available for inhalation exposure during formulating cleaning products.

Modelled data

Adding liquid NaOH (T = 20^{o}C) to a process (vapour pressure very low, no aerosol formed, LEV present, use pattern non dispersive use) EASE predicts a typical inhalation exposure of 0-0.17 mg/m^3 (0 – 0.1 ppm). Assuming NaOH concentration of 50% a typical exposure value of 0.04 mg/m^3 (0.025 ppm) is estimated (half of range 0 – 0.05 ppm). Estimating the reasonable worst case exposure gives a value of 0.08 mg/m^3 (0.05 ppm, upper value of the range).

Summary of the inhalation exposure level

The value 0.04 mg/m^3 (0.025 ppm, mean value of the modelled data) is taken as typical exposure level and 0.08 mg/m^3 (0.05 ppm, highest modelled value) as reasonable worst case exposure level to the risk characterisation for adding liquid NaOH (50%) to a process.

Dermal exposure

The NaOH products that are handled in this scenario are corrosive, therefore effective control measures are expected to be in place to prevent dermal exposure. Furthermore protective clothing and gloves are considered to be used consistently when handling corrosive substances. Production companies report the use of protective gloves, suits and boots while

handling pure NaOH. Repeated daily dermal exposure to the pure substance is therefore considered negligible.

4.1.1.2.3 Occupational exposure from end use of formulated products

Scenario use of certain cleaning products

As mentioned before (see Section 4.1.1.2.2), floor strippers, oven cleaners and drain openers for professional users contain a certain amount of NaOH. For estimating worker exposure only oven cleaners or products like oven cleaners will be relevant. In all other cases no relevant inhalation exposure is expected. If concentrations of NaOH are above 2% the concentrations are corrosive and in this case control measures are expected to prevent dermal exposure.

Inhalation exposure

Oven cleaners are used as liquid (concentration NaOH 1% to 15%) and as spray (concentration NaOH 1% to 5%).

Measured data

In April 1998, a health hazard evaluation concerning the cleaning, overhauling and repair of aircraft lavatory tanks and hardware was conducted at one company. The main purpose was to study the potential exposure to infectious micro-organisms but also some measurements of NaOH exposure were conducted (Burton et al., 2000).

NaOH was a component in the soaps and cleaning agents used in the cleaning room. One personal breathing zone and four area samples (three inside and one outside the lavatory cleaning room) were collected. The samples were analysed for alkaline dust and mist by acid-base titration according to NIOSH Method 7401. In **Table 4.2** the results of these measurements are presented.

Table 4.2 NaOH air sampling results from cleaning aircraft lavatory facility sites

Location	Personal/Area	Sample time (min)	Concentration (mg/m³)
Mechanic	Personal	250	< 0.11
Table outside CR	Area	364	< 0.11
CR: sidewall on electrical box	Area	364	< 0.11
CR: centre on unused equipment	Area	360	< 0.11
CR: black wall on tool cart	Area	362	< 0.11

CR Cleaning room

Following Burton et al. (2000) the results were expected to be low since little spraying of the soap was done on the day of the monitoring. Because the exact exposure level is unknown, these measurements are not taken to the risk characterisation.

Modelled data

Liquid oven cleaner

EASE estimates (assuming very low vapour pressure, no aerosol formed, direct handling, non-dispersive use) 0 - 0.17 mg/m^3 (0 – 0.1 ppm) for typical inhalation exposure. Assuming dilution of 1:50 (oven cleaner is not used purely) and NaOH concentration of 7.5% (mean concentration NaOH) typical inhalation value is estimated (by taken the mean value of the range) as $1.3 \cdot 10^{-4}$ mg/m^3 ($0.02 \cdot 0.075 \cdot 0.085$). A reasonable worst case inhalation exposure is estimated by taking the upper range value which gives an estimation of $2.6 \cdot 10^{-4}$ mg/m^3 ($0.02 \cdot 0.075 \cdot 0.17$). Both, typical and worst case estimates, can be considered to be negligible.

Spray oven cleaner

NaOH is a non-volatile substance and therefore EASE is not suitable for estimating inhalation exposure occurred by spraying. The TGD (appendix I C) refers to a model derived by De Pater and Marquart (1999) to estimate inhalation exposure to non-volatile substances during spraying. This model is based on measured exposure levels to polyisocyanates in spray coating and is also considered to be relevant for spray cleaning. Model:

$$Es = Em \cdot (Cs/Cm)$$

Es = the estimated inhalation exposure (mg/m^3),
Em = the measured exposure to non-volatiles (mg/m^3),
Cs = the percentage of the notified substance and
Cm = the percentage total non-volatile substances.

Assuming a NaOH concentration of 3% (mean concentration of NaOH in spray) Cs is 0.03. Because the measured exposure to non-volatiles and the percentage non-volatile substances are unknown, the estimates for spray painting are used as indicative values: Em = 10 mg/m^3 and Cm = 0.3. This results in an estimated inhalation exposure of 1 mg/m^3 ($10 \cdot 0.03/0.3$). If spraying occurs 1 hour/day and rest of the day no exposure is assumed, a reasonable worst case of 0.13 mg/m^3 is estimated.

Summary of the inhalation exposure level

For using oven pads with liquid NaOH the typical inhalation exposure level is estimated to be negligible. For using oven spray with NaOH the 8-hour TWA reasonable worst case inhalation exposure level is estimated as 0.13 mg/m^3. It must be mentioned that this estimate is very uncertain due to the lack of data.

Dermal exposure

Measured data

No measured data is available for dermal exposure using oven cleaners in professional use.

Modelled data

In both, liquid and spray, dermal exposure is modelled assuming the highest non-corrosive concentration of 2% NaOH.

Liquid oven cleaner

EASE estimates (assuming pattern of use wide dispersive use, direct handling and contact level intermittent) a range of 1 – 5 mg/cm^2/day to the product. Assuming exposure to one hand (420 cm^2) and a NaOH concentration of 2%, reasonable worst case dermal exposure level is estimated as 42 mg/day (5 · 420 · 0.02).

Spray oven cleaner

EASE estimates (assuming pattern of use wide dispersive use, direct handling and contact level intermittent) a range of 1 – 5 mg/cm^2/day to the product. Assuming exposure to both hands (840 cm^2, due to spraying NaOH which increases the possible contaminated area) and a NaOH concentration of 2%, reasonable worst-case dermal exposure level is estimated as 84 mg/day (5 · 840 · 0.02).

Summary of the dermal exposure level

Assuming a liquid oven cleaner containing 2% NaOH (maximum non-corrosive concentration) reasonable worst case exposure is estimated as 42 mg/day.

Assuming a spray oven cleaner containing 2% NaOH (maximum non-corrosive concentration) reasonable worst case exposure is estimated as 84 mg/day.

Scenario use of hair straightening products

Several hair straightening products used by professional hairdressers contain a certain amount of NaOH. Hair straightening products, containing more than 2% of NaOH, are applied to the hair with a brush and after a period of interaction with the hair the product is rinsed out with water. For estimating worker exposure no relevant inhalation exposure is expected because of the low volatility and the lack of aerosol formation. Dermal exposure is only relevant when concentrations of NaOH are below 2%, which probably will occur when the product is rinsed out of the hair. Above 2% the product will be corrosive which means control measures are expected to prevent dermal exposure. The exposure is therefore expected to occur mainly when the hairdresser decided to do a final rinsing step after the first rinsing is done.

Dermal exposure

Measured data

No measured data is available for dermal exposure using hair straightening products.

Modelled data

EASE is used to model dermal exposure. Assuming non-dispersive use, pattern of control direct handling and contact level incidental (because hair straightening products will be used infrequently) EASE estimates a dermal exposure value of 0 – 0.1 mg/cm^2/day. If a concentration of 2% NaOH is used, the range will become 0 – 0.002 mg/cm^2/day. Assuming 2 hands exposed (840 cm^2), typical exposure is estimated as 0.84 mg/day (0.001, middle of range x 840) and reasonable worst case exposure is estimated as 1.68 mg/day (0.002, maximum of range x 840).

Summary of the dermal exposure level

When washing out a hair straightening product containing at maximum 2% NaOH (maximum non-corrosive concentration) reasonable worst case exposure is estimated as 1.68 mg/day.

4.1.1.2.4 Occupational exposure from industrial end use of NaOH

To collect the required information related with occupational exposure when using NaOH, a questionnaire has been developed by Euro Chlor in cooperation with the Rapporteur Member State. In September 2004 questionnaires have been sent by e-mail to:

- The Confederation of European Paper Industries (CEPI). They have forwarded the questionnaires to their members (paper producing companies which use NaOH).
- Five different contact persons from Euro Chlor member companies (NaOH producers). Afterwards each producer of NaOH has sent the questionnaire to 20 customers (in most cases end users of NaOH).

The responses were analysed and the results reported by Euro Chlor (2005).

A total number of 58 replies were received, originating from about 10 different EU member states. The majority (59%) originated from the pulp and paper industry and and therefore the data for this sector can be considered as highly representative for the situation in Europe. For the pulp and paper industry one questionnaire was received from Germany (National Federation), which represented the common practice in this country.

The response from other industry customers was less but still covered a broad range of applications of NaOH. A total of 17 questionnaires (29%) were received from the chemical industry (e.g. production of crop protection chemicals, organic pigments, epoxy resins). The remaining 7 questionnaires were received from steel industry, textile industry, rubber production, food industry, metal industry, aluminium industry and distribution. This shows that 23 end users of NaOH replied, while one distributor completed the questionnaire.

In most cases the NaOH was used as a reactant during the manufacturing/production of chemicals. In a few other cases it was used for neutralisation (steel industry, rubber production), cleaning and water treatment (food industry) or for extraction (aluminium industry). Only four end users reported that NaOH was present in their final product, all of them using the NaOH for chemical production. The amount of NaOH present in the final product was 0.7; 1.0; 2.5 and 5%. For the remaining end users the NaOH was not present in their final product. A majority (19 respondents) used the 50% NaOH product. Three respondents used flakes and only one respondent used pearls/prills.

A total of 8 of 22 customers (36%) replied that they used local exhaust ventilation when they handle NaOH on their site. NaOH was never sprayed by the customers. Twentynine percent of the customers replied that inhalation exposure was possible, while 71% answered that skin exposure was possible and finally 75% replied that eye exposure was possible. In most cases no PPE was used to prevent inhalation. To prevent skin exposure, 46% of the respondents reported that gloves were used, while 25% reported that special clothes were used and finally 29% replied that no PPE was used. To prevent eye exposure 67% of the customers answered that safety glasses or a full facemask was used and the remaining customers replied in most cases that no PPE was used (Euro Chlor, 2005).

Based on these questionnaires occupational exposure in 4 industries are described more precisely. Use of NaOH in the pulp and paper industry, chemical (organic and inorganic) industry, aluminium industry and textile industry

Use of NaOH in the pulp and paper industry

NaOH is used in the papermaking industry to dissolve the lignin between the wood fibres, thereby enabling the fibres to separate relatively undamaged. To do this, wood pulp and chemicals (NaOH, Na_2S) are cooked together in a pressure vessel (digester) which can be operated on a batch or continuous basis. In case of batch filling the digester is filled through a top opening. This can cause exposure to the used chemicals. Modern kraft pulping is usually carried out in a continuous digester often lined with stainless steel and exposure to NaOH is then expected to be minimised. The temperature of the digester is raised slowly to approximately 170^{o}C and held at that level for approximately 3 to 4 hours. The pulp is screened to remove uncooked wood, washed to remove the spent cooking mixture, and send either to the bleach plant or to the pulp machine. At the end of the process step, sodium hydroxide is reformed in the recausticizing plant (EOHS, 2001). During the bleaching process NaOH is used in the so-called alkali extraction where the organic acids and alcohols react with the NaOH to form organic sodium compounds and water. These organic substances dissolve in water. The purpose of the bleaching is to remove lignin without damaging the cellulose. NaOH is also used for waste paper recycling: adding water, NaOH and heat repulps recycled material

In the pulp and paper industry liquid NaOH is the most important type of product and use of solid forms is quite rare. The major NaOH strength used by respondents was the 50% liquid, other concentrations with strengths ranging from 10% to 48% were mentioned less often. Almost all plants (97%) indicated having an automated closed system. Still 50% indicated that handling with NaOH still occurs during (re)filling of tanks/containers, cleaning, maintenance, unloading lorries, adding reactant, emptying drums or bags and sampling (average of 4 workers per plant). 24% of the plants reported to use local exhaust ventilation when handling NaOH. Spraying in the process applications of NaOH never occured, so there is no risk for exposure through aerosols. Exposure through inhalation was possible indicated 35% of the plants. PPE used were half facemask and full facemask.

From all respondents, 88% indicated a possibility for skin or eye exposure. In all cases where potential skin and eye exposure was considered possible, PPE was applied. The PPE listed for skin protection where ‘gloves’, ‘overall’ and ‘special clothing’ and for ‘eye’ these were ‘safety glasses’ or ‘full facemasks’ (Euro Chlor, 2005).

In several cases it was indicated that specific measures are not necessary, referring to the fully automated or closed system conditions. The specific measures listed are safety working procedures for specified tasks (discharging and repair measurements, unloading trucks and filling storage tanks), (Euro Chlor, 2005).

Inhalation exposure

Measured data

In 1988 measurements were conducted in a paper mill (Kennedy et al., 1991). A total of 28 area samples were taken at different locations with a minimum measurement time of 8 hours (see **Table 4.3**). It is unclear how measurements were collected.

Table 4.3 Measurements conducted at a paper mill

Location	Number)	Duration (hour)	TWA (mg/m³)
Woodplant	2	> 8	< 0.5
Pulping	12	> 8	< 0.5
Bleach/ chem. Preparation	9	> 8	< 0.5
Machine room	2	> 8	< 0.5
Recover and recaust	3	> 8	< 0.5/16.0*

* A single high reading because of upset conditions at the slaker/causticizer

None of the measurements exceeded the detection level. All measured areas where exposed for over 8 hours to a NaOH concentration below 0.5 mg/m^3.

In an international epidemiological study of workers exposure to chemical agents in the pulp and paper industry a database with a total of 3873 measurements were analysed (Korhonen et al., 2004). Most of the measurements were from 1980 to 1994 and from a total 12 countries. A total of 15 measurements were conducted to NaOH (see **Table 4.4**).

Table 4.4 Results of exposure to NaOH in paper, paperboard and recycled paper industry

Department	Type of measurement	N	n	AM (mg/m³)	Range (mg/m³)
Pulping, refining, etc. of stock	TWA	5	2	0.001	0.001 – 0.002
Paper/paperboard machine	TWA	5	1	0.05	0.01 – 0.23
De-inking of waste paper	TWA	5	5	0.70	0.30 – 1.20

TWA Time weighted average of air samples, duration > 1 hour
N Total number of measurements
n Number of detectable measurements
A M Arithmetic mean

Two measurements during pulping stock and one measurement at the paperboard machine were exceeding the detection limit. When de-inking waste paper all measurements were exceeding the detection limit with an AM of 0.70 mg/m^3 (range 0.30 – 1.20 mg/m^3). The duration of the measurements was more than one hour, but the exact duration was unclear. It was not clear from the article which tasks were conducted during the measurements.

Modelled data

Adding liquid NaOH (T = 20^{o}C) to a process (vapour pressure very low, no aerosol formed, use pattern non dispersive use) EASE predicts a typical inhalation exposure of 0 – 0.17 mg/m^3 (0 – 0.1 ppm). Assuming NaOH concentration of 50% a typical exposure value of 0.04 mg/m^3 (half of range 0 – 0.08 mg/m^3) and a reasonable worst case exposure of 0.08 mg/m^3 (upper value of the range) is estimated.

Summary of the inhalation exposure level

Almost all measured data in **Table 4.3** and **4.4** are not exceeding the detection limit and are therefore not valuable for risk characterisation. In **Table 4.4** de-inking of waste paper can cause an exposure of up to 1.20 mg/m^3. De-inking seems to be a process with a higher

possibility on exposure. Therefore a different value will be taken to risk characterisation for de-inking of waste paper as part of pulp and paper. For adding liquid NaOH to a process in pulp and paper industry the value 0.04 mg/m^3 (mean of the modelled data) is taken as typical exposure level and 0.08 mg/m^3 (highest modelled value) is taken as reasonable worst case exposure level to the risk characterisation. For de-inking waste paper a typical value of 0.70 mg/m^3 (mean of measured data) was taken to risk characterisation and a value of 1.20 mg/m^3 (highest value of measured data) was taken as reasonable worst case value. Because measurement duration is more than one hour, 8-hour TWA is assumed as reasonable worst case.

Dermal exposure

NaOH products with a concentration > 2% are corrosive, therefore effective control measures are expected to be in place to prevent dermal exposure. Furthermore protective clothing and gloves are considered to be used consistently when handling corrosive substances. Production companies report the use of protective gloves, suits and boots while handling pure NaOH. Repeated daily dermal exposure to the pure substance is therefore considered negligible.

Use of NaOH by chemical industry (organic and inorganic)

At the production sites of organic and inorganic chemicals, NaOH is used as pH stabiliser or as reactant for synthesis of other chemicals. In all cases NaOH must be added to a reaction vessel and will react after which no NaOH is left. In some plants NaOH is recycled to the process.

Inhalation exposure

Highest inhalation exposure is expected to occur by loading NaOH from tanker to process vessel. Most of the industries use a closed and/or automated process and liquid 50% NaOH. The NaOH will not be sprayed and only one-third of all industries use local exhaust ventilation when handling NaOH.

Measured data

No measured data is available for inhalation exposure during use of NaOH in organic or inorganic processes.

Modelled data

Adding liquid NaOH (T = 20^{o}C) to a process (vapour pressure very low, no aerosol formed, use pattern non dispersive use) EASE predicts a typical inhalation exposure of 0 – 0.17 mg/m^3 (0 – 0.1 ppm). Assuming an NaOH concentration of 50% a typical exposure value of 0.04 mg/m^3 (0.025 ppm) is estimated (half of range 0 – 0.05 ppm). For estimating the reasonable worst case exposure 0.08 mg/m^3 (0.05 ppm, upper value of the range) is taken.

Summary of the inhalation exposure level

The estimated value 0.04 mg/m^3 (0.025 ppm, mean value of the modelled data) is taken as typical exposure level and 0.08 mg/m^3 (0.05 ppm, highest modelled value) as reasonable worst case exposure level to the risk characterisation for adding liquid NaOH to a process. These values are probably substantial overestimates of true exposures due to the very low vapour pressure of NaOH.

Dermal exposure

NaOH products with a concentration > 2% are corrosive, therefore effective control measures are expected to be in place to prevent dermal exposure. Furthermore protective clothing and gloves are considered to be used consistently when handling corrosive substances. Production companies report the use of protective gloves, suits and boots while handling pure NaOH. Repeated daily dermal exposure to the pure substance is therefore considered negligible.

Use of NaOH in the aluminium industry

Aluminium is produced from bauxite by the Bayer process. Mixed with steam and a (strong) NaOH solution, alumina in the bauxite forms a concentrated sodium aluminate solution leaving undissolved impurities. The conditions to extract the monohydrate alumina are about 250^{o}C and a pressure of about 3,500 kPa (Queensland Alumina Limited, 2004)). At the end of the process NaOH is returned to the start and used again.

Inhalation exposure

Relatively high inhalation exposure to NaOH is expected to be caused during the mixing of bauxite with NaOH and steam due to the high temperatures and high concentrations of NaOH.

Measured data

At company A static measurements were conducted in 1997 and 1999 to "caustic mist" during production of aluminium. In **Table 4.5** a summary is given of these measurements. In another study (Fritschi et al., 2001) the results for exposure to caustic mist were qualitatively presented and therefore not suitable for the risk assessment.

Table 4.5 Measurements of caustic mist (NaOH) at an aluminium production site

Year	Location	Medium	N	AM sample time (min)	Conc. AM (mg/m^3)
1997	During caustic wash	Impinger	3	111	0.32
1999	During caustic wash	Impinger	2	105	0.30
1997	Sand trap dump (at operator location)	Impinger	1	5	5.8*
1999	Sand trap dump	Filter	1	15	0.17
1999	Caustic wash recycle tank	Filter	1	15	0.47
1999	Screw conveyor new building	Filter	1	15	0.06
1999	Overflow tank old building	Filter	1	15	0.06
1999	Sampling point decanter 2	Filter	5	80	0.1
1999	Sampling point decanter 1	Impinger	2	76	0.08
1999	Filter wash #13 top floor at hoist control	Impinger	1	45	0.14
1999	Over caustic tank ground floor	Impinger	1	107	0.56

Table 4.5 continued overleaf

Table 4.5 continued Measurements of caustic mist (NaOH) at an aluminium production site

Year	Location	Medium	N	AM sample time (min)	Conc. AM (mg/m³)
1997	At drum filters/normal operating:	Impinger			
	on filter platform		1	38	2.4**
	on ground in front of filter		1	14	4.7**
	on filter platform		1	117	0.4
	on workbench on filter floor		1	65	0.5
	1st floor at filter drain valve platform		1	78	0.6
	1st floor by conveyor belt		1	78	1.1
1999	Over Launder gate during caustic wash	Filter	2	100	0.06
1999	Over precipitation tank during caustic wash	Filter	2	100	0.033
1999	At operator location when descaling launder gates	Impinger	2	20	0.85
1999	Caustic wash filling Primary B tank, sample on top of tank	Impinger	2	50	1.00
1999	Adjacent to cyclones during normal processing	Impinger	2	20	0.55

N Number of measurements
AM Arithmetic mean
* Sample known to be contaminated as no steam/mist came in contact with sampler during sampling; samples was taken up-wind of steam source due to prevailing wind conditions
** Samples were taken in very wet steam/ mist clouds; problems with pumps cutting out and pumps flooding were recorded

Measurements were conducted to caustic mist with a 37 mm, 0.8 μm, MCEF, membrane filter with a cellulose backup pad in a closed face 3 piece cassette or with a SKC midget impinger containing ultra pure water. All measurements performed (see **Table 4.5**) are worst case area samples and many of the locations selected for sampling were ones where high concentrations were expected. The arithmetic mean of all measurements is 0.39 mg/m^3 with a range 0.033-1.1 mg/m^3 (excluding measurements in accidental situations with failing equipment). Mean measurement time is 57 min. Because operators are not routinely present at the measured locations it is assumed that total present time during a day is the same as the approximate mean measurement time (1 hour). Expecting an 8 hour working day with an exposure of 1.1 mg/m^3 for 1 hour and zero exposure during the rest of the day gives a full shift reasonable worst case exposure level of 0.14 mg/m^3. The short term reasonable worst case value is estimated as 1.1 mg/m^3. Expecting an 8 hour working day with an exposure of 0.39 mg/m^3 for 1 hour and zero exposure rest of the day gives a full shift typical exposure level of 0.05 mg/m^3. The short term typical exposure value is estimated as 0.39 mg/m^3.

Modelled data

EASE predicts a typical inhalation exposure of 0 – 0.1 ppm (vapour pressure very low, no aerosol formed, use pattern non dispersive use). Assuming an NaOH concentration of 50% a typical exposure value of 0.04 mg/m^3 (0.025 ppm) is estimated (half of range 0 – 0.05 ppm).

For estimating the reasonable worst case exposure level 0.08 mg/m^3 (0.05 ppm, upper value of the range) is taken.

Summary of the inhalation exposure level

Although the number of measurements is limited, they are considered to be more representative than modelling exposure of evaporation of NaOH from water. Therefore 0.05 mg/m^3 is taken as typical exposure level and 0.14 mg/m^3 as 8-hour TWA reasonable worst case exposure level to the risk characterisation.

Dermal exposure

NaOH products with a concentration > 2% are corrosive, therefore effective control measures are expected to be in place to prevent dermal exposure. Furthermore protective clothing and gloves are considered to be used consistently when handling corrosive substances. Production companies report the use of protective gloves, suits and boots while handling pure NaOH. Repeated daily dermal exposure to the pure substance is therefore considered negligible.

Use of NaOH in the textile industry

In the viscose process, cellulose derived from wood pulp is steeped in a sodium hydroxide solution (20-25%), and the excess liquid is squeezed out by compression to form alkali cellulose. Impurities are removed and, after being torn into shreds similar to white crumbs that are allowed to age for several days at controlled temperature, the shredded alkali cellulose is transferred into another tank were it is treated with carbon disulphide to form cellulose xanthate. These are dissolved in diluted sodium hydroxide to form a viscous orange liquid called viscose. The acids and alkalis used in the process are fairly dilute, but there is always danger from the preparing of the proper dilutions and splashes into the eyes. The alkaline crumbs produced during the shredding may irritate workers' hand and eyes.

The major part of the sodium hydroxide used in the textile industry is used in the mercerization, bleaching, scouring and washing of cotton. In these processes the concentration of the solutions varies between 1 and 25% and the temperature between room temperature and 100^{o}C.

Inhalation exposure

Exposure to NaOH can occur when steeping woodpulp and during dissolving cellulose xanthate. Most of the industries use a closed and/or automated process. The NaOH will not be sprayed. Only one-third of all industries use local exhaust ventilation when handling NaOH.

Measured data

In 1981 measurements were conducted at different textile producing companies in Finland (Nousiainen et al., 1981). A total of 198 area samples were taken at different locations for a whole shift duration (see **Table 4.6**). During the measurements the fixed apparatus was positioned so that the best possible approximate values of the worker exposure would be obtained, without disturbing normal work routines. The distance from the outer edge of the mercerization, leaching or washing machine was 1 m and the sampling height from the floor or work platform was 1-5 m. The measurements were made at the front, middle and back part of each mercerization machine. The contents measured at the middle were often highest

because the solution was hot there. For bleaching the measurements were likewise made at different points of the machine.

Table 4.6 Measurements conducted in different textile producing companies

Location	Number	Number of workers exposed	NaOH (mg/m³) AM	NaOH (mg/m³) GM
Mercerization	86	8	1.7	1.0
Bleaching	74	13	3.3	1.1
Washing	16	1	2.9	1.8
Mixing and concentration	14	3	6.8	2.0
Storage	8	1	2.2	1.4

Most measurements were conducted during mercerisation and bleaching and the number of workers possibly exposed is, in comparison with other locations, high.

Modelled data

Steeping cellulose in sodium hydroxide solution can be compared with mixing. In this case cellulose will be added to sodium hydroxide. When assuming a closed system with vapour pressure very low, no aerosol formed and use pattern non-dispersive, EASE predicts a value of 0 – 0.17 mg/m^3 (0 – 0.1 ppm). If a concentration of 25% NaOH is used, the range will become 0 – 0.043 mg/m^3.

Summary of the inhalation exposure level

It is assumed that the measured data represent rather old fashioned situations. However the highest AM will be used as an estimate of present reasonable worst case exposure level. Due to the fact that measured data are stationary samples and an operator normally works half a day near measured locations, an overestimation of exposure is assumed. Taking this into account the value 1.7 mg/m^3 (3.3 · 4 hour/8 hour) is taken as typical exposure level and 3.4 mg/m^3 (6.8 · 4 hour/8 hour) as reasonable worst case exposure to the risk characterisation.

Dermal exposure

NaOH products with a concentration > 2% are corrosive, therefore effective control measures are expected to be in place to prevent dermal exposure. Furthermore protective clothing and gloves are considered to be used consistently when handling corrosive substances. Production companies report the use of protective gloves, suits and boots while handling pure NaOH. Repeated daily dermal exposure to the pure substance is therefore considered negligible. Dilutions of NaOH containing less than 2% of the substance do not have corrosive properties. For this concentration a dermal exposure value is estimated.

Measured data

No measured data to dermal exposure is available.

Modelled data

Because there is a chance workers are exposed to concentration of NaOH below 2%, EASE is used to model dermal exposure. Assuming non-dispersive use, pattern of control direct handling and contact level incidental, EASE estimates a dermal exposure value of 0-0.1 mg/cm^2/day. If a concentration of 2% NaOH is used, the range will become 0-0.002 mg/cm^2/day. Assuming 1 hand exposed (420 cm^2), typical exposure is estimated as 0.42 mg/day (0.001, middle of range x 420) and reasonable worst case exposure is estimated as 0.84 mg/day (0.002, maximum of range x 420).

Summary of the dermal exposure level

As typical dermal exposure level 0.42 mg/day will be taken to risk characterisation and as reasonable worst case exposure 0.84 mg/day will be taken to risk characterisation for handling concentrations < 2% NaOH.

4.1.1.2.5 Summary of occupational exposure

In **Table 4.7** all estimated levels for the different scenario's are summarised.

Table 4.7 Conclusions of the occupational exposure assessment

Scenario	Activity [1]	Frequence Days/year	Duration Hours/day	Inhalation				Dermal			
				Reasonable worst case		Typical concentration		Reasonable worst case		Typical concentration	
				Mg/m3	Method[2]	Mg/m3	Method [2]	Mg/day	Method [2]	Mg/day	Method [2]
Production											
Drumming liquid NaOH	Full shift	200	8	0.33	Modelled	0.14	Modelled	--	--	--	--
Bagging NaOH	Full shift	200	8	1.8	Measured	0.33	Measured	--	--	--	--
Formulation											
Cleaning products	Full shift	--	--	0.08	Modelled	0.04	Modelled	--	--	--	--
End use formulated products											
Oven cleaner liquid	Full shift	--	8	Negligible	Modelled	Negligible	Modelled	42	Modelled	--	--
Oven cleaner spray	Full shift	--	8	0.13	Modelled	--	--	84	Modelled	--	--
Hair straightening products	Full shift	--	--	--	--	--	--	1.68	Modelled	0.84	Modelled
Industrial uses											
Chemical industry	Full shift	--	--	0.08	Modelled	0.04	Modelled	--	--	--	--
Aluminium	Full shift	--	8	0.14	Measured	0.05	Measured	--	--	--	--
	Short term	--	1	1.1	Measured	0.39	Measured	--	--	--	--
Pulp/paper	Full shift	--	--	0.08	Modelled	0.04	Modelled	--	--	--	--
De-inking of waste paper	Full shift	--	> 1*	1.20	Measured	0.70	Measured	--	--	--	--
Textile	Full shift	--	8	3.4	Measured	1.7	Measured	0.84	Modelled	0.42	Modelled

*) Assuming 8-hour TWA as reasonable worst case
1) Full shift, short term, etc.
2) Measured, EASE, Expert judgment, Calculated, etc.

4.1.1.3 Consumer exposure

Sodium hydroxide has many industrial and domestic uses and it is available to the general public. For consumer exposure it is important to stress, that sodium hydroxide exposure is an external exposure. Contact with tissue and water will give sodium and hydroxide ions. These ions are abundantly available in the body. A significant amount of sodium is taken up via the food because the normal uptake of sodium via food is 3.1-6.0 g/day according to Fodor et al. (1999). Therefore, for external exposure the amount of sodium hydroxide in percentages in the products will be used for the risk assessment as the effects of sodium hydroxide are expressed in percentages sodium hydroxide in solutions.

Besides this, the external exposure concentrations in mg/kg will also be calculated and compared with the sodium intake via food to see whether this is a relevant exposure route. Important for the normal external exposure for sodium hydroxide are the irritant and corrosive properties. These will be addressed in the risk characterisation. At the end of this section the accidental exposure to sodium hydroxide will be addressed using information received from questionnaires, which were sent to the European Poison Centres.

Five scenarios will be used: floor strippers, hair straighteners, oven cleaners, drain openers and other cleaning products because these products contain a significant amount of sodium hydroxide.

The table below gives an overview on use of sodium hydroxide in consumer products.

Table 4.8 Overview on use of sodium hydroxide in consumer products

Product category	Maximum content	Exposure
Floor, carpet and furniture products		
- floor strippers	10%[1,2]	Dermal
Cosmetics		
- hair straigtheners	2%[4]	Dermal
Cleaners		
- oven cleaners	15% (liquid)[1] 5% (aerosol spray can)[2]	Dermal, inhalation
- drain openers	30% (liquid)[3] 100% (solid, pellets)	Dermal

1) Vollebregt et al. (1994)
2) Household Products Database (2004)
3) Information from the Dutch Association for Soap Manufacturers (NVZ) (2004), compilation of different manufacturers
4) EU Cosmetics Directive, Annex III entry 15a (page 63)

4.1.1.3.1 Exposure from general use

Scenarios

I Floor strip products

Floor strippers are used to remove old protective layers. The maximum content of sodium hydroxide in floor strippers is 10% and this will be used in the risk assessment. Only dermal exposure is expected.

Dermal exposure

For stripping the floor of the living room 550 g of the product is needed for an area of 22 m^2. This is done with the undiluted product. The product is sprinkled on a cloth and is manually rubbed on the floor. Dermal exposure occurs by hand contact with the cloth. Worst-case is estimated that 1% of the product amount comes into contact with the skin i.e. 5.5 grams (Prud'homme et al., 2004). Given a maximum content of 10% NaOH in these products, the potential dermal uptake per event for a person of 70 kg will be:

5,500 mg • 10% = 550 mg NaOH/70 kg = 7.9 mg/kg bw

This amount is negligible compared to the daily dietary intake of sodium ions.

II Hair straighteners

The maximum content of sodium hydroxide in hair straighteners for use by the general public is 2% (EU Cosmetics Directive) and will be used in the risk assessment. Dermal exposure is expected.

Sodium hydroxide as a caustic type of chemical will actually soften hair fibres. It will also cause the hair to swell at the same time. As the sodium hydroxide solution is applied to the hair, it penetrates into the cortical layer and breaks the cross-bonds. The cortical layer is actually the middle or inner layer of the hair shaft that provides the strength, elasticity and shape of the curly hair.

For estimating the external exposure to NaOH in hair straighteners the exposure estimation for getting a permanent was used (Bremmer et al., 2002). Worst-case is estimated that 100% of the product amount comes into contact with the skin and that 80 grams are used. Given a maximum content of 2% NaOH in these products, the potential dermal exposure per event for a person of 70 kg will be:

80 g • 2% = 1.6 g NaOH/70 kg = 23 mg/kg bw

This amount is negligible compared to the daily dietary intake of sodium ions.

III Oven cleaners

The maximum content of sodium hydroxide in a spray can is 5% and will be used in the risk assessment. Dermal and inhalatory exposure is expected.

Oven cleaners are strong degreasers and they are suitable for removing dirt stuck on ovens, grills etc. Oven cleaners contain strong alkaline ingredients. Strong alkali is necessary to remove burned-on soils. There are trigger sprays and spray cans. When using a spray can, foam is formed on the target area and there is mainly dermal exposure. Using a trigger spray

causes also inhalatory exposure and therefore the exposure assessment will focus on the application of a trigger spray.

Inhalation exposure

For certain specific uses, e.g. cleaning ovens and disinfection of sheds the use of a trigger spray is possible and the formation of aerosols can not be excluded completely. Aerosols of sodium hydroxide are not stable. They are rapidly transformed due to an uptake of carbon dioxide from the atmosphere resulting in the formation of sodium bicarbonate and sodium carbonate. The transformation of respirable sodium hydroxide aerosols into sodium carbonate aerosols can occur in seconds (Cooper et al., 1979).

The maximum weight fraction of sodium hydroxide in a trigger spray is assumed to be 5%. The cleaner will be used indoors for a relatively short period of time per event (about 13 seconds). Because of this short-term use and the fact that sodium hydroxide is unstable in air, inhalatory exposure following the use of oven cleaners is considered to be negligible.

Dermal exposure

After spraying, the oven door is closed and the foam has to soak 30 minutes (product information). Then the oven is wiped clean with a wet cloth or sponge and one has to rinse frequently. The product information recommends to wear long rubber gloves and to avoid contact with skin, eyes, mucous membranes and clothing. Not all users will do this; therefore it is assumed that the user wears no gloves while cleaning the oven (worst-case estimation). Given the instability of sodium hydroxide it is assumed that after the 30 minutes of soaking time all of the substance has reacted. Therefore, dermal exposure to sodium hydroxide after spraying of an oven cleaner is considered to be negligible.

IV Drain openers

Drain openers open slow running and obstructed drains by dissolving and by loosening grease and organic waste. There are different kinds of drain openers, products containing either sodium hydroxide (caustic soda or caustic liquid) or sulphuric acid.

Liquid drain openers have a maximum NaOH content of 30%. Pellets, which can also be used for opening the drain, have contents up to 100%. For the risk assessment 30% as maximum content has been used, as it is considered that dermal exposure to spatters when using pellets is comparable to that after using liquid drain openers. Only dermal exposure is considered relevant.

Dermal exposure

The use of liquid drain openers is comparable with the dosing of liquid cleaners. The drain opener must be dosed slowly down the drain. One has to wait at least 15 minutes so that the drain opener can clear the blockage. As the products are corrosive, hands and eyes must be protected. It is assumed that consumers do not use gloves. Dermal contact with the product can occur if droplets of caustic soda spatter on the skin. It is assumed that two drops of 50 µl with a density of 1 g/cm^3 will end on the skin of the hands (0.1 g undiluted drain opener). Content of NaOH in the drain opener is set to 30%. The potential dermal uptake per event for a person of 70 kg will then become:

$$2 \cdot 50 \text{ mg} \cdot 30\% = 30 \text{ mg NaOH}/70 \text{ kg} = 0.4 \text{ mg/kg bw}$$

This amount is negligible compared to the daily dietary intake of sodium ions.

V Other cleaning products

Caustic soda (NaOH) is used during the production phase of various cleaning products although in most cases the amounts are low and NaOH additions are mainly for pH adjustment. The amounts used will react with other ingredients in acid-base reactions and thus practically no NaOH is left in the final consumer product. However, hypochlorite products may contain 0.25-0.45% of NaOH in the final formulation. As these contents of sodium hydroxide are very low in comparison with the other mentioned consumer products this scenario will not be used for exposure calculation.

4.1.1.3.2 Exposure from accidental uses

Sodium hydroxide has many industrial and domestic uses and it is available to the general public. The substance has been used already for a long time. For this reason accidental or intentional acute exposures (suicide) have been described extensively in the medical literature. Many medical case reports and reviews of medical treatment methods of sodium hydroxide burns are available.

Additionally, to get an actual picture of the number of accidents, the main kind of exposure route and the products involved, a questionnaire was sent to the European Poisons Centres as listed by EAPCCT (European Association of Poisons Centres and Clinical Toxicologists, 2003). Responses from eight of the listed institutions were received. The results are summarised in **Table 4.9** at the end of this section. In this table the total numbers of accidents with sodium hydroxide as single agent or associated with other agents are given for the period 2000-2003. Furthermore, percentages are given on the route of exposure and numbers of accidents occurring from consumer/worker exposure.

Accidental exposures described in literature

Inhalation exposure

For production and major uses of sodium hydroxide aerosols do normally not occur. However, for certain specific uses, e.g. cleaning ovens and disinfection of sheds, the formation of aerosols can not be excluded completely. For example the cleaning of ovens could result in an irritation of the throat due to the presence of sodium hydroxide in the air. However, it should be realised that aerosols of sodium hydroxide are not stable. They are rapidly transformed due to an uptake of carbon dioxide from the atmosphere resulting in the formation of sodium bicarbonate and sodium carbonate. The transformation of respirable sodium hydroxide aerosols into sodium carbonate aerosols can occur in seconds (Cooper et al., 1979).

Ocular exposure

A total of 23 burns of the eye due to NaOH or KOH were admitted to the eye clinic of the RWTH Aachen in Germany from 1985 to 1992 (Kuckelkorn et al., 1993). In 17 cases the accident happened during work, while 6 cases occurred at home using NaOH/KOH as drain cleaner. The alkali burns were of special interest because of the rapid and deep penetration of alkali into the ocular tissues.

From January 1984 to June 1991 a total number of 24 patients were treated for NaOH related eye injury in the Department of Ophthalmology, Postgraduate Institute of Medical Education and Research, Chandigarh, India (Saini et al., 1993). Over half of the patients which had ocular chemical burns were young people working in laboratories and factories.

Oral exposure

According to Schober et al. (1989) between January 1976 and October 1988 a total number of 6 cases of ingestion of NaOH was reported by the Children Surgery Department (University of Graz, Austria). The University Hospital of Santiago de Compostela (Spain) reported about 67 cases of accidental ingestion of NaOH by children between 1981 and 1990 (Casasnovas et al., 1997). Most of the accidents occurred at home and the container was located within easy reach of the children. A nationwide survey of ingestion of corrosives has been performed for the period 1984-1988 in Denmark (Clausen et al., 1994). It revealed 57 admissions to hospital of children (0-14 years) due to NaOH ingestion. The authors were confident that all children with serious complications after ingestion of corrosives were included in the study.

At the Department of Paediatric Surgery (Adana, Turkey) 71 cases of NaOH ingestion by children were reported in a period of 12 years (Keskin et al., 1991). On the West Bank of Israel a total number of 29 children were admitted to hospital due to accidental NaOH ingestion between 1990 and 1997 (Yasser et al., 1998). Lye is used in this area for home made soap. At the Shands Hospital at the University of Florida 15 children were admitted between 1973 and 1984 which had ingested NaOH (Moazam et al., 1987).

All previously mentioned publications reported accidental ingestion of NaOH by children. Wijburg et al. (1985) reviewed the records of 170 patients admitted to the Department of Otolaryngology of the University Hospital of Amsterdam in the period January 1, 1971 to December 31, 1981 with suspected caustic ingestion. Of these 170 patients about 15 patients had ingested NaOH. Only in this case it was not clear if children were involved.

Current information on accidental exposures from Poisons Centres

The Dutch NVIC (Nationaal Vergiftigingen Informatie Centrum) recorded 272 accidents in the period 2000-2003. About 85% of these cases were consumer accidents mainly with drain openers (96% of cases) or oven cleaners. Ingestion was the main route of exposure (Scholtens, personal communication, 2004).

The Belgian Posion Centre recorded 277 sodium hydroxide exposures during the year 2003. Exposures were mostly accidental (96%) and involved adult patients (87.7%). Projection on the skin was the main route of exposure. After these accidents about 90% of the cases required medical intervention. Drain openers and products containing sodium hydroxide as a major ingredient were involved in most of the accidents (de Coninck and Mostin, personal communication, 2004).

The Scottish Poisons Information Bureau reported information on accidental and deliberate exposures to both sodium hydroxide itself and products containing sodium hydroxide as a major or minor ingredient for the period 2000-2003. Of the reported 112 cases about 98% were accidental. Ingestion/mouth contact was the main exposure route. Products involved in consumer accidents were cleaning products, household liquid bleach, oven cleaners, drain openers and sodium hydroxide/caustic soda as basis product (Good, personal communication, 2004).

Ireland recorded 222 accidents with sodium hydroxide in the period 2000-2003. Most of the accidents occurred after dermal or oral exposure to caustic soda granules, drain openers, oven cleaners or other sodium hydroxide products (Donohoe, personal communication, 2004).

In the Slovak republic 37 accidents with sodium hydroxide were reported to the Toxicological Information Centre in the period 2000-2003. All accidents occurred from consumer exposure by the oral route. Accidents happened after use of siphon cleaners (majority of cases), toilet cleaners, oven cleaners, limescale and blemish removers. In two kinds of these siphon cleaners the amount of sodium hydroxide was up to 100% (Klobusicka, personal communication, 2004).

The National Poisons Information Service in Newcastle upon Tyne (United Kingdom) reported 182 cases for the period 2000-2002. Of these cases about 73% were accidents during consumer use. Main exposure route was by skin. Oven cleaners account for the majority of household exposures. Degreasers, drain openers and general cleaners were also important in household exposures (Weatherall, personal communication, 2004).

The Swiss Toxicological Information Centre recorded 295 cases in the period 2000-2003. Occupational and consumer exposure occurred in the same order of magnitude. Dermal exposure was the main route and the products mainly involved were: drain openers (with NaOH concentration 1-98%), oven cleaners (5% NaOH concentration) and undetermined cleaners (0.5-25% of NaOH) (Kupferschmidt, personal communication, 2004).

From one institution in Germany (Informationszentrale gegen Vergiftigungen, Bonn) information was received on 114 cases of sodium hydroxide and drain pipe opener exposure in the period 1999-2003. Most of these cases were accidents with drain pipe openers after oral and inhalation exposure. The drain pipe opener contains up to 52% of sodium hydroxide (Gräf, personal communication, 2004).

Table 4.9 Information from European Poisons Centres on accidents involving sodium hydroxide

	The Netherlands	Belgium	Scotland	Ireland	Slovak Republic	United Kingdom	Switzerland	Germany
Number of cases with NaOH (as single agent/with other agents) in year:								
2000	55	No data	27	58	8	69	67	25
2001	59	No data	29	53	4	68	78	35
2002	79	No data	27	67	4	45	89	25
2003	79	277	29	44	21	49	61	29
Total	272	277	112	222	37	231	295	114
Exposure route:								
Skin	11%	51%	17%	33%	0%	42%	40%	21%
Ingestion/mouth contact	75%	21%	42%	44%	100%	25%	27%	40%
Eye	3%	12%	17%	15%	0%	20%	13%	4%
Inhalation	11%	12%	14%	8%	0%	8%	20%	36%
Injection		0%	<1%	0%	0%	0%		
More than one route		4%	9%	No data	No data	No data		
unknown						5%		

Table 4.9 continued overleaf

Table 4.9 continued Information from European Poisons Centres on accidents involving sodium hydroxide

	The Netherlands	Belgium	Scotland	Ireland	Slovak Republic	United Kingdom	Switzerland	Germany
Victims age								
Adults Children	No data	243 34 (22 in the 0-4 group)	No data	No data	No data	No data	No data	No data
Circumstances of exposure						No data for 2003		
Accidental								
– consumers	232	245	64	172	37	133	161	94
– workers	40	20	26	50	0	44	120	8
Suicide	No data	10	2	No data	No data	No data		12
Not identified		2	20			6	14	

Conclusions and summary

From this questionnaire it can be concluded that most of the consumers' accidents were the result of using products like drain cleaners and oven cleaners. The main route of exposure is after ingestion, followed by dermal exposure. Most accidents happen during consumer use and one study indicated that adults are mainly involved in these accidents. It should be noted that the cases described above do not represent all incidents that occurred in the individual countries. Patients may seek immediate medical advice or present to a hospital ward without calling a Poison Centre. Medical professionals familiar with corrosive exposure will treat the patient without notifying the Poison Centre. Nevertheless, the results of the questionnaire show, that many accidental exposures to sodium hydroxide occur at home. Sodium hydroxide in drain openers is the main source of exposure. Manipulation without skin or eye protection could be an explanation for most of the accidents. Inexperienced users may mix various corrosive and/or bleach products to obtain better results, which can result in vigorous reactions.

4.1.1.3.3 Summary of consumer exposure

Exposure to sodium hydroxide during general and accidental use of floor strippers, oven cleaners and drain openers can be summarised by the following table:

Table 4.10 Overview of consumer exposure

Scenario	Maximum content of NaOH	Potential dermal uptake after general use (mg/kg event)	Potential inhalatory uptake after general use (mg/kg event)	Accidental exposures in 2000-2003 in some European countries
I Floor strippers	10%	Negligible	Not relevant	About 37-277 cases (total per country)
II Hair straighteners	2%	23	Not relevant	
III Oven cleaners	5%	Negligible	Negligible	
IV Drain openers	30%	Negligible	Not relevant	

The systemic exposure in all scenarios can be considered to be negligible. It should be noted, that it is unlikely that sodium ions penetrate the skin to a considerable extent. In an extreme

worst case assumption dermal absorption of these ions will be 1-10% following recommendations of the TGD (EC, 2003). This would lead to a 1 to 2 orders of magnitude lower systemic dose then described above. This amount is negligible compared to the daily dietary intake of sodium ions.

In the risk characterisation the direct exposure expressed in percentages of sodium hydroxide will be discussed as well as the accidental exposure.

4.1.1.4 Humans exposed via the environment

Production and use of sodium hydroxide is normally not expected to increase the pH of the environment. Even after an accidental release the substance will be neutralised finally and therefore the human exposure to sodium hydroxide via the environment is expected to be negligible.

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

NaOH has been used for a long time and has wide dispersive use and therefore there is information on human exposure and effects. For this reason the human health hazard assessment is not only based on animal toxicity data but also on human experience (including medical data). For this unique situation it was thought more appropriate to discuss the animal data and human data together.

The major human health hazard (and the mode of action) of NaOH is local irritation and/or corrosion.

4.1.2.1 Toxicokinetics, metabolism and distribution

Sodium is a normal constituent of the blood and an excess is excreted in the urine. A significant amount of sodium is taken up via the food because the normal uptake of sodium via food is 3.1-6.0 g per day according to Fodor et al. (1999). Exposure to NaOH could potentially increase the pH of the blood. However, the pH of the blood is regulated between narrow ranges to maintain homeostasis. Via urinary excretion of bicarbonate and via exhalation of carbon dioxide the pH is maintained at the normal pH of 7.4-7.5.

When humans are dermally exposed to low (non-irritating) concentrations, the uptake of NaOH should be relatively low due to the low absorption of ions. For this reason the uptake of NaOH is expected to be limited under normal handling and use conditions. Under these conditions the uptake of OH^-, via exposure to NaOH, is not expected to change the pH in the blood. Furthermore the uptake of sodium, via exposure to NaOH, is much less than the uptake of sodium via food under these conditions. For this reason NaOH is not expected to be systemically available in the body under normal handling and use conditions.

An example will be given for an inhalation exposure scenario. Assume an exposure to an NaOH concentration of 2 mg/m^3, which is the TLV in the USA, and a respiratory volume of 10 m^3 per day. In this case the daily exposure is 20 mg NaOH.

The amount of 20 mg NaOH is equivalent with 11.5 mg sodium which is a negligible amount compared to the daily dietary exposure of 3.1-6.0 g (Fodor et al., 1999). The amount of 20 mg

NaOH is equivalent with 0.5 mmole and if this amount would be taken up in the blood stream it would result in a concentration of 0.1 mM OH^- (assuming 5 litre blood per human). This is a negligible amount when it is compared with the bicarbonate concentration of 24 mM of blood. This example confirms that NaOH is not expected to be systemically available in the body under normal handling and use conditions.

4.1.2.2 Acute toxicity

4.1.2.2.1 Studies in animals

Dermal

The hair of adult mice was clipped and a circular area 2 cm in diameter was painted by applicator with 50% NaOH (Bromberg et al., 1965). Afterwards the area was rinsed with water at various intervals. The mortality of mice was 20, 40, 80 and 71% when they were rinsed 30 minutes, 1 hour, 2 hours or not at all after the application. The animals were observed daily for up to 7 days after the treatment. All animals developed rapidly progressive burns. No mortality or burns were observed when the mice were rinsed immediately after the application.

Oral

No acute oral toxicity study with animals has been carried out using (inter)national guidelines. An acute oral study with 1-10% NaOH and rabbits revealed an LD_{50} of 325 mg/kg bw expressed as 100% NaOH (Naunyn-Schmiedeberg's, 1937). Mortality was also observed when 1% NaOH was dosed but in this case the applied volume was relatively high (24 ml per kg body weight). Another acute oral toxicity study has been reported in secondary literature but the original reference could not be found. This study indicated an LDL0 of 500 mg/kg bw in the rat. The gastric erosive activity of NaOH was studied with rats using a maximum erosion score of 100 (Van Kolfschoten et al., 1983). NaOH concentrations of 0.4; 0.5 and 0.62% resulted in erosion scores of 10, 65 and 70%, respectively.

4.1.2.2.2 Studies in humans

Inhalation

No animal data are available on the acute inhalation toxicity. However, the inhalation of aerosols of 5% NaOH by a 25-year-old woman resulted in irreversible obstructive lung injury after working for one day in a poorly ventilated room (Hansen et al., 1991). Besides NaOH the product contained also smaller amounts of calcium carbonate, soft soap and protein.

Dermal

A fatal burn due to dermal NaOH exposure of a worker at an aluminium plant has been reported (Lee et al., 1995). He was found lying in a shallow pool of concentrated NaOH, which had been heated to ~95°C.

Oral

The degree and type of injury after ingestion of NaOH depend on the physical form. Solid NaOH produces injury to the mouth and pharynx and is difficult to swallow. On the other hand liquid NaOH is easily swallowed, being tasteless and odourless, and is more likely to damage the esophagus and stomach (Gumaste et al., 1992).

Cello et al. (1980) described 9 cases of liquid NaOH ingestion, which resulted in esophageal and gastric injury. One person who ingested 10 g NaOH in water suffered transmural necrosis of the esophagus and stomach and died 3 days after admission to the hospital. A 42-year-old female swallowed approximately 30 ml of 16% NaOH in a suicide attempt (Hugh et al., 1991). This resulted in a 9-cm stricture of the esophagus which was treated by gastric antral patch esophagoplasty.

4.1.2.2.3 Summary of acute toxicity

NaOH is a corrosive substance and for this reason there is no need for further acute toxicity testing.

4.1.2.3 Irritation/corrosion

4.1.2.3.1 Skin

Studies in animals

An *in vivo* test was conducted with Yorkshire weanling pigs using applications of 2N (8%), 4N (16%) and 6N (24%) NaOH on the lower abdominal region (Srikrishna et al., 1991). Gross blisters developed within 15 minutes of application and 8 and 16% NaOH produced severe necrosis in all epidermal layers. A concentration of 24% produced numerous and severe blisters with necrosis extending deeper into the subcutaneous tissue. Also an *in vitro* test was performed with isolated perfused skin flaps of Yorkshire weanling pigs using NaOH concentrations of 4N (16%) and 6N (24%). At both concentrations NaOH showed severe necrosis of all epidermal cell layers and dermis. At times this lesion extended deep into the subcutaneous layers.

Jacobs (1990) evaluated a publication by Young et al. (1988), in which three New Zealand White rabbits were exposed to a concentration of 0.36% NaOH, which is the lowest limit concentration that was calculated using dissociation constant. No skin irritation/corrosion was observed at that concentration. Therefore, an additional study was performed with one animal exposed to the highest concentration (5%). This concentration showed to be corrosive at all observation time points (1, 24, 48, 72 and 144 hours after removal of exposure chamber).

Sodium hydroxide has also been used extensively for *in vitro* skin irritation testing. These studies are all considered invalid, because of an unsuitable test system or insufficient documentation.

Skin explants of female hairless mice were exposed to concentrations of 500, 1000, 2500 and 5000 μg/cm^2 skin (Bartnik et al., 1990). The effects of NaOH were underestimated when only the results of enzyme release and glucose utilisation were assessed. NaOH caused its

destructive effects only by its high pH value and was partly neutralized by the incubation system.

An *in vitro* study, in which Skin model ZS 1300 was exposed to 10% sodium hydroxide, showed a 50% reduction in cell viability in 2.4 minutes, from which this chemical can be classified as corrosive (Perkins et al., 1996).

The skin of Danish Landrace pigs was exposed to NaOH in concentrations up to 1 N NaOH (Karlsmark et al. 1988). After application of NaOH dispersed collagen fibres showed increased eosinophilia and a fine densely spaced cross-striation in polarized light and vesicular nuclei were present within dermal cells. During the following days a narrow demarcation zone of neutrophilic granulocytes separated the zone containing abnormal collagen fibres from normal tissue.

NaOH was applied to the abdomens of 20 rats in a concentration of 2N NaOH (Yano et al., 1993). Afterwards the area was washed with 500 ml distilled water starting at 1, 10 and 30 minutes postinjury. After injury the subcutaneous tissue pH had not recovered to the pre-experimental level by the 90th minute. When washing started within 1 minute of injury the tissue pH value did not exceed 8. Washing had no effect when the delay between injury and the start of washing was 10 and 30 minutes.

Studies in humans

The valid *in vivo* skin irritation studies with solutions of NaOH are summarised in **Table 4.11**. Studies were valid if they were well documented and if they met generally accepted scientific principles.

A NaOH concentration of 0.5% was tested within an interlaboratory evaluation of a human patch test for the identification of skin irritation hazard (Griffiths et al., 1997). A 25 mm Plain Hill Top Chamber containing a Webril pad was used and the treatment sites were assessed for irritation using a four-point scale at 24, 48 and 72 hours after initiation of exposure. NaOH 0.5% was irritating for 55% of the volunteers.

A human skin irritation test with 0.5% NaOH was performed using exposure periods of 15, 30 and 60 minutes (York et al., 1996). The treatment sites were assessed 24, 48 and 72 hours after patch removal. The results showed that after a maximum exposure of 60 minutes, 61% of the volunteers (20 of 33) showed a positive skin irritation reaction.

Four different patch systems, Finn chamber, Hill Top patch, Van der Bend chamber and Webril patch, were used to determine the skin irritation response of 1% NaOH (York et al., 1995). Webril and Hill top patches generated the greatest levels of response. Eleven of 14 and 5 of 14 volunteers showed a positive skin reaction after 30 minutes for Webril and Hill top patches, respectively. With Finn and Van der Bend chambers 5 of 14 and 7 of 14 volunteers showed a positive reaction after 4 hours, respectively, which shows that the reactivity was reduced with these systems.

Table 4.11 Human *in vivo* skin irritation tests with NaOH

Test Type	Protocol	Concentration	Result	Reference
Human, upper outer arm	0.2 ml applied to a Plain Hill Top Chamber with Webril pad, 1 h exposure	0.5%	Irritating for 55% of the volunteers	Griffiths et al. (1997)
Human, upper outer arm	Human patch testing with Hill Top Chambers, exposure between 15 and 60 min, 0.2 ml	0.5%	Positive irritant for 61% of volunteers	York et al. (1996)
Human, intact skin	Four different protocols, ≤ 4 hours	1.0%	Positive irritant for about 50% of volunteers	York et al. (1995)
Human, intact skin of back and forearm	Filter disc with 70 µl solution, 3, 15 and 60 min exposure	0.5 and 1%	Irritating (mainly erythema).	Dykes et al. (1995)
Human, volar side of forearm	Filter disc with 40 µl solution, 24 h exposure	1, 2 and 4%	Normal-reacting and hyper reactive subjects	Seidenari et al. (1995)

The cutaneous response to NaOH has been assessed in human volunteer subjects using both clinical scoring and two non-invasive instrumental methods; erythema measurement using an erythema meter and capillary blood flow using a laser Doppler device (Dykes et al., 1995). Solutions of 0.5 and 1% NaOH were applied to back skin for 3, 15 and 60 minutes with assessments immediately after removal and at 1, 24 and 48 hours. Increased erythema was seen with increasing duration of exposure and an increase was also seen at 1, 24 and 48 hours after removal of the patch. Comparison between back and forearm skin indicated a greater sensitivity to NaOH on the back.

Sodium hydroxide induced irritation was studied in 34 volunteers by means of 24-hour patch testing at different concentrations and by a short-term test using an exposure duration of 10 minutes (Seidenari et al., 1995). The 24-hour patch test with 4% NaOH revealed a classification of subjects in 2 categories: subjects who reacted normally (25 of 34) and hyper-reactors (9 of 34). Hyper-reactors showed an enhanced inflammatory response, a decreased dermal reflectivity and an increase in transepidermal water loss.

According to the 19th ATP (from 1993) of Annex I of Council Directive 67/548/EEC, the concentration limit for corrosivity of NaOH is considered to be 2%. Up to the most recent ATP (29th; April 2004), this has not been changed. Therefore, 2% is taken forward to the risk characterisation as concentration limit for corrosivity.

4.1.2.3.2 Eye

Studies in animals

The valid eye irritation studies conducted with NaOH solutions are summarised in **Table 4.12**. Studies were valid if they were well documented and if they met generally accepted scientific principles.

Table 4.12 *In vivo* eye irritation tests with NaOH

Species	Protocol	Concentrations	Result	Reference
Rabbits	Dose of 0.1 ml in lower conjunctival sac of left eye	0.004; 0.04; 0.2; 0.4 and 1.2%	0.004-0.2: non-irritant 0.4%: mild irritation 1.2% corrosive	Morgan et al. (1987)
Rabbits	Dose of 0.1 ml, washed (after 30 s) and unwashed eyes	0.1; 0.3; 1.0 and 3.0%	0.1 and 0.3%: no conjunctivitis nor iritis 1.0 and 3.0%: conjunctivitis and iritis	Murphy et al. (1982)
Rabbits	OECD Guideline 405	1 and 2%	1%: Not irritating 2%: Irritating	Jacobs (1992)

A volume of 0.1 ml NaOH was placed in the lower conjunctival sac of the left eye of Stauffland Albino rabbits (Morgan et al., 1987). Both the left and the right eye were evaluated for irritation and corneal thickness for up to 21 days using a slit-lamp biomicroscope with a pachymeter attachment. According to EPA criteria 0.001M (0.004%), 0.01M (0.04%) and 0.05M (0.2%) NaOH were considered non-irritant, while the irritation at 0.1M (0.4%) was mild and 0.3M (1.2%) was considered corrosive.

The severity of the effects are influenced by the exposure amount, concentration, duration and the treatment. Alkaline substances produce a liquefaction necrosis and therefore are able to penetrate the tissue (Murphy et al., 1982). When an amount of 100 µl was instilled into the eyes of rabbits concentrations of 1.0 and 3.0% resulted in conjuctivitis which lasted through 7 days, while concentrations of 0.1 and 0.3% did not.

Based on eye irritation tests with New Zealand White Albino rabbits, conducted according to OECD Guideline 405, a concentration of 1% NaOH is not irritating to eyes while a concentration of 2% was irritating to the eyes (Jacobs, 1992). A volume of 100 µl was instilled into the lower conjunctival sac and the classification was based on EC criteria. A concentration of 2% was irritating due to the mean score for conjunctivitis and the mean score for corneal opacity.

4.1.2.3.3 Respiratory tract

Studies in humans

The effects of inhalation exposure to NaOH have not been reliably studied. In survey documents of the ACGIH (2001) and the OEHHA (1999) studies with regard to respiratory tract irritation are mentioned (Patty's (1949), Hervin and Cohen (1973) and NIOSH (1974 and 1976)). In the first edition of 'Patty's', published in 1949, a concentration of 2 mg NaOH/m^3 of air was considered "a concentration that is noticeably, but not excessively, irritating" based on irritant effects of caustic mists encountered in concentrations of 1-40 mg/m^3 of air. Hervin and Cohen (1973) described burning/redness of the nose, throat, or eyes among workers engaged in cleaning operations where airborne concentrations of NaOH between 0.005 and 0.7 mg/m^3 were found. However, solvents, including Stoddard solvent, were also present at concentrations as high as 780 mg/m^3. NIOSH (1974 and 1976) reported some cases of acute respiratory symptoms with nose and throat irritation, chest pains, and shortness of breath

following exposure to NaOH. NIOSH considered these data, however, inadequate for correlation of exposure and effect.

Ott et al. (1977) investigated workers from two production areas exposed to estimated (based on measurements and subjective response data) NaOH time-weighted average (TWA) levels of 0.5 mg/m^3 (production area 1) and 0.5-2 mg/m^3 (production area 2). The number of visits to a Medical Department for episodes of mild (i.e. transient) respiratory irritation were 0.4 and 0 visits per 100 person years for 0.5 mg/m^3 and 0.5-2 mg/m^3 NaOH, respectively. The number of visits to a Medical Department for episodes of moderate severe (i.e. objective damage) respiratory irritation were 0.1 and 0.2 visits per 100 person years for 0.5 mg/m^3 and 0.5-2 mg/m^3 NaOH, respectively.

A cross-sectional survey of 2404 employees in three alumina refineries was performed in 1996 (Fritschi et al., 2001). The participants answered questions about respiratory symptoms and the relationship of those symptoms to work, as well as having spirometry and providing a complete job history. Over 40% of the subjects was currently exposed to caustic mist of NaOH. For caustic mist, the usual hygiene monitoring practice at the refineries was to perform static monitoring in specified locations over a 15-min period, with the sampling heads placed close to the breathing zone of the worker. These samples do not provide information on the duration of exposure for individuals, since the tasks often involve moving in and out of the monitored regions. Since the patterns of exposure to caustic mist are reasonably predictable in a particular task, it was decided to use a semi-quantitative measure to categorise peak exposure to caustic mist. The site hygienists at each of the three refineries estimated which tasks involved exposure to caustic peaks and used available data to classify those tasks into one of three groups: low (< 0.05 mg/m^3), medium (0.05-1.0 mg/m^3) or high (> 1.0 mg/m^3). Each subject was classified according to the highest peak exposure in any of the current tasks performed in the job held at the time of the study. Possible effects due to duration or frequency of the peak exposures could not be examined in the analysis. No account was taken of jobs held prior to the current position as the hygienists were not confident they could accurately estimate caustic mist exposures in previous jobs. Subjects in the highest group of current caustic exposure reported increased prevalence of work related wheeze (Prevalence ratio = 1.8; 95%; CI: 1.0-3.1) and rhinitis (Prevalence ratio = 1.6; 95%; CI: 1.1-2.4), but did not have measurable changes in lung function. It was noted by the authors that the peak levels in the refineries from the highest group (> 1.0 mg/m^3) were lower than the recommended ceiling level (TLV-value) of 2 mg/m^3. Furthermore, the results were not changed when the analysis was restricted to those who had ever worked in a production job.

The studies of Ott et al. (1977) and Fritschi et al. (2001) are considered the most useful and reliable studies for risk characterisation of respiratory tract irritation. The results of the study of Ott et al. (1977) are based on visits to a medical department, while the results of the study of Fritschi et al. (2001) are based on questionnaires. The questionnaires are considered to give a more representative picture of respiratory tract irritation among workers since it is not expected that all workers with respiratory tract irritation have in fact visited a medical department. Therefore, the concentration of 1.0 mg/m^3 from the study of Fritschi et al. (2001) is considered a NOAEL for local effects to the respiratory tract.

4.1.2.3.4 Summary of irritation/corrosion

According to the 19th ATP (from 1993) of Annex I of Council Directive 67/548/EEC, the concentration limit for corrosivity of NaOH is considered to be 2%. Up to the most recent

ATP (29th; April 2004), this has not been changed. Therefore, 2% is taken forward to the risk characterisation as concentration limit for corrosivity.

Based on human data concentrations of 0.5–4% were irritating. In 2 different studies a concentration of 0.5% was irritating for 55 and 61% of the volunteers, respectively.

Based on a study among workers, concentrations up to 1.0 mg/m^3 are not considered adverse with regard to respiratory tract irritation.

The available animal data on eye irritation revealed small differences in eye irritation levels. The non-irritant level was 0.2-1.0%, while the corrosive concentration was 1.2%.

4.1.2.4 Sensitisation

Data on skin sensitisation were reported by Park et al. (1995). Male volunteers were exposed on the back to sodium hydroxide concentrations of 0.063 – 1.0% (induction). After 7 days the volunteers were challenged to a concentration of 0.125%. The irritant response correlated well with the concentration of NaOH, but an increased response was not observed when the previously patch tested sites were rechallenged. Based on this study sodium hydroxide has no skin sensitisation potential. Furthermore NaOH has been used widely and for a long time and no human cases of skin sensitisation have been reported and therefore NaOH is not considered to be a skin sensitiser.

4.1.2.5 Repeated dose toxicity

4.1.2.5.1 Studies in animals

Inhalation

Two repeated dose inhalation studies in rats were available (Dluhos et al. (1969) and Vyskocil et al. (1966)). The specific exposure concentrations were however not reported. In the study by Dluhos et al. (1969) rats were exposed by inhalation to an unknown concentration of NaOH produced from an aerosolised 40% solution for 30 minutes twice daily for 2.5 months. After 3 weeks, exposure was interrupted for 10 days, because animals badly tolerated exposure. Lung examination revealed alveolar wall thickening with cell proliferation and congestion. Ulceration and flattening of the bronchial epithelium and proliferation of lymphadenoid tissue were also reported. Undescribed, isolated tumors were observed in 3 of 10 animals. In the study by Vyskocil et al. (1966), inhalation exposure twice weekly for one month to an aerosol (concentration unspecified) produced from a 40% NaOH solution resulted in the deaths of all 27 rats, predominantly from bronchopneumonia. Exposure to an aerosol produced from a 20% solution of NaOH produced dilatation and destruction of alveolar septae. Although no effects were observed in the group exposed to a 10% solution, in rats exposed to aerosolised 5% sodium hydroxide, bronchial dilatation and mucus membrane degeneration were observed, which suggest a poor dose-response relationship in this study.

Dermal

No animal data are available on repeated dose toxicity studies by the dermal route for NaOH.

Oral

One limited study conducted by Merne et al. (2001) is available in which the systemic (organ) and local (oral mucosal) effects of alkalinity was assessed. For this, drinking water supplemented with $Ca(OH)_2$ or NaOH, with pH 11.2 or 12 was administered to rats (n = 36) for 52 weeks. Tissues were subjected to histopathological examination; oral mucosal biopsy samples were also subjected to immunohistochemical (IHC) analyses for pankeratin, CK19, CK5, CK4, PCNA, ICAM-1, CD44, CD68, S-100, HSP 60, HSP70, and HSP90. At completion of the study, animals in the study groups had lower body weights (up to 29% less) than controls despite equal food and water intake, suggesting a systemic response to the alkaline treatment. The lowest body weight was found in rats exposed to water with the highest pH value and starting the experiment when young (6 weeks). No histological changes attributable to alkaline exposure occurred in the oral mucosa or other tissues studied. Alkaline exposure did not affect cell proliferation in the oral epithelium, as shown by the equal expression of PCNA in groups. The up-regulation of HSP70 protein expression in the oral mucosa of rats exposed to alkaline water, especially $Ca(OH)_2$ treated rats, may indicate a protective response. Intercellular adhesion molecule-1 (ICAM-1) positivity was lost in 6/12 rats treated with $Ca(OH)_2$ with pH 11.2, and loss of CD44 expression was seen in 3/6 rats in both study groups exposed to alkaline water with pH 12. The results suggest that the oral mucosa in rats is resistant to the effects of highly alkaline drinking water. The observed effects on growth can be explained by NaOH neutralising the acid in the stomach which decreases the digestion and the absorption of the food.

4.1.2.5.2 Studies in humans

Inhalation

A 63 year old man was exposed daily for 20 years to mists of NaOH which was probably the cause for the obstructive airway disease which was observed (Rubin et al., 1992). The exposure was heavy but was not quantified and therefore the study has a limited value.

Dermal

No human data are available on repeated dose toxicity studies by the dermal route for NaOH.

Oral

The hazard of repeated human exposure to sodium has been focussed on the effects of sodium on the prevention and control of hypertension. Recommendations on dietary salt intake have been published by Fodor et al. (1999). A daily dietary intake of 2.0-3.0 g was reported to be a moderately restricted intake, 3.1-6.0 was reported as a normal intake, while a dietary intake of > 6 g sodium per day was considered an excessive intake.

4.1.2.5.3 Summary of repeated dose toxicity

Although two inhalation studies showed local effects of the respiratory tract after repeated NaOH exposure, the data were not adequate to establish a N(L)OAEL because the exposure concentrations were not specified.

A limited oral drinking water study with rats revealed effects on growth, which can be explained by NaOH neutralising the acid in the stomach which decreases the digestion and the absorption of the food. Therefore, the results of this test cannot be used for the risk characterisation. In addition the usefulness of this test can be doubted, because the long term hazard of sodium for humans has been characterised sufficiently. Furthermore, oral studies with high concentrations of the substance are corrosive or irritating, while at low concentrations the hydroxide will be neutralised in the stomach by gastric juice, which has a very low pH. Furthermore it should be realised that oral exposure to NaOH is negligible under normal handling and use conditions

4.1.2.6 Mutagenicity

4.1.2.6.1 Studies *in vitro*

NaOH was assayed in the Ames reversion test with *S. typhimurium* strains TA1535, TA1537, TA1538, TA98, TA100 and in a DNA-repair test with *E. coli* strains WP2, WP67 and CM871 (De Flora et al., 1984). Based on the results of these tests NaOH was classified as non genotoxic.

The clastogenic activity of NaOH was studied in an *in vitro* chromosomal aberration test using Chinese hamster ovary (CHO) K1 cells (Morita et al., 1989). No clastogenic activity was found at NaOH concentrations of 0, 4, 8 and 16 mM NaOH, which corresponded with initial pH values of 7.4, 9.1, 9.7 and 10.6, respectively. Incubation of CHO-K1 cells with NaOH in the presence of rat liver S9 increased the clastogenic activity of S9, or induced new clastogens by breakdown of the S9. Therefore, testing at non-physiological pH might give false-positive responses, which means that the effect of sodium hydroxide is of a methodical kind and not valid to asses the genotoxicity under realistic conditions.

4.1.2.6.2 Studies *in vivo*

Valid *in vivo* genotoxicity studies are not available.

A mouse bone marrow micronucleus test using 15 mM NaOH at a dose of 10 mg/kg bw revealed no significant increase of nuclei (Aaron et al., 1989). The test compound was administered as a single i.p. dose to treatment groups (5 males and 5 females) at 30, 48 and 72h. Mouse oocytes of the Swiss strain were used to determine possible aneuploidy-inducing effects (Brook et al., 1985). Mice were injected intraperitoneally with 0.3-0.4 ml of 0.01 M NaOH and chromosome spreads were made 12 h after injection. NaOH was used as control substance. No evidence of non-disjunction was found in control groups up to the age of 40 weeks tested.

4.1.2.6.3 Summary of mutagenicity

Both the *in vitro* and the *in vivo* genetic toxicity test indicated no evidence for a mutagenic activity. Furthermore NaOH is not expected to be systemically available in the body under normal handling and use conditions and for this reason additional testing is considered unnecessary.

4.1.2.7 Carcinogenicity

NaOH did not induce mutagenicity in *in vitro* and *in vivo* studies. Systemic carcinogenicity is not expected to occur because NaOH is not expected to be systemically available in the body under normal handling and use conditions. Finally, no suitable studies are available to assess the risk on local carcinogenic effects.

4.1.2.8 Toxicity for reproduction

No valid studies were identified regarding developmental toxicity nor toxicity to reproduction in animals after oral, dermal or inhalation exposure to NaOH.

It is not useful to do a reproduction or developmental toxicity test with NaOH in rats because the hazard of sodium for humans has been characterised sufficiently (e.g. Fodor et al., 1999). It is also not useful to study the reproduction/developmental toxicity of hydroxide via an oral study because at high concentrations the substance is corrosive or irritating, while at low concentrations the hydroxide will be neutralised in the stomach by gastric juice, which has a very low pH. Furthermore, it should be realised that oral exposure to NaOH is negligible under normal handling and use conditions and therefore an oral reproduction/developmental toxicity study is inappropriate.

NaOH is not expected to be systemically available in the body under normal handling and use conditions and for this reason it can be stated that the substance will not reach the foetus nor reach male and female reproductive organs. It can be concluded that a specific study to determine the developmental toxicity or the toxicity to reproduction is not necessary.

4.1.3 Risk characterisation

4.1.3.1 Workers

Assuming that oral exposure is prevented by personal hygienic measures, the risk characterisation for workers is limited to the dermal and inhalation routes of exposure. The focus is the occurrence of local effects after acute and repeated exposure at those places where NaOH is produced and/or used. This is because NaOH is not expected to become systemically available in the body under normal handling and use conditions, i.e. neither the concentration of sodium in the blood nor the pH of the blood will be increased.

In the scope of the assessment of existing substances, dermal exposure to corrosive concentrations is not assessed. For the handling of corrosive substances and formulations, it is assumed that daily dermal exposure can be neglected because workers are protected from dermal exposure and immediate dermal contacts occur only accidentally. Techniques and equipment (including PPE) are used that provide a high level of protection from direct dermal contact. Eye protection is obligatory for activities where direct handling of NaOH occurs.

However, dermal exposure to non-corrosive dilutions of NaOH (concentrations < 2%) also occurs. Dermal exposure to such non-corrosive dilutions of NaOH will be taken into account. Furthermore, acute and repeated inhalation exposure to NaOH cannot be neglected.

4.1.3.1.1 Acute toxicity

NaOH is not expected to be systemically available in the body under normal handling and use conditions and therefore acute systemic effects of NaOH after acute dermal or inhalation exposure are not expected to occur (**Conclusion (ii)**). Acute local effects after dermal and inhalation exposure are assessed in Section 4.1.3.1.2.

4.1.3.1.2 Irritation and corrosivity

Skin

NaOH is considered to be a severe corrosive agent (concentrations ≥ 2%). Workers can be exposed to corrosive concentrations. However, dermal exposure to NaOH is considered to occur only accidentally if the required protection is strictly adhered to. Therefore, conclusion (ii) is justifiable for scenarios in which corrosive concentrations of NaOH are handled.

Dermal exposure to irritating, but non-corrosive, dilutions of NaOH (concentrations < 2%) also occurs. No quantitative data from animal or human studies on skin irritating effects of NaOH are available which could be used for the risk characterisation. When existing controls (i.e. engineering controls and personal protective equipment based on classification and labelling with R38) are applied conclusion (ii) is applicable. In addition, with regard to the exposure scenario 'End-use formulated products – Hair straightening products', it is referred to the Cosmetics Directive 76/768/EEC. According to this Directive, a concentration up to 4.5% of NaOH is authorised for professional use of hair straighteners.

Theoretically, NaOH is of concern for workers with regard to eye effects because of the corrosive properties of NaOH (concentration of 1.2%) to the eye in an animal study (Morgan et al., 1987). However, eye protection is obligatory for activities where direct handling of corrosive NaOH occurs. If the required protection is strictly adhered to, exposure will occur only incidentally, so conclusion (ii) is justifiable for scenarios in which corrosive concentrations of NaOH are handled.

Exposure to irritating, but non-corrosive, dilutions of NaOH also occurs (concentrations < 2%). When existing controls (i.e. engineering controls and personal protective equipment based on classification and labelling with R36) are applied conclusion (ii) is applicable. In addition, with regard to the exposure scenario 'End-use formulated products – Hair straightening products', it is referred to the Cosmetics Directive 76/768/EEC. According to this Directive, a concentration up to 4.5% of NaOH is authorised for professional use of hair straighteners.

Respiratory tract

Starting point for the risk assessment of respiratory tract irritation is the study of Fritschi et al. (2001). In this study, exposure concentrations up to 1 mg/m^3 were not considered adverse with regard to local effects to the respiratory tract.

The MOS values between this no effect level of 1 mg/m^3 and the exposure levels of the different occupational scenarios are mentioned in **Table 4.13**. The MOSs are evaluated by comparison with the minimal MOS. According to the final draft TGD (EC, 2005), a default factor of 5 is proposed for intraspecies differences. However, as the study of Fritschi et al.

(2001) studied a relatively large random sample of workers (1,045 exposed and 1,553 unexposed workers), it is proposed to use a factor of 3 for intraspecies differences. Therefore, the minimal MOS is changed into 3. There is concern when the MOS is lower than the minimal MOS. For inhalation exposure to NaOH, it cannot be excluded that respiratory tract irritation may occur in the scenarios 'Production – Bagging NaOH', 'Uses – Aluminium', 'Uses – De-inking of waste paper' and 'Uses - Textile' (Conclusion (iii)). This conclusion is even more justified by the fact that the exposure values for the scenarios 'Production – Bagging NaOH', 'Uses – De-inking of waste paper' and 'Uses - Textile' represent 8-hour TWA values instead of short-term peak values.

Table 4.13 Occupational risk assessment for respiratory tract irritation

Scenario	Activity	Inhalation exposure value (mg/m³)	No effect level (mg/m³)	MOS value	Minimal MOS value	Conclusion
Production						
Drumming liquid NaOH	Full shift	0.33	1	3	3	ii
Bagging NaOH	Full shift	1.8	1	0.6	3	iii
Formulation						
Soap	Full shift	0.08	1	12.5	3	ii
End use formulated products						
Oven cleaner liquid	Full shift	Negligible	1	>>>>1	3	ii
Oven cleaner spray	Full shift	0.13	1	7.7	3	ii
Uses						
Chemical industry	Full shift	0.08	1	12.5	3	ii
Aluminium	Full shift	0.14	1	7.1	3	ii
	Short term	1.1	1	0.9	3	iii
Pulp/paper	Full shift	0.08	1	12.5	3	ii
De-inking of waste paper	Full shift	1.20	1	0.83	3	iii
Textile	Full shift	3.4	1	0.3	3	iii

4.1.3.1.3 Sensitisation

Skin

A study with human volunteers did not indicate skin sensitisation potential of NaOH, which is supported by extensive human experience.

Conclusion (ii).

Respiratory tract

No studies are available on respiratory sensitisation of NaOH. However, there are no reports on respiratory sensitisation, despite the long and widespread use of NaOH. Therefore, it is concluded that there is no concern for this endpoint.

Conclusion (ii).

4.1.3.1.4 Repeated dose toxicity

Inhalation

NaOH is not expected to be systemically available in the body under normal handling and use conditions and therefore systemic effects of NaOH after repeated inhalation exposure are not expected to occur.

Conclusion (ii).

The risk of respiratory tract irritation after acute peak exposures is addressed in Section 4.1.3.1.2 using the study of Fritschi et al. (2001). In this study, it is mentioned that the results were not changed when the analysis was restricted to people who had ever worked in a production job. Therefore, the same conclusions as specified in Section 4.1.3.1.2 can be drawn for local effects after repeated inhalation exposure. Thus, it cannot be excluded that respiratory tract irritation may occur in the scenarios 'Production – Bagging NaOH', 'Uses – Aluminium', 'Uses – De-inking of waste paper' and 'Uses - Textile' (**conclusion (iii)**) after repeated inhalation exposure.

Dermal

NaOH is not expected to be systemically available in the body under normal handling and use conditions and therefore systemic effects of NaOH after repeated dermal exposure are not expected to occur.

Conclusion (ii).

No repeated dermal dose data on local effects of NaOH is available. For the handling of corrosive substances and formulations, immediate dermal contacts occur only occasionally and it is assumed that repeated daily dermal exposure can be neglected because workers are protected from repeated dermal exposure and only accidental exposure may occur. Techniques and equipment (including PPE) are used that provide a very high level of protection from direct dermal contact. Therefore, it is concluded that NaOH is of no concern for workers with regard to repeated dermal exposure in scenarios in which corrosive concentrations of NaOH are handled.

Conclusion (ii).

Repeated dermal exposure to non-corrosive dilutions of NaOH also occurs. It is assumed that existing controls (i.e. engineering controls and personal protective equipment based on classification and labelling with R38) are applied for these exposure situations. Therefore, it is concluded that NaOH is of no concern for workers with regard to skin irritation for scenarios in which non-corrosive concentrations are handled

Conclusion (ii).

4.1.3.1.5 Mutagenicity

Given the results from the *in vitro* and *in vivo* mutagenic studies, it is concluded that NaOH is of no concern for workers with regard to mutagenicity.

Conclusion (ii).

4.1.3.1.6 Carcinogenicity

No suitable studies are available to assess the risk on carcinogenic effects. However, it is not expected that NaOH will induce tumors. First of all, NaOH did not induce mutagenicity in *in vitro* and *in vivo* studies. Secondly, systemic carcinogenicity is not expected to occur because NaOH is not expected to be systemically available in the body under normal handling and use conditions. Finally, the anticipated level of protection for dermal and inhalatory exposure is assumed to prevent possible carcinogenic responses that may be due to chronic local irritation. Therefore, it is concluded that NaOH is of no concern for workers with regard to carcinogenicity.

Conclusion (ii).

4.1.3.1.7 Toxicity for reproduction

NaOH is not expected to be systemically available in the body under normal handling and use conditions and for this reason it can be stated that the substance will not reach the foetus nor reach male and female reproductive organs. Therefore, it is concluded that NaOH is of no concern for workers with regard to toxicity for reproduction.

Conclusion (ii).

4.1.3.2 Consumers

As sodium hydroxide is not expected to become systemically available in the body under normal handling and use conditions, the risk characterisation for consumers will focus on possible risks from acute exposure (local effects).

4.1.3.2.1 Acute toxicity

NaOH is not expected to be systemically available in the body under normal handling and use conditions and therefore acute systemic effects of NaOH after acute oral, dermal, ocular or inhalatory exposure are not expected to occur (**Conclusion (ii)**). Acute local effects after oral, dermal, ocular and inhalatory exposure are assessed in Section 4.1.3.2.2. Irritation and corrosivity

Oral

During normal use the exposure of consumers to NaOH is considered negligible (Conclusion (ii)). In view of the high number of accidents with NaOH following oral, dermal, ocular and inhalatory exposure conclusion (iii) is justifiable for foreseeable misuse.

Skin

NaOH is considered to be a strong corrosive agent (concentrations ≥ 2%). During normal use consumers can be exposed to corrosive concentrations. However, sufficient measurements (i.e. classification and labelling) have been taken for these exposure situations. Therefore, conclusion (ii) is justifiable for scenarios in which corrosive concentrations of NaOH are handled.

Dermal exposure to irritating, but non-corrosive, dilutions of NaOH (concentrations < 2%) also occurs. It is assumed that existing controls (i.e. personal protective equipment based on classification and labelling with R38) are applied for these exposure situations. Therefore, it is concluded that NaOH is of no concern during normal use for consumers with regard to skin irritation for scenarios in which non-corrosive concentrations are handled.

Conclusion (ii).

In view of the high number of accidents with NaOH following oral, dermal, ocular and inhalatory exposure conclusion (iii) is justifiable for foreseeable misuse.

Eye

Theoretically, NaOH is of concern for consumers with regard to eye effects because of the corrosive properties of NaOH (concentration of 1.2%) to the eye in an animal study. However, during normal use exposure will occur only incidentally, so conclusion (ii) is justifiable for scenarios in which corrosive concentrations of NaOH are handled.

Exposure to irritating, but non-corrosive, dilutions of NaOH also occurs. It is assumed that during normal use exposure will only occur incidentally. Therefore, it is concluded that NaOH is of no concern for consumers with regard to eye irritation for scenarios in which non-corrosive concentrations are handled.

Conclusion (ii).

In view of the high number of accidents with NaOH following oral, dermal, ocular and inhalatory exposure conclusion (iii) is justifiable for foreseeable misuse.

Respiratory tract

In one scenario (oven cleaners) consumers can be exposed to contents of NaOH that can cause respiratory tract irritation. However, during normal use (short spraying time and closing of the oven door) and because NaOH is unstable in air, exposure concentrations are considered to be negligible.

Conclusion (ii).

In view of the high number of accidents with NaOH following oral, dermal, ocular and inhalatory exposure conclusion (iii) is justifiable for foreseeable misuse.

4.1.3.2.2 Sensitisation

Skin

A study with human volunteers did not indicate skin sensitisation potential of NaOH, which is supported by extensive human experience.

Conclusion (ii).

Respiratory tract

No studies are available on respiratory sensitisation of NaOH. However, there are no reports on respiratory sensitisation, despite the long and widespread use of NaOH. Therefore, it is concluded that there is no concern for this endpoint.

Conclusion (ii).

4.1.3.2.3 Repeated dose toxicity

The systemic availability of NaOH after inhalation and dermal exposure is too low to cause repeated dose toxicity. Therefore, it is concluded that NaOH is of no concern for consumers with regard to repeated dose toxicity.

Conclusion (ii).

4.1.3.2.4 Mutagenicity

Given the results from the *in vitro* and *in vivo* mutagenicity studies, it is concluded that NaOH is of no concern for consumers with regard to mutagenicity.

Conclusion (ii).

4.1.3.2.5 Carcinogenicity

No suitable studies are available to assess the risk on carcinogenic effects. However, it is not expected that NaOH will induce tumors. First of all, NaOH did not induce mutagenicity in *in vitro* and *in vivo* studies. Secondly, systemic carcinogenicity is not expected to occur because NaOH is not expected to be systemically available in the body under normal handling and use conditions. Finally, the anticipated level of protection for dermal and inhalatory exposure is assumed to prevent possible carcinogenic responses that may be due to chronic local irritation. Therefore, it is concluded that NaOH is of no concern for consumers with regard to carcinogenicity.

Conclusion (ii).

4.1.3.2.6 Toxicity for reproduction

NaOH is not expected to be systemically available in the body under normal handling and use conditions and for this reason it can be stated that the substance will not reach the foetus nor reach male and female reproductive organs. Therefore, it is concluded that NaOH is of no concern for consumers with regard to toxicity for reproduction.

Conclusion (ii).

4.1.3.2.7 Summary of risk characterisation for consumers

Following the normal use of corrosive and irritating concentrations of sodium hydroxide it is concluded that the substance is of no risk for consumers if the required protection is used.

Conclusion (ii).

However, the number of accidents that occur with sodium hydroxide is still high, which points out that consumer protection against improper use of sodium hydroxide is insufficient (Conclusion (iii)). Therefore, instructions for use should contain a warning against dangerous mixtures. For reducing the number of accidents in which (young) children are involved, it is advisable to use these products in the absence of children. Information regarding safe disposal of empty containers could also be useful.

4.1.3.3 Humans exposed via the environment

Production and use of sodium hydroxide is normally not expected to increase the pH of the environment. Even after an accidental release the substance will be neutralised finally and therefore the human exposure to sodium hydroxide via the environment is expected to be negligible. Therefore, no direct or systemic exposure is expected from sodium hydroxide.

Conclusion (ii).

4.2 HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

NaOH is neither explosive, flammable nor oxidising and therefore it is concluded that NaOH is of no concern with regard to the physical-chemical hazards.

Conclusion (ii).

5 RESULTS

5.1 INTRODUCTION

5.2 ENVIRONMENT

Conclusions to the risk assessment for the aquatic compartment:

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

Conclusion (ii) applies to:

- **Production**; based on the results from a questionnaire among producers, it is concluded that discharges of NaOH from production to STPs/WWTPs and receiving waters are well controlled in all investigated cases (see Section 3.1.2.1). Taking into account the existing EU Directives for pH control for surface water (see Section 2.4.1) and the data of many Member States on (additional) national regulations to control the pH of waste waters (STP influents) and surface waters it is concluded that STPs and surface waters are sufficiently protected with regard to pH changes.
- **Use**; the results from a questionnaire among users indicate that in most cases the final effluent did not contain NaOH anymore, so it is concluded that discharges of NaOH from the various downstream applications rarely occur. If discharges do occur they are well controlled in all investigated cases (see Section 3.1.2.2.) and are often covered by EU and/or national regulations (see Section 2.4.1).

  Regarding conclusion (ii) for the aquatic compartment it is emphasised that it cannot be excluded that there are (some) sites with NaOH discharges to the aquatic environment, resulting in significant pH changes and effects on biological STPs/WWTPs or receiving surface waters. However, the available data clearly indicate that neutralisation of NaOH containing waste waters and effluents is common practice, either from a legal point of view (legislation for surface waters) or from a practical point of view (protection of the functioning of biological STPs/WWTPs). Regarding surface water, the enforcement of the (EU) legislation is an important issue for the validity of conclusion (ii)**.**

5.3 HUMAN HEALTH

5.3.1 Human health (toxicity)

5.3.1.1 Workers

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

Conclusion (iii) is reached because it cannot be excluded that respiratory tract irritation may occur in the production when bagging NaOH, and when using NaOH in aluminium and textile industry and in the de-inking of waste paper in pulp and paper industry. Risk reducing measures should be taken for these occupational scenarios.

It might be possible that in some workplaces adequate worker protection measures are already being applied.

In relation to all other potential adverse effects and the worker population it is concluded that based on the available information at present there is no concern and no further information/testing on the substance is needed.

5.3.1.2 Consumers

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

Conclusion (iii) applies to the high number of accidents (foreseeable misuse) that occur with sodium hydroxide, which points out that consumer protection against improper use of sodium hydroxide is insufficient. Because sodium hydroxide has local effects the conclusion (iii) is applicable for the endpoint "irritation and corrosivity" for all routes of exposure (oral, dermal, ocular, and inhalatory exposure). To prevent improper use of sodium hydroxide, instructions for use should contain a warning against dangerous mixtures. For reducing the number of accidents in which (young) children are involved, it is advisable to use these products in the absence of children. Information regarding safe disposal of empty containers could also be useful.

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

Conclusion (ii) applies to the normal use of corrosive and irritating concentrations of sodium hydroxide if the required protection is used.

5.3.1.3 Humans exposed via the environment

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

Production and use of sodium hydroxide is normally not expected to increase the pH of the environment. Even after an accidental release the substance will be neutralised finally and therefore the human exposure to sodium hydroxide via the environment is expected to be negligible. Therefore, no direct or systemic exposure via the environment is expected from sodium hydroxide.

Conclusion (ii).

5.3.2 Human health (physico-chemical properties)

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

This conclusion is reached because the risk assessment shows that risks are not expected. Risk reduction measures already being applied are considered sufficient.

6 REFERENCES

Aaron et al. (1989) The Mouse Bone Marrow Micronucleus Test : Evaluation of 21 Drug Candidates Mutation Research **223**, 129–140.

Alabaster JS and Lloyd R (1980) Water Quality criteria for Freshwater Fish. ISBN 0 408 10673 5. Butterworth & Co Ltd. (Publishers), London, United Kingdom

American Conference of Governmental Industrial Hygienists (ACGIH) (2001) Documentation of the Threshold Limit Values for Chemical Substances, 7th Ed.; ACGIH; Cincinnati, OH.

Bartnik FG et al. (1990) Skin organ culture for the study of skin irritancy. Toxicol. In Vitro **4**, 293-301

Bremmer HJ, Prud'homme de Lodder LCH and van Veen MP (2002) Factsheet Cosmetica; Ten behoeve van de schatting van de risico's voor de consument. RIVM report 612810013 (only in Dutch). Bilthoven, The Netherlands.

Bromberg BE et al. (1965) Hydrotherapy of Chemical Burns. Plast. Reconstr. Surg. **35**, 85–95.

Brook et al. (1985) Testing of 3 chemical compounds for aneuploidy induction in the female mouse. Mutation Res. **157**, 215-220.

Bulich AA, Tung, K and Schreibner (1990) The luminescent bacteria toxicity test: its potential as an *in vitro* alternative. Journal of Bioluminescence and Chemoluminescence, **5**, 71-77.

Burton NC and McCleery RE (2000) Health Hazard Evaluation Report HETA 98-0203-2778, United Airlines, Indianapolis, Indiana.

CA (2001) Use of Targeted Risk Assessments in the EU. Discussion paper for the 18th meeting of the CA for the implementation of Council Regulation 793/93. The Netherlands, January 2001.

Caldeira K and Berner R (1999) Seawater pH and atmospheric carbon dioxide. Science, **286**, 2043a.

Casasnovas et al. (1997) A retrospective analysis of ingestion of caustic substances by children. Ten-year statistics in Galicia. Eur. J. Pediatr. **156**, 410-414.

Cello et al. (1980) Liquid caustic ingestion – Spectrum of Injury. Arch. Intern. Med. **140**, 501-504.

Clausen et al. (1994) Admission to Danish hospitals after suspected ingestion of corrosives. Danish Med. Bull. **41**, 234-237.

CMAI (2000) Fifteenth Annual World Petrochemical Conference, March 29 & 30, 2000. Houston, Texas, USA.

Cooper et al. (1979) A critique of the U.S. standard for industrial exposure to sodium hydroxide aerosols. Amer. Ind. Hyg. Ass. J. **40**, 365-371.

De Flora et al. (1984) Genotoxicity activity and potency of 135 compounds in the Ames reversion test in a bacterial DNA-repair test. Mut. Res. **133**, 161-198.

De Groot WA and Van Dijk NRM (2002) Addition of sodium hydroxide to a solution with sodium bicarbonate to a fixed pH. Solvay Pharmaceuticals Int. Doc. No. 8320/47/01, Weesp, The Netherlands.

De Pater AJ and Marquart J (1999) Inhalation exposure to non-volatile compounds during spray painting, TNO Report V98.1340.

De Schamphelaere KAC, Heijerick DG and Janssen CR (2003) Development and Validation of Biotic Ligand Models for Predicting Chronic Zinc Toxicity to Fish, Daphnids and Algae (Final report of ILZRO project ZEH-WA-01). Laboratory of Environmental Toxicology and Aquatic Ecology, Ghent University, Belgium (Sponsor: International Lead and Zinc Research Organization, ILZRO, Research Triangle Park, North Carolina, United States).

Dluhos M, Sklensky B and Vyskocil J (1969) Effect of aerosol inhalation of soda hydroxide on the respiratory tract of rats. Vnitr Lek, **15**, 38-42. [cited in NIOSH, 1975.]

Dykes et al. (1995) A stepwise procedure for evaluating irritant materials in normal volunteer subjects. Hum. Exper. Toxic. **14**, 204-211.

EAPCCT (2003) http://www.eapcct.org (website visited in 2003)

EC (2001) Reference Document on Best Available Techniques in the Chlor-Alkali Manufacturing industry. European Integrated Pollution Prevention and Control Bureau, Sevilla, Spain. Report available via: http://eippcb.jrc.es/

EC (2003) Technical Guidance Document (TGD) on Risk Assessment in support of Commission Directive 93/67/EEC on Risk Assessment for new notified substances and Commission Regulation (EC) 1488/94 on Risk Assessment for existing substances and Directive 98/8/EC of the European Parliament and of the Council concerning the placing of biocidal products on the market. Part I, II, III, IV, Second Edition. European Chemicals Bureau (ECB), Ispra, Italy, 2003.

EC (2005) Technical Guidance Document (TGD) on Risk Assessment in support of Commission Directive 93/67/EEC on Risk Assessment for new notified substances and Commission Regulation (EC) 1488/94 on Risk Assessment for existing substances and Directive 98/8/EC of the European Parliament and of the Council concerning the placing of biocidal products on the market. Human Health Risk Characterisation. Revised Chapter, Final Draft. European Chemicals Bureau (ECB), Ispra, Italy, 2005.

ECB (2003) European Chemicals Bureau, Newsletter No. 4, available via: http://ecb.jrc.it/existing-chemicals/

EOHS (2001) Encyclopedia of Occupational Health and Safety, 4th Edition, Part III, International Labour Office, Geneva.

Euro Chlor (1999) Chlorine Industry Review, 1998-1999, Brochure of 16 pages.

Euro Chlor (2004a) Data submitted according to Annex VIIA of Directive 67/548/EEC.

Euro Chlor (2004b) European chlor-alkali industry, plant & production data 1970-2003.

Euro Chlor (2004c) Risks of NaOH at production sites, Results of questionnaires, 20 september 2004.

Euro Chlor (2005) Risks of NaOH during use. Results of questionnaires sent to major end users, 4 May 2005.

Fodor et al. (1999) Recommendations on dietary salt. Canadian Medical Association Journal, 160, S29-S34.

Fritschi L, De Klerk N, Sim M, Benke G and Musk AW (2001) Respiratory morbidity and exposure to bauxite, alumina and caustic mist in alumina refineries. J. Occup. H. **43**, 231-237.

Gohar HAF et al. (1961) Tolerance of Vector Snails of Bilharziasis and Fascioliasis to Some Chemicals. Proc Egypt Acad. Sci. **16**, 37–48.

Griffiths et al. (1997) Interlaboratory evaluation of a Human patch test for the identification of skin irritation Potential/Hazard. Food and Chem. Toxic. **35**, 255-260.

Gumaste VV et al. (1992) Ingestion of Corrosive Substances by Adults. Am. J. Gastroenterol. **87**, 1–5.

Hansen et al. (1991) Obstructive Lung Injury after Treating Wood with Sodium Hydroxide. J. Soc. Occup. Med. **41**, 45-46.

Heijerick DG, De Schamphelaere KAC and Janssen CR (2003) Application of Biotic Ligand Models for Predicting Zinc Toxicity in European Waters (Final report of ILZRO project ZEH-WA-02). Laboratory of Environmental Toxicology and Aquatic Ecology, Ghent University, Belgium (Sponsor: International Lead and Zinc Research Organization, ILZRO, Research Triangle Park, North Carolina, United States).

Hervin RL and Cohen SR (1973) Health hazard evaluation determination-Report No. 72-97-135. Cincinnati (OH): NIOSH.

Household Products Database (2004) National Institute of Health and U.S. National Library of Medicine. See: http://hpd.nlm.nih.gov/products.htm.

Hugh TB et al. (1991) Gastric Antral Patch Esophagoplasty for Extensive Corrosive Stricture of the Esophagus. World J. Surg. **15**, 299–303.

Jacobs G (1990) Dermal irritation/corrosion study: “pH/reserve”. Evaluation of the publication of Young et al. (1988). Report D/1990/2505/23 of the Institute of Hygiene and Epidemiology, Div. Toxicology, J.Wytsmanstraat 14, B-1050 Brussels, Belgium.

Jacobs GA (1992) OECD Eye Irritation Tests on Sodium hydroxide. J. Amer. Coll. Toxicol. **11**, 725.

Jensen RA (1978) A Simplified Bioassay Using Finfish for Estimating Potential Spill damage. Proc. control of hazardous material spills, Rockvill, MD, 104–108.

Jordan DHM and Lloyd R (1964) The resistance of rainbow trout (Salmo gairdnerii Richardson) and roach (Rutilus rutilus (L.) to alkaline solutions. Int. J. Air. Wat. Pollut. **8**, 405-409.

Juhnke I and Lüdemann D (1978) Ergebnisse der Untersuchung von 200 chemischen Verbindungen auf akute Fischtoxizität mit den Goldorfentest. Z. f. Wasser- und Abwasser-Forschung, **11**, 161-164.

Kavin et al. (1996) Chronic esophagitis evolving to verrucous squamous cell carcinoma: Possible role of exogenous chemical carcinogens. Gastroenterol. **110**, 904-914.

Karlsmark et al. (1988) The effect of sodium hydroxide and hydrochloric acid on pig dermis. A light microscopic study. For. Sci. Int. **39**, 227-233.

Kennedy SM, Enarson DA, Janssen RG, Yeung M Chan (1991). Lung health consequences of reported accidental chlorine gas exposures among pulpmill workers, American review of respiratory disease, Vol. 143, No. 1, pag 74-79.

Keskin E et al. (1991) The Effect of Steroid Treatment on Corrosive Oesophageal Burns in Children. Eur. J. Pediatr. Surg. **1**, 335–338.

Korhonen K et al. (2004) Occupational exposure to chemical agents in the paper industry. Int. Arch. Occup. Environ. H. **77**, 451-460.

Kuckelkorn et al. (1993) Retrospektive Betrachtung von schweren Alkaliverätzungen der Augen. Klin. Monatsbl. Augenheilkd. **203**, 397-402.

Lee et al. (1995) Fatal alkali burns. For. Sci. Int. **72**, 219-227.

McKee JE and Wolf (1963) Water Quality Criteria (2^{th} edition; reprint 1976). California State Water Resources Control Board, United States.

Merne ME, Syrjanen KJ and Syrjanen SM (2001) Systemic and local effects of long-term exposure to alkaline drinking water in rats. Int. J. Exp. Pathol. **82**(4), 213-9.

Moazam F et al. (1987) Caustic Ingestion and Its Sequelae in Children. South Med. J. **80**, 187–190.

Morgan et al. (1987) Prediction of Ocular Irritation by Corneal Pachymetry. Food Chem. Toxicol. **25**, 609-613.

Morita et al. (1989) Effects of pH in the *in vitro* chromosomal aberration test. Mutat. Res. **225**, 55-60.

Murphy et al. (1982) Ocular irritancy responses to various pHs of acids and bases with and without irrigation. Toxicol. **23**, 281-291.

National Institute for Occupational Safety and Health (NIOSH) (1974) Sodium hydroxide. In: The Toxic Substances List 1974 Edition. Publication No. 74-134. Rockville (MD): US Dept. of Health Education and Welfare, Public Health Service, Center for Disease Control, DHEW (NIOSH) 721.

National Institute for Occupational Safety and Health (NIOSH) (1976) Criteria for a recommended Standard – Occupational exposure to sodium hydroxide. DHEW (NIOSH) Pub. No. 76-105; NTIS Pub. No. PB-246-694. National Technical Information Service, Springfield, VA.

National Institute for Occupational Safety and Health (NIOSH) (1987) Guide to industrial respiratory protection OHHS. Publication no 87-116.

Naunyn–Schmiedeberg's (1937) Archiv für experimentelle Pathologie und Pharmakologie (Berlin, Germany), **184**, 587.

Nousiainen P and Sundquist J (1981) Chemical Hazards in Textile Industry, Technical Research Centre of Finland (VTT), Espoo.

NVZ (2004) Dutch Association for Soap Manufacturers. www.isditproductveilig.nl.

OECD (2002) Screening Information Data Set (SIDS) Initial Assessment report for sodium hydroxyde. Organisation for Economic Co-operation and Development, UNEP Publication (Available on internet: http://www.chem.unep.ch/irptc/sids/OECDSIDS/INDEXCHEMIC.htm)

Office of Environmental Health Hazard Assessment (OEHHA) (1999) Determination of acute reference exposure levels for airborne toxicants. Acute toxicity summary for Sodium Hydroxide. OEHHA, California Environmental Protection Agency: Sacramento, CA, pp. C290-5.

Ott MG, Gordon HL and Schneider EJ (1977) Mortality among employees chronically exposed to caustic dust. J. Occup. Med. **19**, 813-816.

OVB (2002). Calendar (2002) Organisation for the Improvement of Inland Fisheries (OVB), Nieuwegein, The Netherlands.

OxyChem (2000) Caustic Soda Handbook See: http://www.oxy.com/oxychem/Products/caustic_soda/literature/caustic%5B1%5D.pdf

Park KB et al. (1995) A study of skin responses to follow-up, rechallenge and combined effects of irritants using non-invasive measurements. J. Dermatol. Sci. **10**, 159-165.

Parker JG (1984) The Effects of Selected Chemicals and Water Quality on the Marine Polychaete. Wat. Res. **18**, 865–868.

Patty FA (1949) Sodium hydroxide. In Industrial Hygiene and Toxicology, Vol. II, pp.560-561. F.A. Patty, Ed. Interscience, New York.

Perkins et al. (1996) Development of an *in Vitro* Method for Skin Corrosion Testing. Fund. Appl. Toxicol. **31**, 9-18.

Portugal (2003) Proposal Targeted Risk Assessment on NAOH RA (EC Regulation 793/93) Portugal, November 2003.

Prud'homme LCH, Bremmer HJ and van Engelen JGM (draft 2004). Cleaning Products Fact Sheet. RIVM report, Bilthoven, Netherlands.

Queensland Alumina Limited (2004) www.qal.com.au , 15-09-04.

Rand GM (Ed.) (1995) Fundamentals of Aquatic Toxicology: Effects, Environmental Fate and Risk Assessment (Second Edition). ISBN 1-56032-090-7 (case), ISBN 1-56032-091-5 (paper), Taylor & Frances Ltd (Publishers), Washington D.C., United States.

Rubin AE et al. (1992) Obstructive Airway Disease Associated With Occupational Sodium Hydroxide Inhalation. Brit. J. Ind. Med. **49**, 213–214.

Rustamova SA (1977) The Chronic Effect of Alkali on the Growth, Development and Fecundity of the Guppy. Gidrobiol Zh, **13**, 83-85.

Saini et al. (1993) Ocular chemical burns – clinical and demographic profile. Burns, **19**, 67-69.

Schober PH et al. (1989) Ingestion von ätzenden Substanzen im Kindesalter. Wiener Klin Wschr, **101**, 318–322.

Seidenari et al. (1995) Sodium Hydroxide-induced Irritant Dermatitis as Assessed by Computerized Elaboration of 20 Mhz B-scan images and by TEWL Measurement: A Method for Investigating Skin Barrier Function. Act Derm Venereol, **75**, 97-101.

Silverman, J and Pennisi, S (1987) Evaluation of *Tetrahymena thermophila* as an *in vitro* alternative to ocular irritation studies in rabbits. J. Toxicol.-Cut. Ocular Toxicol. **6** (1), 33-42.

Srikrishna V et al. (1991) The Effects of Sodium Hydroxide and Hydrochloric Acid on Isolated Perfused Skin. In Vitro Toxicol. 4, 207–215.

Stangenberg M (1975) The Influence od the Chemical Composition of Water on the Pike Perch Fry From the Lake Gopio. Limnologica. **9**, 421–426.

Stumm W and JJ Morgan (1981) Aquatic Chemistry. John Wiley & Sons, New York.

The Netherlands (2004) European Union Risk Assessment Report Zn Metal (draft of July 2004).

UNEP (1995) Water quality of world river basins. UNEP Environment Library No. 14, Nairobi, Kenya.

US EPA (1988) Estimation of the half-live of sodium hydroxide aerosol in the atmosphere (Guinnup, D. and Scheffe, R.). United States Environmental Protection Agency, Office of air quality planing and standards, Research Triangle Park, North Carolina, United States.

US EPA (1989) Assessment of sodium hydroxide as a potentially toxic air pollutant. Federal Register, 54, No. 9, January 1989. United States Environmental Protection Agency, United States.

Van Kolfschoten et al. (1983) Protection by Paracetamol against Various Gastric Irritants in the Rat. Toxicol. Appl. Pharmacol. **69**, 37-42.

Vilogi et al. (1985) Oven-cleaner Pads: New risk for corrosive injury. Am. J. Emerg. Med. **3**, 412-414.

Vollebregt L and P van Broekhuizen (1994) Tussen wasmand en afdruiprek. Amsterdam: Chemiewinkel UvA.

VROM (2001) Environmental Quality Standards in the Netherlands (1999) – A review of environmental quality standards and their policy framework in the Netherlands. Ministry of Housing, Spatial Planning and the Environment (VROM), Directorate-General for Environmental Protection. Kluwer (Publishers), Alphen aan den Rijn, The Netherlands.

Vyskocil J, Tuma J and Dluhos M (1966) Effect of aerosol inhalation of sodium hydroxide on the elimination of quartz dust from the lung of rats. Scr. Med. Fac. Med. Univ. Brun. Purkynianae **39**, 25-29. [cited in NIOSH, 1975.]

Wallen IE et al. (1957) Toxicity to *Gambusia affinis* of Certain Pure Chemicals in Turbid Waters. Sewage Ind. Wastes **29**, 695–711.

Warne MSJ et al. (1999) Toxicity of Laundry Detergent Components to a Freshwater Cladoceran and their Contribution to Detergent Toxicity. Ecotoxicol. Environ. Safety **44**, 196-206.

Wijburg FA et al. (1985) Nasogastric Intubation as Sole Treatment of Caustic Esophageal Lesions. Ann. Otol. Rhinol Laryngol **94**, 337–341

Windholz M (1983) (Ed.) The Merck Index – An encyclopedia of chemicals, drugs, and biologicals (10th Edition). ISBN 911910-27-1. Merck & Co., Inc. (Publishers), Rahway, N.J., United States.

Yano K et al. (1993) Experimental study on alkaline skin injuries – periodic changes in subcutaneous tissue pH and the effects exerted by washing. Burns **19**, 320-323.

Yarzhombek et al. (1991) Toxicity of Substances in Relation to Form of Exposure.

Voprosy Ikhtiologii **31**, 496-502.

Yasser et al. (1998) Lye-induced esophagitis course and follow up of 29 patients. Gastroenterol. **114**, A273.

York, M et al. (1995) Skin irritation testing in man for hazard assessment – evaluation of four patch systems. Hum. Exper. Toxicol. **14**, 729-734.

York, M et al. (1996) Evaluation of a human patch test for the identification and classification of skin irritation potential. Contact Dermatitis **34**, 204-212.

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
bw	body weight / *Bw*, *b.w.*
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
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
DT50	Degradation half-life or period required for 50 percent dissipation / degradation
DT90	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)
EASE	Estimation and Assessment of Substance Exposure Physico-chemical properties [Model]
EbC50	Effect Concentration measured as 50% reduction in biomass growth in algae tests
EC	European Communities

EC10	Effect Concentration measured as 10% effect
EC50	median Effect Concentration
ECB	European Chemicals Bureau
ECETOC	European Centre for Ecotoxicology and Toxicology of Chemicals
ECVAM	European Centre for the Validation of Alternative Methods
EDC	Endocrine Disrupting Chemical
EEC	European Economic Communities
EINECS	European Inventory of Existing Commercial Chemical Substances
ELINCS	European List of New Chemical Substances
EN	European Norm
EPA	Environmental Protection Agency (USA)
ErC50	Effect Concentration measured as 50% reduction in growth rate in algae tests
ESD	Emission Scenario Document
EU	European Union
EUSES	European Union System for the Evaluation of Substances [software tool in support of the Technical Guidance Document on risk assessment]
F(+)	(Highly) flammable (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
FAO	Food and Agriculture Organisation of the United Nations
FELS	Fish Early Life Stage
GLP	Good Laboratory Practice
HEDSET	EC/OECD Harmonised Electronic Data Set (for data collection of existing substances)
HELCOM	Helsinki Commission -Baltic Marine Environment Protection Commission
HPLC	High Pressure Liquid Chromatography
HPVC	High Production Volume Chemical (> 1000 t/a)
IARC	International Agency for Research on Cancer
IC	Industrial Category
IC50	median Immobilisation Concentration or median Inhibitory Concentration
ILO	International Labour Organisation
IPCS	International Programme on Chemical Safety
ISO	International Organisation for Standardisation
IUCLID	International Uniform Chemical Information Database (existing substances)
IUPAC	International Union for Pure and Applied Chemistry
JEFCA	Joint FAO/WHO Expert Committee on Food Additives
JMPR	Joint FAO/WHO Meeting on Pesticide Residues
Koc	organic carbon normalised distribution coefficient
Kow	octanol/water partition coefficient
Kp	solids-water partition coefficient

L(E)C50	median Lethal (Effect) Concentration
LAEL	Lowest Adverse Effect Level
LC50	median Lethal Concentration
LD50	median Lethal Dose
LEV	Local Exhaust Ventilation
LLNA	Local Lymph Node Assay
LOAEL	Lowest Observed Adverse Effect Level
LOEC	Lowest Observed Effect Concentration
LOED	Lowest Observed Effect Dose
LOEL	Lowest Observed Effect Level
MAC	Maximum Allowable Concentration
MATC	Maximum Acceptable Toxic Concentration
MC	Main Category
MITI	Ministry of International Trade and Industry, Japan
MOE	Margin of Exposure
MOS	Margin of Safety
MW	Molecular Weight
N	Dangerous for the environment (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC
NAEL	No Adverse Effect Level
NOAEL	No Observed Adverse Effect Level
NOEL	No Observed Effect Level
NOEC	No Observed Effect Concentration
NTP	National Toxicology Program (USA)
O	Oxidizing (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
OECD	Organisation for Economic Cooperation and Development
OEL	Occupational Exposure Limit
OJ	Official Journal
OSPAR	Oslo and Paris Convention for the protection of the marine environment of the Northeast Atlantic
P	Persistent
PBT	Persistent, Bioaccumulative and Toxic
PBPK	Physiologically Based PharmacoKinetic modelling
PBTK	Physiologically Based ToxicoKinetic modelling
PEC	Predicted Environmental Concentration
pH	logarithm (to the base 10) (of the hydrogen ion concentration $\{H^+\}$
pKa	logarithm (to the base 10) of the acid dissociation constant

pKb	logarithm (to the base 10) of the base dissociation constant
PNEC	Predicted No Effect Concentration
POP	Persistent Organic Pollutant
PPE	Personal Protective Equipment
QSAR	(Quantitative) Structure-Activity Relationship
R phrases	Risk phrases according to Annex III of Directive 67/548/EEC
RAR	Risk Assessment Report
RC	Risk Characterisation
RfC	Reference Concentration
RfD	Reference Dose
RNA	RiboNucleic Acid
RPE	Respiratory Protective Equipment
RWC	Reasonable Worst Case
S phrases	Safety phrases according to Annex III of Directive 67/548/EEC
SAR	Structure-Activity Relationships
SBR	Standardised birth ratio
SCE	Sister Chromatic Exchange
SDS	Safety Data Sheet
SETAC	Society of Environmental Toxicology And Chemistry
SNIF	Summary Notification Interchange Format (new substances)
SSD	Species Sensitivity Distribution
STP	Sewage Treatment Plant
T(+)	(Very) Toxic (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
TDI	Tolerable Daily Intake
TG	Test Guideline
TGD	Technical Guidance Document
TNsG	Technical Notes for Guidance (for Biocides)
TNO	The Netherlands Organisation for Applied Scientific Research
UC	Use Category
UDS	Unscheduled DNA Synthesis
UN	United Nations
UNEP	United Nations Environment Programme
US EPA	Environmental Protection Agency, USA
UV	Ultraviolet Region of Spectrum
UVCB	Unknown or Variable composition, Complex reaction products of Biological material
vB	very Bioaccumulative
vP	very Persistent

vPvB	very Persistent and very Bioaccumulative
v/v	volume per volume ratio
w/w	weight per weight ratio
WHO	World Health Organization
WWTP	Waste Water Treatment Plant
Xn	Harmful (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
Xi	Irritant (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)

Appendix A Effect of pH on chemical speciation and toxicity of other substances in water

Besides a 'direct' effect. i.e. pH increase, NaOH can also have an 'indirect' effect, as the pH change can affect the chemical speciation and thus the toxicity of other substances in water. It is emphasised that these 'indirect' effects are beyond the scope of this risk assessment report for NaOH, but two examples are given below, for illustration.

Metals

If the pH increases, the solubility of metals may decrease, resulting in a different chemical speciation, including lower free metal ion concentrations in the water and at the biotic ligand of organisms. Biotic ligands are uptake (transport) sites and toxic action sites. This may result in a lower bioavailability and thus lower toxicity, as has been found for example for the toxicity of zinc to fish and daphnids, in the pH range of 5.5-8.0. On the other hand, a pH increase can also result in a higher toxicity, as has been found for the toxicity of zinc to algae, in the pH range of 5.5-8.0 (De Schamphelaere, Heijerick and Janssen, 2003). For aluminium, the aquatic toxicity data indicate that the toxicity is lowest at around neutral pH and that both under acidic and alkaline conditions the toxicity increases.

Ammonia (NH_3)/ammonium (NH_4OH; NH_4^+)

The chemical speciation of ammonia/ammonium in water is determined by the following equilibrium reactions:

$$NH_3 + H_2O \leftrightarrow NH_4OH \leftrightarrow NH_4^+ + OH^-$$

If the pH increases, the concentration of un-ionised NH_3 increases and un-ionised NH_3 is much more toxic to fish and most likely also to other aquatic organisms than ionised NH_4^+, as un-ionised NH_3 more easily will pass biological membranes than ionised NH_4^+. The lowest lethal NH_3 concentration for fish (salmonids) is 0.2 mg/l, but at prolonged exposure of fish to NH_3 concentrations of $\geq$ 0.025 mg/l other adverse effects have been found (NOEC: 0.025 mg/l). Concentrations of total ammonia (NH_3 and NH_4^+) which contains this amount of NH_3 range from 0.12 mg/l (at pH 8.5 and 30 ^{0}C) to 19.6 mg/l (at pH 7.0 and 5 ^{0}C). The NH_3 concentration also increases with increasing temperature (Alabaster and Lloyd, 1980). In the Netherlands, the Maximum Permissible Concentration (MPC) for unionised NH_3 is 0.02 mg/l (VROM, 2001).

The above data for two metals (zinc and aluminium) and ammonia/ammonium, which are only some of the naturally occurring substances in water, indicate that the indirect effect of NaOH on aquatic organisms and especially on ecosystems is very difficult to predict.

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sodium hydroxide, Volume 73

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The report provides the targeted risk assessment of the substance sodium hydroxide (NaOH) It has been prepared by Portugal 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. In this context 'targeted' means that not all endpoints, as defined in the Technical Guidance Documents (TGD), are addressed thoroughly in this risk assessment. In a targeted risk assessment one deviates therefore from the standard comprehensive risk assessment that covers all possible exposure routes of the chemical and all protection goals.

The evaluation considers the emissions and the resulting exposure to the environment and the human populations in all life cycle steps. However, considering the likely emission pathways, the environmental exposure assessment was targeted at the aquatic emissions, and consequently, the environmental risk characterisation was targeted at the aquatic compartment. For human health, the scenarios for occupational exposure, consumer exposure and humans exposed via the environment have been examined and the possible risks for local effects at the site of contact with sodium hydroxide (NaOH) have been identified.

Part I - Environment

The environmental risk assessment is targeted solely on the aquatic environment, as the emissions of NaOH mainly apply to waste water, and to effects on organisms/ecosystems due to possible local pH changes. Based on the results from a questionnaire among producers and users of sodium hydroxide, it is concluded that discharges of NaOH from production are well controlled, and that discharges from downstream applications rarely occur. Regarding surface water, the enforcement of the (EU) legislation is an important assumption for the conclusion of no concern for the aquatic environment.

Part II – Human Health

The human health risk assessment is targeted on the risks for local effects at the site of contact with NaOH, both for workers and consumers. For workers, there is concern that respiratory tract irritation may occur in the production when bagging NaOH, when using NaOH in aluminium and textile industry, and in the de-inking of waste paper in pulp and paper industry. For consumers, there is no concern for the normal use of NaOH, where it is assumed that the required protection is used. However, there is a high number of accidents (foreseeable misuse) that occur with NaOH, which points out that consumer protection against improper use of NaOH is insufficient. Because NaOH has local effects, there is concern for irritation and corrosivity for all routes of exposure (oral, dermal, ocular, and inhalatory exposure) after foreseeable misuse of NaOH by consumers. There is no concern for humans exposed via the environment or for the physico-chemical properties.

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

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