VERBAND DER CHEMISCHEN INDUSTRIE e.V.

REACH Praxisführer zur Expositionsbewertung und zur Kommunikation in den Lieferketten
Beispiele zu Teil II: Expositionsszenarien und Kommunikation in den Lieferketten

Beispiel 6: Stoffsicherheitsbericht Natriumhydroxid

Dieses Beispiel veranschaulicht die Stoffsicherheitsbeurteilung und die Gestaltung eines Expositionsszenarios für eine Grundchemikalie mit weiten Anwendungsgebieten.

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Wichtiger Hinweis für den Leser:

Dieses Beispiel eines Stoffsicherheitsberichtes ist von der Verfasserin in einer Arbeitsgruppe des VCI erstellt worden als Teil des gemeinsamen Projektes von Cefic/VCI zur Entwicklung von Instrumenten und Leitfäden – für die Stoffsicherheitsbeurteilung, den Stoffsicherheitsbericht und für Expositionsszenarien.

Das vorliegende Dokument gehört zum Teil II des „REACH Praxisführers zur Expositionsbewertung und zur Kommunikation in den Lieferketten“. Der Praxisführer besteht aus mehreren Teilen. Eine Übersicht finden Sie im Vorwort zu Teil I.

Eine Beschreibung der Inhalte und des Praxisführers steht auf der folgenden Internetseite zur Verfügung:

VCI: http://www.vci.de/default~cmd~shd~docnr~125022~lastDokNr~102474.htm

Alle zugehörigen Materialien (inklusive der Projektbeispiele zu Teil II und III) können von dieser Seite heruntergeladen werden. Sie finden hier zusätzlich Hinweise zu verwandten Themen und neuen Entwicklungen.

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CHEMICAL SAFETY REPORT

Substance Name: **Sodium hydroxide**

EINECS Number: **215-185-5**

CAS Number: **1310-73-2**

Registrant's identity: **BASF SE**

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PART A

1 SUMMARY OF RISK MANAGEMENT MEASURES

Manufacture and own use: Summary of conditions of use needed to ensure control of risk	
	Use of Sodium hydroxide in all industrial, professional and consumer uses (except aerosol use), e.g. use as intermediate in glass-, paper-, aluminium- and detergent production, use for the production of many chemicals, use as process aid (neutralisation, cleaning agent) **The following RMM have to be seen as examples. Other RMM can be applied also when the same level of safety could be achieved.** **<u>Risk management measures related to workers (industrial and professional)</u>** General instructions: Avoid skin contact – Do not touch (in case of skin contact, rinse intensely with water) **a) Personal measures:** During operation with NaOH use always protective gloves and protection goggles Do not spray NaOH-solutions Do not inhale in vapours/aerosols during heating Use disposable gloves for short term use Use gloves with 8 h-break-through guarantee for long term use **b) Technical Measures** Closed systems e.g. through pipelines Use grippers and grabbers **c) Product related Measures** Dilution < 1% before use as cleaning agent (e.g.) Hand over only in tank vehicles or in barrels **d) Organisational Measures** Training about hazard assessment before handling of the substance Access to production/formulation only for qualified personnel Delivery only to specialised trade

	Risk management measures related to consumers **a) Personal Measures** Instructions given on packaging and in the instructions for use (analogous to those for the worker) **b) Product related Measures** Dilution below 1 % Childproof packaging Hand-over only with integrated volumetric metering Hand-over only in small amounts Hand-over only in highly viscous preparations Insert of protection gloves and goggles in the packaging **c) Organisational Measures** Hand-over only to persons over 18 after information about hazard **Risk management measures related to environment** **a) Instructions:** e.g. Do not discharged undiluted into waste water **b) Technical Measures:** Neutralisation required to a pH-value of… Dilution to a pH-value of…….. **Waste related measures** Discharges of NaOH from production to sewage treatment plants (STP)/waste water treatment plants and receiving waters are well controlled. Only bigger amounts of NaOH waste have to be neutralised or diluted. Taking into account the existing EU Directives for pH-control for surface water and national regulations to control the pH of waster waters and surface waters is is concluded that STPs and surface waters are sufficiently protected with regard to pH changes.
Downstream use: Summary of conditions of use to ensure control of risk	

2 DECLARATION THAT RISK MANAGEMENT MEASURES ARE IMPLEMENTED

3 DECLARATION THAT RISK MANAGEMENT MEASURES ARE COMMUNICATED

PART B

The hazard data are referred to the European Union Risk Assessment Report to Sodium hydroxide. It has to be checked which data are necessary for the hazard assessment of Sodium hydroxide (100%).

1 IDENTITY OF THE SUBSTANCE AND PHYSICAL AND CHEMICAL PROPERTIES

1.1 Name and other identifiers of the substance

Table 1: Substance identity

EC number:	215-185-5
EC name:	Sodium hydroxide
CAS number (EC inventory):	1310-73-2
CAS number:	1310-73-2
CAS name:	Sodium hydroxide
IUPAC name:	Sodium hydroxide
Annex I index number	011-002-00-6
Molecular formula:	NaOH
Molecular weight range:	40

Structural formula: NaOH

Remarks: -

1.2 Composition of the substance

Table 2: Constituents

Constituent	Typical concentration	Concentration range	Remarks
Sodium hydroxide 1310-73-2	100 % (w/w) (solid)	20-40 % (w/w) liquid	

Table 3: Impurities

Impurities	Typical concentration	Concentration range	Remarks

Sodium chloride 7647-14-599 Sodium carbonate 497-19-8 Sulfate 203-806-2 Others	< 2 % (w/w) <= 1.0 % (w/w) <= 0.2 % (w/w) < 0.1 % (w/w)		

Table 4: Additives

Constituent	Function	Typical concentration	Concentration range	Remarks

1.3 Physico-chemical properties

Table 5: Summary of physico- chemical properties

Property	Value	Remarks
Physical state at 20°C and 101.3 kPa	Solid/liquid	
Melting/freezing 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, 80 %) 160 °C at 1013 hPa (solution, 60%) 128 °C at 1013 hPa (solution,40%) 118 °C at 1013 hPa (solution, 20 %)	
Relative density	2.13 g/cm3 at 20 °C (solid 100 %) 1.43 g/cm3 at 20 °C (solution, 40 %) 1.22 g/cm3 at 20 °C (solution, 20 %)	
Vapour pressure	$<10^{-5}$ hPa at 25 °C (calculated)	
Surface tension	based on chemical structure, no surface activity is predicted	
Water solubility	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	
Partition coefficient n-octanol/water (log value)	Not applicable	
Flash point	Not applicable	
Flammability	Not applicable.	
Explosive properties	Not applicable	
Self-ignition temperature	Not applicable	
Oxidising properties	Not applicable	
Granulometry	Substance is marketed or used in a non-solid form or granular form	
Stability in organic solvents and identity of relevant degradation products		
Dissociation constant	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.	
Viscosity		

Remarks: -

Testing proposal: -

2 MANUFACTURE AND USES

2.1 Manufacture

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).
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 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).

2.1.2 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).

2.1.3 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

2.2 Identified uses

Table 6: Identified uses described by process category (PROC) and sector of use (SU)

	Sectors of Use (SU)					
	All SU	***SU 6 -7***				
Process category (PROC)						
ALL PROCs						
ALL PROCs						

Table 7: Identified specific or individual uses described by preparation category (PC) processed into an article category (AC)

	Preparation category (PC)					
Article category (AC)						
AC 11						
AC 13						

Table 8: Identified uses described by sectors of uses and preparation category

	Preparation category (PC)					
Sector of use (SU)						
SU 6						
SU 7						

2.3 Uses advised against

Application of aerosols.

3 CLASSIFICATION AND LABELLING

3.1 Classification in Annex I of Directive 67/548/EEC

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

Concentration	Classification
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).

3.2 Self classification(s)

Table 9: Classification according to Directive 67/548/EEC criteria

Endpoints	Classification	Reason for no classification	Justification for (non) classification can be found in section
Explosiveness		Classification criteria not met	6.1
Oxidising properties		Classification criteria not met	6.3
Flammability		Classification criteria not met	6.2
Thermal stability		Classification criteria not met	
Acute toxicity		Classification criteria not met	5.2.3
Acute toxicity- irreversible damage after single exposure		Classification criteria not met	5.2.3
Repeated dose toxicity		Classification criteria not met	5.6.3
Irritation / Corrosion	C; R35 –Corrosive, causes severe burns		5.3.4 and 5.4.3
Sensitisation		Classification criteria not met	5.5.3
Carcinogenicity		Classification criteria not met	5.8.3
Mutagenicity - Genetic Toxicity		Classification criteria not met	5.7.3
Toxicity to reproduction- fertility		Classification criteria not met	5.9.3
Toxicity to reproduction- development		Classification criteria not met	5.9.3
Toxicity to reproduction – breastfed babies		Classification criteria not met	5.9.3
Environment		Classification criteria not met	7.6

4 ENVIRONMENTAL FATE PROPERTIES

4.1 Degradation

4.1.1 Abiotic degradation

When hydroxides are dissolved in water, they dissociate to produce free hydroxide ions (thus raising the pH of the solution) and the counter metal cations: NaOH <==> Na+ + OH
The hydroxide ion may then react with free H+ or any acidic species that may be present, forming water: OH- + H= <==> H_2O, K = 10E14 (25°C)
Solubility of NaOH in solution is 109 g/100 g H_2O; this solubility is affected by pH, temperature and the presence of other species in solution:
--> increased pH causes decreased solubility because a higher OH concentration reduces the amount of solid hydroxide that can dissociate into free metal ions and OH - ions.
--> with increased temperature, the alkali metal hydroxide become more soluble

4.1.1.1 Hydrolysis

In water, NaOH is present as the sodium ion (Na^+) and hydroxyl ion (OH^-), as solid NaOH rapidly dissolves and subsequently dissociates in water
The dissolution of alkali hydroxides in water is a strongly exothermic process; their solutions generate heat when diluted.
- Dilution of sodium hydroxide solutions of 40% or greater concentration can generate enough heat to raise the temperature above boiling point, causing dangerous eruptions of the solution.

Data waiving *(if applicable)*

4.1.1.2 Phototransformation/photolysis

4.1.1.2.1 Phototransformation in air

Table 10: Overview of studies on phototransformation in air

Method	Results	Remarks	Reference

Data waiving *(if applicable)*

Not applicable.

Testing proposal

Not applicable.

Discussion

Not applicable.

4.1.1.2.2 Phototransformation in water

The studies on phototransformation in water are summarised in the following table:

Table 11: Overview of studies on phototransformation in water

Method	Results	Remarks	Reference
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Data waiving *(if applicable)*

Reason: study scientifically unjustified

Justification: It is not considered relevant. In water, NaOH is present as the sodium ion (Na^+) and hydroxyl ion (OH^-), as solid NaOH rapidly dissolves and subsequently dissociates in water

Testing proposal

Not applicable.

Discussion

Not applicable

4.1.1.2.3 Phototransformation in soil

The studies on phototransformation in soil are summarised in the following table:

Table 12: Overview of studies on phototransformation in soil

Method	Results	Remarks	Reference
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Data waiving *(if applicable)*

Reason: study scientifically unjustified

Justification: It is not considered relevant. In water, NaOH is present as the sodium ion (Na^+) and hydroxyl ion (OH^-), as solid NaOH rapidly dissolves and subsequently dissociates in water

Testing proposal

Not applicable.

Discussion

The following information is taken into account for any hazard / risk / persistency assessment:

Not applicable

4.1.2 Biodegradation

4.1.2.1 Biodegradation in water

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-}):

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

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 10th–percentile, mean and 90th-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 < 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):
Log (alkalinity in eq/l) = - 0.2877 + 0.8038 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^- (10th 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^- (90th 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

4.1.2.1.1 Estimated data:

The estimated data for biodegradation in water are summarised in the following table:

Table 13: Estimated data for biodegradation in water

Estimation method	Results	Remarks	Reference
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4.1.2.1.2 Screening tests

The test results are summarised in the following table:

Table 14: Screening tests for biodegradation in water

Method	Results	Remarks	Reference

Data waiving *(if applicable)*

Not applicable

4.1.2.1.3 Simulation tests

Table 15: Simulation tests for biodegradation in water

Method	Results	Remarks	Reference
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Data waiving *(if applicable)*

Not applicable

4.1.2.1.4 Summary and discussion of biodegradation in water

Discussion (simulation testing)

Not applicable

4.1.2.2 Biodegradation in sediments

The test results are summarised in the following table

Table 16: Overview of simulation tests for biodegradation in sediments

Method	Results	Remarks	Reference
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Data waiving *(if applicable)*

Not applicable

Discussion

Not applicable

4.1.2.3 Biodegradation in soil

The test results are summarised in the following table:

Table 17: Overview of studies on biodegradation in soil

Method	Results	Remarks	Reference
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Data waiving *(if applicable)*

The terrestrial compartment 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.

Discussion

Not applicable.

4.1.2.4 Summary and discussion on biodegradation

Testing proposal

Not applicable.

4.1.3 Summary and discussion on degradation

Abiotic degradation

Strong alkaline substance that dissociates fully. The concentration of OH- (pH) is in general regulated by the equilibria between CO2, HCO3- and CO32-. In general the buffer capacity depends on the concentration of these substances.

Biotic degradation

Degradation rate in water	Not applicable
Degradation rate in sediment	Not applicable
Degradation rate in soil	Not applicable
Degradation rate in air	Not applicable

4.2 Environmental distribution

4.2.1 Adsorption/desorption

The studies on adsorption/desorption are summarised in the following table:

Table 18: Overview of studies on adsorption/desorption

Method	Results	Remarks	Reference
			BASF SE 2003

Data waiving *(if applicable)*

The high water solubility and low vapour pressure indicate that NaOH will be found predominantly in water. In soil, mobility depends directly on the importance of the liquid phase of the soil and the possibility to form metal hydroxo-complexes with metal solid species. The 73% aqueous solution of NaOH at ambient temperatures is a highly viscous, gelatinous material and without additional dilution (precipitation), it is not expected to infiltrate soil to any significant extent. The 50% aqueous solution of NaOH us liquid and is expected to infiltrate soil to a measurable degree. As the dilution of NaOH increases, its speed of movement through soil increases. During movement

through soil, some ion exchange will occur. Also, some of the hydroxide may remain in the aqueous phase and will move downward through soil in the direction of groundwater flow.

Testing proposal

Not applicable.

Discussion

4.2.2 Volatilisation

The studies on volatilisation are summarised in the following table:

Table 19: Overview of studies on volatilisation

Method	Results	Remarks	Reference

Data waiving *(if applicable)*

The air compartment is considered not relevant for NaOH because sodium hydroxide has low vapour pressure (< 10^{-5} hPa at 25 °C) and is totally miscible in water. An evaporeation into atmosphere is highly unlikely.
In case the substance is 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).

Testing proposal

Not applicable.

Discussion

Not applicable

4.2.3 Distribution modelling

The data from distribution modelling studies are summarised in the following table:

Table 20: Overview of distribution modelling studies

Method	Results	Remarks	Reference

Data waiving *(if applicable)*

Fugacity calculation is not applicable for ionised substances. Based on its phyiochemical properties sodium hydroxide will be mainly distributed into the compartment water, where it exists in ionized form

Testing proposal

Not applicable.

4.3 Bioaccumulation

4.3.1 Aquatic bioaccumulation

The studies on aquatic bioaccumulation are summarised in the following table:

Table 21: Overview of studies on aquatic bioaccumulation

Method	Results	Remarks	Reference

Data waiving *(if applicable)*

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.

4.3.2 Terrestrial bioaccumulation

The results of terrestrial bioaccumulation studies are summarised in the following table:

Table 22: Overview of studies on terrestrial bioaccumulation

Method	Results	Remarks	Reference

Data waiving *(if applicable)*

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.

4.3.3 Summary and discussion of bioaccumulation

Aquatic bioaccumulation

Not applicable

Terrestrial bioaccumulation

Not applicable

Testing proposal

Not applicable.

4.4 Secondary poisoning

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

5 HUMAN HEALTH HAZARD 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.

5.1 Toxicokinetics (absorption, metabolism, distribution and elimination)

5.1.1 Non-human information

5.1.2 Human information

5.1.3 Summary and discussion on toxicokinetics

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.

5.2 Acute toxicity

5.2.1 Non-human information

5.2.1.1 Acute toxicity: 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.

5.2.1.2 Acute toxicity: inhalation

No animal data are available on the acute inhalation toxicity.

5.2.1.3 Acute toxicity: 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.

5.2.1.4 Acute toxicity: other routes

5.2.2 Human information

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.

5.2.3 Summary and discussion of acute toxicity

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

5.3 Irritation

5.3.1 Skin

5.3.1.1 Non-human information

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/cm2 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 preexperimental
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.

5.3.1.2 Human information

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.
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.

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)

5.3.2 Eye

5.3.2.1 Non-human information

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.
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.

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)

5.3.2.2 Human information

Not available

5.3.3 Respiratory tract

5.3.3.1 Non-human information

Not available

5.3.3.2 Human information

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 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/m3), medium (0.05-1.0 mg/m3) or high (> 1.0 mg/m3). 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/m3) were lower than the recommended ceiling level (TLV-value) of 2 mg/m3. 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/m3 from the study of Fritschi et al. (2001) is considered a NOAEL for local effects to the respiratory tract.

5.3.4 Summary and discussion of irritation

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/m3 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%.

5.4 Corrosivity

Covered by 5.3 Irritation, see information above

5.4.1 Non-human information

Covered by 5.3 Irritation, see information above

5.4.2 Human information

Covered by 5.3 Irritation, see information above

5.4.3 Summary and discussion of corrosion

Sodium hydroxide is a corrosive.substance.

5.5 Sensitisation

5.5.1 Skin

5.5.1.1 Non-human information

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.

5.5.1.2 Human information

No experimental study is available

5.5.2 Respiratory system

5.5.2.1 Non-human information

The results of experimental studies on respiratory sensitisation are summarised in the following table:

Table 23: Summary of experimental studies on respiratory sensitisation

Method	Results	Remarks	Reference

The results of estimated data on respiratory sensitisation are summarised in the following table

Table 24: Summary of estimated data ((Q)SAR) on respiratory sensitisation

Method	Results	Remarks	Reference

Data waiving *(if applicable)*

5.5.2.2 Human information

The exposure-related observations in humans are summarised in the following table:

Table 25: Summary of exposure-related observations in humans

Subjects / Study type	Results	Remarks	Reference

5.5.3 Summary and discussion of sensitisation

Skin 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.

Respiratory sensitisation

Justification for classification or non classification

5.6 Repeated dose toxicity

5.6.1 Non-human information

5.6.1.1 Repeated dose toxicity: 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.

5.6.1.2 Repeated dose toxicity: 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.

5.6.1.3 Repeated dose toxicity: dermal

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

5.6.1.4 Repeated dose toxicity: other routes

5.6.2 Human information

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.

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.

5.6.3 Summary and discussion 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

5.7 Mutagenicity

5.7.1 Non-human information

5.7.1.1 In vitro data

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.

5.7.1.2 In vivo data

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.

5.7.2 Human information

5.7.3 Summary and discussion 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.

5.8 Carcinogenicity

5.8.1 Non-human data

5.8.1.1 Carcinogenicity: oral

5.8.1.2 Carcinogenicity: inhalation

5.8.1.3 Carcinogenicity: dermal

5.8.2 Human data

5.8.3 Summary and discussion of 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.

5.9 Toxicity for reproduction

5.9.1 Effects on fertility

5.9.1.1 Non-human data

5.9.1.2 Human data

5.9.2 Developmental toxicity

5.9.2.1 Non-human data

5.9.2.2 Human data

5.9.3 Summary and discussion of reproductive toxicity

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.

5.10 Other effects

5.10.1 Non-human data

5.10.2 Human data

5.10.3 Summary and discussion

5.11 Derivation of DNEL(s) /DMELs

Workers

Study Type	Point of departure	AF	DNEL

5.11.1 Overview of typical dose descriptors for all endpoints

Table 26: Available dose-descriptor(s) per endpoint for a certain substance as a result of its hazard assessment.

Endpoint		**Quantitative dose descriptor[1] (appropriate unit) or qualitative assessment**		**Associated relevant effect[2]**	**Remarks on study[3]**
		Local[4]	Systemic[5]		
Acute toxicity[6]	oral				
	dermal				
	inhalation				
Irritation/Corrosivity	skin		NA[7]		
	eye		NA		
	resp. tract		NA		
Sensitisation	skin		NA		
	resp. tract		NA		
Repeated dose toxicity sub-acute/ sub-chronic/ chronic	oral				
	dermal				
	inhalation				
Mutagenicity	in vitro				
	in vivo				
Carcinogenicity	oral				
	dermal				
	inhalation				
Reproductive toxicity[8] fertility impairment	oral	NA			
	dermal	NA			
	inhalation	NA			
Reproductive toxicity developmental tox	oral	NA			
	dermal	NA			
	inhalation	NA			

1 NOAEL (NOAEC), LOAEL , T25, BMD(L)10 or any other dose descriptor; indicate whether this concerns a no or lowest observed effect level etc
2 In this column the relevant effect for which the dose descriptor is determined is provided
3 This column is for indicating whether data were available, whether the substance is classified for this endpoint, for shortly describing specifics of the study (e.g. 28-d gavage rat, 5 d/wk or 2-gen diet rat, 7 d/wk), and for indicating (additional) uncertainty in available data
4 Local exposure: units are mg/m3 for inhalation, and mg/cm2 or ppm for dermal exposure
5 Systemic: units are mg/m3 for inhalation, and mg/kg bw/day for oral and dermal exposure
6 In general, sublethal toxicity is a more rational starting point for acute toxicity than mortality data; information on acute toxicity may also be derived from e.g. repeated dose toxicity studies or reproductive toxicity studies
7 Not Applicable
8 These repeated exposure studies may also show relevant acute effects of the test substance; these should be accounted for under the endpoint acute toxicity

5.11.2 Correction of dose descriptors if needed (for example route-to-route extrapolation), application of assessment factors and derivation of the endpoint specific DN(M)EL

Table 27: Corrected dose descriptor(s) per endpoint and endpoint-specific DNEL(s)/DMEL(s) for the relevant exposure pattern[9]

Endpoint		**Most relevant quantitative dose descriptor[10] (appropriate unit)**		**Corrected dose descriptor (appropriate unit)**		**Overall AF applied**	**Endpoint-specific DNEL/DMEL (appropriate unit)**	
		Local[11]	Systemic[12]	Local[3]	Systemic[4]		Local[3]	Systemic[4]
Acute toxicity	oral							
	dermal							
	inhalation							
Irritation/Corrosivity	skin		NA[13]		NA			NA
	eye		NA		NA			NA
	resp. tract		NA		NA			NA
Sensitisation	skin		NA		NA			NA
	resp. tract		NA		NA			NA
Repeated dose toxicity sub-acute/ sub-chronic/ chronic	oral							
	dermal							
	inhalation							
Mutagenicity	in vitro							
	in vivo							
Carcinogenicity	oral							
	dermal							
	inhalation							

[9] Repeat as appropriate for the different populations (workers/general population and eventually specific sensitive population)

[10] NOAEL (NOAEC), LOAEL , T25, BMD10 etc or any other dose descriptor; indicate whether this concerns a no or lowest observed effect level etc

[11] Local exposure: units are mg/m^3 for inhalation, and mg/cm^2 or ppm for dermal exposure

[12] Systemic: units are mg/m^3 for inhalation, and mg/kg bw/day for oral and dermal exposure

[13] Not Applicable

Endpoint		**Most relevant quantitative dose descriptor[10] (appropriate unit)**		**Corrected dose descriptor (appropriate unit)**		**Overall AF applied**	**Endpoint-specific DNEL/DMEL (appropriate unit)**	
		Local[11]	Systemic[12]	Local[3]	Systemic[4]		Local[3]	Systemic[4]
Reproductive toxicity fertility impairment	oral	NA		NA			NA	
	dermal	NA		NA			NA	
	inhalation	NA		NA			NA	
Reproductive toxicity developmental tox	oral	NA		NA			NA	
	dermal	NA		NA			NA	
	inhalation	NA		NA			NA	

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5.11.3 Selection of the critical DNEL(s)/DMELs and/or qualitative/semi-quantitative descriptor for critical health effects

Table 28: DN(M)ELs for workers

Exposure pattern	Route	Descriptors	DNEL/DMEL (appropriate unit)	Most sensitive endpoint
Acute - systemic effects	dermal (mg/kg bw /day)	14		
	Inhalation (mg/m^3)			
Acute - local effects	Dermal (mg/cm^2)			
	Inhalation (mg/m^3)			
Long-term - systemic effects	Dermal (mg/kg bw /day)			
	Inhalation (mg/m^3)			
Long-term – local effects	Dermal (mg/cm^2)			
	Inhalation (mg/m^3)			

Discussion

Table 29: DN(M)ELs for the general population[15]

Exposure pattern	Route	Descriptors	DNEL/DMEL (appropriate unit)	Most sensitive endpoint
Acute - systemic effects	Dermal (mg/kg bw /day)			
	Inhalation (mg/m^3)			
	Oral (mg/kg bw /day)			
Acute - local effects	Dermal (mg/cm^2)			
	Inhalation (mg/m^3)			
Long-term - systemic effects	dermal(mg/kg bw /day)			
	Inhalation (mg/m^3)			
	oral(mg/kg bw /day)			
Long-term – local effects	Dermal (mg/cm^2)			
	Inhalation (mg/m^3)			

Discussion

[14] Values in IUCLID 5 are DNEL/DMEL/ not quantifiable

[15] General population includes consumers and humans via the environment. In rare cases it may also be relevant to derive a DNEL for specific subpopulations, such as children. In this case the table need to be repeated.

6 HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

6.1 Explosivity

The substance has no explosive properties.

6.2 Flammability

The substance is non flammable.

6.3 Oxidising potential

The substance has no oxidizing properties.

7 ENVIRONMENTAL HAZARD ASSESSMENT

7.1 Aquatic compartment (including sediment)

7.1.1 Toxicity data

7.1.1.1 Fish

7.1.1.1.1 Short-term toxicity to fish

The results are summarised in the following table:

Table 30: Overview of short-term effects on fish

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)

Data waiving *(if applicable)*

Not applicable

Discussion

The results of single-species acute toxicity tests with NaOH are summarised in **Table 34**, 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 following information is taken into account for any hazard / risk / persistency assessment:

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

Additional data on acute toxicity (not listed in Table 34)
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.

7.1.1.1.2 Long-term toxicity to fish

The results are summarised in the following table:

Table 31: Overview of long-term effects on fish

See Discussion

Data waiving *(if applicable)*

Not applicable

Testing proposal *(if applicable)*

Not applicable

Discussion

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.

The following information is taken into account for any hazard / risk / persistency assessment:

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

7.1.1.2 Aquatic invertebrates

7.1.1.2.1 Short-term toxicity to aquatic invertebrates

The results are summarised in the following table:

Table 32: Overview of short-term effects on aquatic invertebrates

Freshwater invertebrates					
Daphnia 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

Data waiving *(if applicable)*

Not applicable

Discussion

The results for invertebrates are very similar to those for fish, with a range of 33 to 450 mg/l.

The following information is taken into account for any hazard / risk / persistency assessment:

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

Additional data on acute toxicity (not listed in Table 36)

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.

7.1.1.2.2 Long-term toxicity to aquatic invertebrates

The results are summarised in the following table:

Table 33: Summary of long-term effects on invertebrates

Method	Results	Remarks	Reference
--	--	--	--

Data waiving *(if applicable)*

Not applicable

Testing proposal *(if applicable)*

Not applicable

Discussion

The following information is taken into account for any hazard / risk / persistency assessment:

7.1.1.3 Algae and aquatic plants

The results are summarised in the following table:

Table 34: Overview of effects on algae and aquatic plants

Method	Results	Remarks	Reference

Data waiving *(if applicable)*

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.

Discussion

Not applicable

Effects on algae / cyanobacteria

Not applicable

The following information is taken into account for any hazard / risk / persistency assessment:

Not applicable

Effects on aquatic plants other than algae

Not applicable

The following information is taken into account for any hazard / risk / persistency assessment:

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: > 25 mg/l) and there are sufficient data on the pH ranges that are tolerated by major taxonomic groups.

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.

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.

7.1.1.4 Sediment organisms

The results are summarised in the following table:

Table 35: Overview of effects on sediment organisms

Method	Results	Remarks	Reference

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Data waiving *(if applicable)*

Not applicable

Discussion

Not applicable

The following information is taken into account for any hazard / risk / persistency assessment

7.1.1.5 Other aquatic organisms

The results are summarised in the following table:

Table 36: Overview of effects on other aquatic organisms

Method	Results	Remarks	Reference
--	--	--	--

Data waiving *(if applicable)*

Not applicable

Discussion

Not applicable

The following information is taken into account for any hazard / risk / persistency assessment:

Not applicable

7.1.2 Calculation of Predicted No Effect Concentration (PNEC)

7.1.2.1 PNEC water

Table 37: PNEC aquatic

	Value	Assessment factor	Remarks/Justification

7.1.2.2 PNEC sediment

Table 38: PNEC sediment

	Value	Assessment factor	Remarks/Justification

Discussion PNECs:

A generic PNEC cannot be derived from single-species toxicity data for NaOH because the risk assessment will only deal with the (potential) pH changes related to local OH^- discharges. 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.
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.

7.2 Terrestrial compartment

7.2.1 Toxicity data

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.

7.2.1.1 Toxicity to soil macro organisms

The results are summarised in the following table:

Table 39: Overview of effects on soil macro-organisms

Method	Results	Remarks	Reference
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Data waiving *(if applicable)*

Not applicable

Testing proposal *(if applicable)*

Not applicable

Discussion of effects on soil macro-organisms except arthropods

The following information is taken into account for effects on soil macro-organisms except arthropods for the derivation of PNEC

Not applicable

Discussion of effects on soil arthropods

Not applicable

The following information is taken into account for effects on soil arthropods for the derivation of PNEC:

Not applicable

7.2.1.2 Toxicity to terrestrial plants

The results are summarised in the following table:

Table 40: Overview of effects on terrestrial plants

Method	Results	Remarks	Reference
--	--	--	--

Data waiving *(if applicable)*

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.

Testing proposal *(if applicable)*

Not applicable

Discussion

The following information is taken into account for toxicity on terrestrial plants for the derivation of PNEC:

Not applicable

7.2.1.3 Toxicity to soil micro-organisms

The results are summarised in the following table:

Table 41: Overview of effects on soil micro-organisms

Method	Results	Remarks	Reference
--	--	--	--

Data waiving *(if applicable)*

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.

Testing proposal *(if applicable)*

Not applicable

Discussion

The following information is taken into account for toxicity on soil micro-organisms for the derivation of PNEC:

Not applicable

7.2.1.4 Toxicity to other terrestrial organisms

The results are summarised in the following table:

Table 42: Overview of effects on terrestrial arthropods other than soil macro-organisms

Method	Results	Remarks	Reference
--	--	--	--

Data waiving *(if applicable)*

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.

Testing proposal *(if applicable)*

Not applicable

Discussion

The following information is taken into account for any hazard / risk assessment:

Not applicable

7.2.2 Calculation of Predicted No Effect Concentration (PNEC_soil)

Table 43: PNEC soil

	Value	Assessment factor	Remarks/Justification

Discussion PNECs

No relevant distribution into the terrestrial compartment is to be expected. The main target for environmental distribution is water.

7.3 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).

7.4 Microbiological activity in sewage treatment systems

7.4.1 Toxicity to aquatic micro-organisms

The results are summarised in the following table:

Table 44: Overview of effects on terrestrial arthropods other than soil macro-organisms

Species	Results	Remarks	Reference

Data waiving *(if applicable)*

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. Taking into account the existing EU Directives for pH control for surface water 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.
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 and are often covered by EU and/or national regulations.
Conclusion:
Regarding the conclusion 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.

Testing proposal *(if applicable)*

Not applicable

Discussion

The following information is taken into account for effects on aquatic micro-organisms for the derivation of PNEC:

Not applicable

7.4.2 PNEC for sewage treatment plant

Table 45: PNEC sewage treatment plant

	Value	Assessment factor	Remarks/Justification

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

7.5 Non compartment specific effects relevant for the food chain (secondary poisoning)

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

7.5.1 Toxicity to birds

The results are summarised in the following table:

Table 46: Overview of effects on birds

Method	Results	Remarks	Reference
--	--	--	--

Data waiving *(if applicable)*

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

Testing proposal *(if applicable)*

Not applicable

Discussion

Not applicable

The following information is taken into account for effects on birds for the derivation of PNEC:

Not applicable

7.5.2 Toxicity to mammals

The results are summarised in the following table:

Table 47: Overview of effects on mammals

Method	Results	Remarks	Reference
--	--	--	--

Data waiving *(if applicable)*

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

Discussion

Not applicable

The following information is taken into account for effects on mammals for the derivation of PNEC:

Not applicable

7.5.3 Calculation of PNECoral (secondary poisoning)

Table 48: PNEC oral

	Value	Assessment factor	Remarks/Justification
PNEC oral (mg/kg food)	--	--	--

7.6 Conclusion on the environmental classification and labelling

The hazard assessment of NaOH reveals no need to classify the substance as dangerous to the environment. Furthermore bioaccumulation in organisms is not relevant for NaOH. Based on this, there is no need to perform a risk assessment for secondary poisoning.

8 PBT AND VPVB ASSESSMENT

8.1 Assessment of PBT/vPvB Properties – Comparison with the Criteria of Annex XIII

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

8.1.1 Persistence Assessment

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

8.1.2 Bioaccumulation Assessment

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

8.1.3 Toxicity Assessment

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.

8.1.4 Summary and overall Conclusions on PBT or vPvB Properties

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.

8.2 Emission Characterisation

NaOH is neither classified as PBT nor classified as vPvB substance, therefore no emission characterisation and no environmental exposure assessment has been carried out.

9 EXPOSURE ASSESSMENT

Overview on the types of exposure (protected resource, exposure duration, uptake route, type of use) which were considered during the chemical safety assessment of sodium hydroxide (as pure solid or in aqueous solution (caustic soda solution)).

Exposure			Industrial use	Professional use	Consumer use
Human	oral	Short-term	1 –	2 –	3 –
		Long-term	4 –	5 –	6 –
	dermal	Short-term	7 +	8 +	9 +
		Long-term	10 +	11 +	12 +
	inhalative	Short-term	13 +	14 +	15 0
		Long-term	16 0	17 0	18 0
Environment	Water	Short-term	19 +	20 +	21 +
		Long-term	22 +	23 +	24 +
	Air	Short-term	25 +	26 +	27 +
		Long-term	28 +	29 +	30 +
	Soil	Short-term	31 +	32 +	33 +
		Long-term	34 +	35 +	36 +

Explanation:

\+ Assessed exposure

– A priori excluded uses

0 Not assessed/not intended exposure

9.1 Generic Exposure Scenarios

9.1.1 Generic Exposure Scenarios by ECETOC TRA

In case of Sodium hydroxide exists a comprehensive EU-Risk Assessment Report, therefore no calculation was performed with ECETOC TRA. In 9.2 an improved Substance group scenario will be reported (high lighted in green).

Table 49: Overview on workplace exposure scenarios by ECETOC TRA

process cate-gories [PROC]	Use Scenarios	Duration of activity [hours]	LEV (Y/N)	Estimated Exposition [ppm]	MoE [DNEL/ est expo]	Further assessment required
PROC 1	Use in a closed process with no likelihood of exposure					
PROC 2	Use in closed process with occasional controlled exposures e.g. during sampling					
PROC 3	Use in a closed batch process i.e. where only limited opportunity for breaching arises e.g.					

	sampling					
PROC 4	Use in a batch or other process (including related process stages e.g. filtration, drying) where opportunities for exposure arise e.g. sampling, dis/charging of materials					
PROC 5	Use in a batch process including chemical reactions and/or the formulation by mixing, blending or calendaring of liquid and solid-based products					
PROC 6	Spraying of the substance or preparations containing the substance in industrial applications e.g. coatings					
PROC 7	Dis/charging the substance (or preparations containing the substance) to/from vessels					
PROC 8	Filling containers with the substance or its preparations (including weighing)					
PROC 9	Roller application or brushing of adhesives and other surface coatings					
PROC 10	Use as a blowing agent in the manufacture of foams, etc.					
PROC 11	Use for coating/treatment of articles, etc. (including cleaning) by dipping or pouring					
PROC 12	Production of products or articles from substance by compression, tabletting, extrusion or pelletisation					
PROC 13	Use as a laboratory reagent					
PROC 14	Use as a fuel					
PROC 15	Use as a lubricant (including metal working fluids)					

9.1.2 Generic Exposure Scenarios by other sources

(optional)

9.2 Title of Exposure Scenario (1)

Use of Sodium hydroxide in all industrial, professional and consumer uses (except aerosol use), e.g. use as intermediate in glass-, paper-, aluminium- and detergent production, use for the production of many chemicals, use as process aid (neutralisation, cleaning agent)

9.2.1 Exposure scenario

9.2.1.1 Short title of the exposure scenario

Every industrial, commercial and private application excluding applications of aerosols with attention to the RMM. (e.g. as intermediate product for the production of glass, paper, aluminium, the manufacture of detergents, the production of a multiplicity of chemical compounds, as process aids, as neutralization agents, as cleaning agents, e.g. PC 0, PC 10, PC 15,PC 19, PC 20, PC 21, PC 23, PC35, PC 37)

9.2.1.2 Description of activities and processes covered in the exposure scenario

Every industrial, commercial and private application excluding applications of aerosols with attention to the RMM16 (PROC 1 to PROC 24 with the exception of PROC 7, PROC 11, PROC 22, PROC 24 as a component of products: Accumulators, batteries, AC 3). Cleaning sprays for baking ovens for consumers are not evaluated (aerosol use)

9.2.1.3 Operational conditions related to frequency, duration and amount of use

Not relevant when considering the Risk Management Measures

Justification:

The different quantities in handling in different uses and also the amounts of exposure are not relevant for the exposure assessment, because the risk management measures (e.g. neutralisation of waste water to a pH-value mandatory for the water body) are independent of the released amounts.

Table 50: Duration, frequency and amounts related to exposure of workers

Information type	Data field	Explanation
Use amount per worker [workplace] per day	--	See justification
Duration per day at workplace [for one worker]	Industrial and professional use: < 0.5 h/d for short term use > 0.5 h/d for long term use	
Frequency at workplace [for one worker]	See above	
Other determinants related to duration, frequency and amount of use	none	

16 Longer term inhalative exposures cannot occur, except with spray applications, because of the physical and chemical properties (NaOH solid is hygroscopic and has a very low vapour pressure), particularly when aerosol formation is prevented by a very high viscosity.

Remarks or additional information:

Table 51: Duration, frequency and amount related to consumer uses

Information type	Data field	Explanation
Number of uses/applications per day/year by one consumer [in one flat]	See below	
Amount of product per application	--	See justification
Duration of use per day or per year	< 0.5 h/week or <1d/a for short term use > 0.5 h/week or > 1d/a for long term use	
Fraction of amount available for exposure via air (migration fractions, release fraction)[1]	--	See justification
Fraction of amount available for exposure via skin (migration fractions, release fraction)[1]	--	See justification
Fraction of amount available for exposure via ingestion (migration fractions, release fraction)[1]	--	See justification
Other determinants related to duration, frequency and amount	None	

1) see Guidance Table D.5.3 and section R.15.4

Remarks or additional information:

Table 52: Duration, frequency and amounts related to emissions from industrial sites

Information type	Data field	Explanation
Annual amount used per site	Kg/y	See justification
Emission days per site	d/y	See justification
Other determinants related to duration, frequency and amount		none

Remarks or additional information:

Table 53: Duration, frequency and amounts related to emissions from wide disperse use

Information type	Data field	Explanation
Annual amount used in a preparation category (ies) selected in 9.1.1.1	kg/y	See justification
Emission days per year related to that preparation category	d/y	See justification
Other determinants related to duration, frequency and amount		none

Remarks or additional information:

Table 54: Duration, frequency and amounts related to emissions from article service life

Justification:

In articles NaOH is contained only in a couple of cases, mostly in closed systems (e.g. batteries)

Information type	Data field	Explanation
annual amount processed into an article category (ies) selected in 9.1.1.1	kg/y	
Fraction of amount available for releases to the environment (migration fractions, release fraction)[1]	%	
Emission days per year related to that article category	d/y	
Other determinants related to duration, frequency and amount		

[1)] *See Guidance Chapter R.17 on releases from articles*

Remarks or additional information:

9.2.1.4 Operational conditions related to product characteristics

Table 55: Product Characteristic

Information type	Data field	Explanation
Type of product the information relates to	Sodium hydroxide (solid) and preparations with water ()	
Physical state of product	Solid and liquid	
For solids: Categorisation of dust grades see table Guidance R.14-8	Low	
Concentration of substance in product	Solid: 100 %, Liquid: >1% - < 100%	
Concentration after dilution for use (if relevant)	> 1 %	
Surface-mass ratio of the article	Not relevant	
Service life span of the article	Not relevant	
Condition of use promoting release from article (*see environmental release category (ERC) 10a to 11b in table R.16-22)*	Not relevant	Mostly closed systems

Remarks or additional information:

9.2.1.5 Other operational conditions of use

Table 56: Respiration volume and skin contact under conditions of worker uses

Information type	Data field	Explanation
Respiration volume under conditions of use	10 m^3/d	
Skin contact area with the substance under conditions of use	None, because risk management measures	

	prevent skin contact	
Other determinants related to respiration and skin contact	None	

Table 57: Respiration volume, skin contact and ingestion under condition of consumer uses

Information type	Data field	Explanation
Skin/mouth contact area	Not relevant	Not allowed due to irritant property, prevention through risk management measures
Inhalation rate under conditions of use	Not relevant	Use as aerosol is not intended
Body weight of	Not relevant	
Other determinants related to …	None	

Remarks or additional information:

Table 58: Conditions leading to dilution of initial release related to human health

Information type	Data field	Explanation
Room size and ventilation rate	m^3 ; exchange per hour	
Other determinants related to dilution		

Remarks or additional information:

Justification:

Not relevant: Risk Management Measures which are already being applied shall be taken into account

Table 59: Conditions leading to dilution of initial release related to environment

Information type	Data field	Explanation
Discharge volume of sewage treatment plant	m^3/d	
Available river water volume to receive the emissions from a site	m^3/d	
Other determinants related to dilution		

Remarks or additional information:

Justification:

Not relevant: Risk Management Measures which are already being applied shall be taken into account

Table 60: Process condition

Justification:

Not relevant: Risk Management Measures which are already being applied shall be taken into account

Table 61: Technical fate of substance and losses from process to waste, waste water and air

Information type	Data field	Explanation
Fraction of applied amount lost from process to waste gas,	kg/kg	Short description
Fraction of applied amount lost from process to waste water (after internal recycling of substance, if any)	kg/kg	
Fraction of applied amount lost from process to waste (after internal recycling of substance, if any)	kg/kg	
Fraction of applied amount leaving the site with products	kg/kg	Short description
Fraction consumed in process	kg/kg	Short description

Remarks or additional information:

Justification:

Risk Management Measures which are already being applied shall be taken into account

9.2.1.6 Risk management measures

The following RMMs have to be seen as examples. Other RMMs can be applied also when the same level of safety could be achieved.

9.2.1.6.1 Risk management measures related to workers (industrial and professional)

a) Instructions:

Skin contact inadmissible - Touching forbidden
- Use without protective gloves, eye protector banned
- spilled caustic soda solution immediately eliminate or neutralize,
- Do not inhale aerosols, fumes
- additional instruction, e.g.
- clean contaminated protective gloves with flowing water before taking off.
- clean or take off protective clothing immediately after contaminating.
- Examine protective gloves for damage before beginning the activity.

Use caustic soda solution only after dilution with water to final concentrations of less than 1%. –

Pour only with small heads (20 cm or less) or let liquid flow on the rim of container (avoidance of splashes)
(valid for all activities/all PROCs - as well as for the array nrs. 7, 8, 11, 13, 14.).

b) Product-related measures, e.g.

- Dilution under 1% before further use as…. (Cleaning agents), (in principle for all activities/PROCs, examine whether application in diluted form is possible - substitution principle).
- High viscosity adjustment with aids to avoid splashes
- Use in spray products inadmissible.
- Delivery only as barrel commodity and/or in the tank car

c) Organizational measures:

- Handling permissible only after instruction on the dangers.
- Regular control of the observance of the instructions - sanctioning for offence,
- Regular control of the effectiveness of the technical measures,
- Regular control of the application of the personal measures, (valid for all indicated activities/all aforementioned PROCs)

Additional measures, e.g.
- Entrance to production/processing only for technical personnel,
- Delivery only to the specialized trade.
- Hold only the quantity necessary for the processing ready.

d) Technical measures, e.g.:

- Closed systems (PROC 1 - PROC 3)
- Covering of open containers (e.g. screens)
- Transport over pipes, technical barrel filling/emptying of barrel with automatic systems (suction pumps etc.) (PROC 8 - PROC 9):
- Use of pliers, grip arms with long handles with manual use "to avoid direct contact and exposure by splashes (no working over one's head) (PROC 10, PROC 13, PROC 19).

e) Personal protection measures:

- Disposable gloves for brief application
- Gloves with 8-hour break-through security for longer application
- Eye protector (all activities/PROCs)

Additional measures, e.g.
- Protective clothing, aprons, shield, protective helmet

Table 62: Measures related to the design of product (other than concentration) related to workers

Table 63: Containment and local exhaust ventilation related to workers

Information type	Data field	Explanation
Containment plus good work practice required	Effectiveness in terms of residual exposure	Short description on the technical type and level of containment
Local exhaust ventilation required plus good work practise	Effectiveness in terms of reduction factor against situation without LEV or residual exposure	short description

Remarks or additional information:

Table64 : Personal protection equipment (PPE) required under regular working conditions

Table65 : Other risk management measures related to workers

9.2.1.6.2 Risk management measures related to consumers

Instructions:

Skin contact inadmissible - touching forbidden.

**Specific RMMs for consumer protection will be elaborated together with DU organizations (Examples for such risk management measures might be: use permissible only with protective gloves which are impermeable against caustic soda solution, and with eye protector (if possible, solution provided together with gloves/eye protector); Before application read instructions and obey; Clean protective gloves thoroughly with much water before taking off; Eating/drinking banned - (strongly) corrosive ; Store inaccessible for children (e.g. cleaning agents in a locked cabinet) b) Product-related measures, e.g.: - „Dilution under 1% “ - Child-secured packing - Delivery only with integrated dosing equipment - Delivery only in small amounts - Delivery only in very viscous preparations - Providing together with protective gloves/eye protector c) Organizational measures: - Delivery only to persons over 18 years after instruction).

Table 66: Measures related to the design of product (other than concentration) related to consumers

Table 67: Instructions addressed to consumers

Table 68: PPE required under regular conditions of consumer use

9.2.1.6.3 Risk management measures related to environment

a) Instructions, e.g.:

-.May not be let undiluted into wastewater:

-.Neutralize before introduction in open waters;

- Remainders on application devices (e.g. putties) with much water clean.

b) Product-related measures: none.

c) Organizational measures:

- regular control of the pH value during introduction into open waters.

d) Technical measures:

- Neutralization to the locally prescribed pH value.

- Dilution to the locally prescribed pH value.

Table 69 : Measures related to the design of products (other than concentration)

Table 70: Risk management measures related to environmental emissions from sites

Information type	Data field	Explanation
Onsite pre-treatment of waste water	Effectiveness [fraction in waste water related fraction emitted to sewage]	Short description of technique
Resulting fraction of applied amount in waste water released from site	Kg/kg	
Air emission abatement	Effectiveness [fraction in waste air compared to fraction emitted]	Short description of technique
Resulting fraction of applied amount in waste gas released to environment	kg/kg	
Fraction of substance in waste treated onsite aiming at final disposal (with or without recovery of heat.)	kg/kg	
Fraction of applied amount sent to external waste treatment (sum of direct losses from processes and residues from waste water and waste gas treatment)	kg/kg	Short description of technique
Municipal or other type of external waste water treatment	Effectiveness of substance removal [fraction of substance in treated waste water compared to fraction emitted into sewer]	Short description of technique
Recovery of sludge for agriculture or horticulture	Yes/no	
Other risk management measures		

Remarks or additional information:

Justification: Not relevant because of required neutralisation of NaOH when discharged to waters

Table 71: Risk management measures related to emissions to the environment from wide disperse use

Information type	Data field	Explanation

Information type	Data field	Explanation
Municipal waste water treatment	Yes/no	Short description of technique including sludge disposal
Other risk management measures		

Remarks or additional information:

Table 72: Other RMM

9.2.1.7 Waste related measures

Discharges of NaOH from production to sewage treatment plants (STP)/waste water treatment plants and receiving waters are well controlled. Only bigger amounts of NaOH waste have to be neutralised or diluted. Taking into account the existing EU Directives for pH-control for surface water and national regulations to control the pH of waster waters and surface waters is is concluded that STPs and surface waters are sufficiently protected with regard to pH changes.

No special measures necessary. Only for larger quantities of waste, possible neutralization.

Table 73: Waste management measures

Information type	Data field	Explanation
Amount of substances in waste resulting from the activities/processes covered in the exposure scenario	kg/y	
Amount of substances in waste resulting from service life of articles	kg/y	
Type of waste, suitable waste codes		
Type of external treatment aiming at recycling or recovery of substances	Type of treatment according to Appendix R.18-1	
Fraction of the amount of substance in waste stream recovered.	kg/kg	
Type of external treatment aiming at final disposal of the waste	Type of treatment according to Appendix R.18-1	
Fraction of substance released into the environment via air (after abatement)	kg/kg	
Fraction of substance released into the environment via waste water (after abatement)	kg/kg	
Fraction of substance released into the environment via air (after abatement)	kg/kg	
Fraction of substance released into the environment via waste water (after abatement)	kg/kg	
Fraction of substance disposed of as secondary waste	kg/kg	
Other waste management measures		

Remarks or additional information:

Table 74: Other waste management measures

9.2.2 Exposure estimation

Justification:

If risk reduction measures which are already being applied are taken into account no human and environmental exposure resulting in irritating effects of NaOH are expected.

9.2.2.1 Workers exposure

9.2.2.1.1 Acute/Short term exposure

Table 75: Acute exposure concentrations to workers

Routes of exposure	Estimated Exposure Concentrations		Measured exposure concentrations		Explanation / source of measured data
	value	unit	Value	unit	
Dermal exposure					
Inhalation exposure					

Summary of the short-term exposure values.

Table 76: Summary of acute exposure concentrations to workers

Routes of exposure	Concentrations	Justification
Dermal exposure (in mg/cm^2)		
Inhalation exposure (in mg/m^3)		

9.2.2.1.2 Long-term exposure

Table 77: Long-term exposure concentrations to workers

Routes of exposure	Estimated Exposure Concentrations		Measured exposure concentrations		Explanation / source of measured data
	value	unit	Value	unit	
Dermal exposure					
Inhalation exposure					

Summary of the long-term exposure values.

Table 78: Summary of long-term exposure concentration to workers

Routes of exposure	Concentrations	Justification
Dermal exposure (in mg/cm^2)		
Inhalation exposure (in mg/m^3)		

9.2.2.2 Consumer exposure

9.2.2.2.1 Acute/Short term exposure

When several life cycle steps are relevant for the exposure scenario, then exposure at these different stages should be taken into account (e.g. service life of article)

Table 79: Acute exposure concentrations to consumers

Routes of exposure	Estimated Exposure Concentrations		Measured exposure concentrations		Explanation / source of measured data
	value	unit	Value	unit	
Oral exposure					
Dermal exposure					
Inhalation exposure					

Summary of the short-term exposure values.

Table 80: Summary of acute exposure concentrations to consumers

Routes of exposure	Concentrations	Justification

Oral exposure (in mg/kg bw/d)		
Dermal exposure (in mg/cm^2)		
Inhalation exposure (in mg/m^3)		

9.2.2.2.2 Long-term exposure

Table 81: Long term exposure concentrations to consumers

Routes of exposure	**Estimated Exposure Concentrations**		**Measured exposure concentrations**		**Explanation / source of measured data**
	value	**unit**	**value**	**unit**	
Oral exposure					
Dermal exposure					
Inhalation exposure					

Summary of the long-term exposure values.

Table 82: Summary of long term exposure concentrations to consumers

Routes of exposure	**Concentrations**	**Justification**
Oral exposure (in mg/kg bw/d)		
Dermal exposure (in mg/cm^2)		
Inhalation exposure (in mg/m^3)		

9.2.2.3 Indirect exposure of humans via the environment (oral)

Table 83: Concentration for oral exposure of humans via the environment

	Estimated exposure concentrations		**Measured exposure concentrations**		**Explanation / source of measured data**
	value	**unit**	**value**	**unit**	
Wet fish					
Drinking water					

	Estimated exposure concentrations		Measured exposure concentrations		Explanation / source of measured data
	value	unit	value	unit	
Meat					
Milk					
Other					

Summary of the exposure concentration in to be used for the risk characterisation of indirect exposure of man via the environment

Table 84: Total daily dose for oral exposure of humans via the environment

Total daily dose for oral exposure via the environment (mg/kg bw/d)		Justification
Exposed via local concentration	Exposed via local and regional concentration	

9.2.2.4 Environmental exposure

In case the exposure scenario is covering several life stages, the section below has to be repeated to cover those different life stages within this section.

9.2.2.4.1 Environmental releases

Table 85: Releases to the environment from point source

compartments	Predicted releases (kg/d)	Measured release (kg/d)	Explanation / source of measured data
Aquatic (without WWTP)	[17]		*These data correspond to release to waste water*
Aquatic (after WWTP)			*These correspond to release to natural waters after the waste water treatment plant.*
Air (direct + WWTP)			
Soil (direct only)			

[17] The predicted release are estimated from the "annual amount used" and the "number emission days" (cf **Fehler! Verweisquelle konnte nicht gefunden werden.**) and the "fraction of applied amount lost from process to waste water" (cf **Fehler! Verweisquelle konnte nicht gefunden werden.**)

Table 86: Releases to the environment from dispersive use

compartments	Predicted releases (kg/d)	Measured release (kg/d)	Explanation / source of measured data
Aquatic (without WWTP)	18		*These data correspond to release to waste water*
Aquatic (after WWTP)			*These correspond to release to natural waters after the waste water treatment plant.*
Air (direct + WWTP)			
Soil (direct only)			

Summary of the releases taken into account for the exposure estimation.

Table 87: Summary of the releases to the environment

Compartments	Release from point source (kg/d) (local exposure estimation)	Total release for regional exposure estimation (kg/d)	Justification
Aquatic (without WWTP)			
Aquatic (after WWTP)			
Air (direct + WWTP)			
Soil (direct releases only)			

9.2.2.4.2 Exposure concentration in waste water treatment plants (WWTP)

Table 88: Concentrations in waste water

Compartments	Estimated exposure concentrations		Measured exposure concentrations		Explanation / source of measured data
	value	unit	value	unit	
Waste water					

[18] The predicted release are estimated from the "annual amount used" and the "number emission days" (cf **Fehler! Verweisquelle konnte nicht gefunden werden.** and/or **Fehler! Verweisquelle konnte nicht gefunden werden.**) and the "fraction of applied amount lost from process to waste water" (cf **Fehler! Verweisquelle konnte nicht gefunden werden.**)

Compartments	Estimated exposure concentrations		Measured exposure concentrations		Explanation / source of measured data
	value	unit	value	unit	
Waste water sludge					

Summary of the exposure concentration in waste water treatment plants taken into account for further exposure estimation (water and soil concentrations) or risk characterisation for micro organisms in the WWTP

	Value	Justification
Concentration in wastewater (PECstp)(in mg/l)		
Concentration in waste water sludge (in mg/kg d.w.)		

9.2.2.4.3 Exposure concentration in aquatic pelagic compartment

Table 89: Local concentrations in water

Compartments	Estimated exposure concentrations		Measured local exposure concentrations		Explanation / source of measured data
	value	unit	value	unit	
Freshwater					*Estimated local exposure concentration based on…*
					*Estimated predicted exposure concentration (PEC) = estimated local exposure concentration + regional concentration (from **Fehler! Verweisquelle konnte nicht gefunden werden.**)*
					Measured concentration in…
Marine water					*Estimated local exposure concentration based on…*
					*Estimated predicted exposure concentration (PEC) = estimated local exposure concentration + regional concentration (from **Fehler! Verweisquelle konnte nicht gefunden werden.**)*
					Measured concentration in…
Intermittent releases to water					

Summary of the Predicted Exposure Concentrations (PEC) in the aquatic pelagic compartment taken into account for risk characterisation

Table 90: Predicted Exposure Concentrations (PEC) in aquatic compartment

Compartments	**Local concentration**	**PEC aquatic (local+regional)**	**Justification**
Freshwater (in mg/l)			
Marine water (in mg/l)			
Intermittent releases to water (in mg/l)			

9.2.2.4.4 Exposure concentration in sediments

Table 91: Local concentrations in sediment

Compartments	**Estimated exposure concentrations**		**Measured local exposure concentrations**		**Explanation** *(including if equilibrium method partitioning has been used, report the partitioning coefficient)* / **source of measured data**
	value	**unit**	**value**	**unit**	
Freshwater sediments					*Estimated local exposure concentration based on…*
					*Estimated predicted exposure concentration (PEC) = estimated local exposure concentration + regional concentration (from **Fehler! Verweisquelle konnte nicht gefunden werden.**)*
					Measured concentration in…
Marine water sediments					*Estimated local exposure concentration based on…*
					*Estimated predicted exposure concentration (PEC) = estimated local exposure concentration + regional concentration (from **Fehler! Verweisquelle konnte nicht gefunden werden.**)*
					Measured concentration in…

.

Summary of the exposure concentration in aquatic sediments taken into account for risk characterisation

Table 92: Predicted Exposure Concentrations (PEC) in sediments

Compartments	**Local concentration**	**PEC sediment (local+regional)**	**Justification**
Freshwater sediments (in mg/kg d.w)			
Marine water sediments (in mg/kg d.w.)			

9.2.2.4.5 Exposure concentrations in soil and groundwater

Table 93: Local concentrations in soil

Compartments	**Estimated exposure concentrations**		**Measured local exposure concentrations**		**Explanation** *(including number of days for averaging)* / **source of measured data**
	value	**unit**	**value**	**unit**	
Agricultural soil averaged					*Estimated local exposure concentration based on…*
					Estimated predicted exposure concentration (PEC) = estimated local exposure concentration + regional concentration (from ***Fehler! Verweisquelle konnte nicht gefunden werden.****)*
					Measured concentration in…
Grassland averaged					
Groundwater					

Summary of the Predicted Exposure Concentration (PEC) in soil taken into account for risk characterisation

Table 94: Predicted Exposure Concentrations (PEC) in soil and groundwater

	Local concentration	**PEC soil/groundwater (local+regional)**	**Justification**
Agricultural soil averaged (mg/kg ww)			
Grassland averaged (mg/kg ww)			
Groundwater(mg/l)			

9.2.2.4.6 Atmospheric compartment

Table 95: Local concentrations in air

	Estimated exposure concentrations		**Measured local exposure concentrations**		**Explanation / source of measured data**
	value	**unit**	**value**	**unit**	
During emission					
annual average					

	Estimated exposure concentrations		Measured local exposure concentrations		Explanation / source of measured data
	value	unit	value	unit	
Annual total deposition					

Summary of the Predicted Exposure Concentration in soil taken into account for risk characterisation

Table 96: Predicted Exposure Concentration (PEC) in air

	Local concentration	PEC air (local+regional)	Justification
During emission (µg/m3)			
annual average (µg/m3)			
Annual deposition (µg/m²/d)			

9.2.2.4.7 Exposure concentration relevant for the food chain (Secondary poisoning)

Table 97: Local concentration relevant for secondary poisoning

	Predicted exposure concentrations		Measured local exposure concentrations		Explanation / source of measured data
	value	unit	value	unit	
Concentration in food of fish eating predator					*Estimated local exposure concentration based on…*
					*Estimated predicted exposure concentration (PEC) = estimated local exposure concentration + regional concentration (from **Fehler! Verweisquelle konnte nicht gefunden werden.**)*
					Measured concentration in…
Concentration in food of fish eating top-predator (marine)					*Estimated local exposure concentration based on…*
					*Estimated predicted exposure concentration (PEC) = estimated local exposure concentration + regional concentration (from **Fehler! Verweisquelle konnte nicht gefunden werden.**)*
					Measured concentration in…
Concentration in earthworm					

.

Summary of the Predicted Exposure Concentration in food for secondary poisoning taken into account for risk characterisation

Table 98: Predicted Exposure Concentration in food (PECoral) for secondary poisoning

	Local concentration	PEC oral (local+regional)	Justification
PECoral predator (in mg/kg w.w)			
PECoral top predator (in mg/kg w.w.)			
Concentration in earthworm (in mg/kg w.w.)			

9.3 (Title of exposure scenario 2)

9.3.1 Exposure scenario

9.3.2 Exposure estimation

…

9.4 Regional exposure concentrations

Table 99: Regional concentrations in the environment

	Predicted regional Exposure Concentrations		Measured regional exposure concentrations		Explanation / source of measured data
	value	unit	value	unit	
Freshwater					
Marine water					
Freshwater sediments					
Marine sediments					
Agricultural soil					
Grassland					
Air					

Table 100: Regional concentrations in food and drinking water

	Predicted regional Exposure Concentrations		Measured regional exposure concentrations		Explanation / source of measured data
	value	unit	value	Unit	
Wet fish					
Drinking water					
Meat					
Milk					

10 RISK CHARACTERISATION

Guidance for Risk Characterisation is provided in Part E.

10.1 (Title of exposure scenario 1)

10.1.1 Human health

10.1.1.1 Workers

Table 101: (Semi) Quantitative risk characterisation for workers

Route		ES 1- exposure concentrations (EC)	Leading toxic end point / Critical effect	DN(M)EL[19]	Risk characterisation ratio[20]
Dermal- local	**Acute**	In mg/cm^2			
	Long term	In mg/cm2			
Dermal- systemic	**Acute**	in mg/kg bw/d			
	Long term	in mg/kg bw/d			
Inhalation - local	**Acute**	in mg/m3			
	Long term	in mg/m3			
Inhalation - systemic	**Acute**	= Inhalation-local in mg/m3			
	Long term				
Combined routes	**Acute**				RCR Inhalation-systemic + RCR Dermal- systemic
	Long term				

[19] *The 8 D(M)NELs relevant here can be extracted from IUCLID 5 and are already reported in* Table 28.

[20] *Equal to the ratio of the relevant EC (reported in column 3) to the relevant D(M)NEL (reported in column 5)*

Table 102: Qualitative risk characterisation for workers

Route		ES 1- exposure concentrations (EC)	Leading toxic end point / Critical effect	Qualitative risk characterisation
Dermal- local	**Acute**	In mg/cm^2		
	Long term	In mg/cm2		
Dermal- systemic	**Acute**	in mg/kg bw/d		
	Long term	in mg/kg bw/d		
Inhalation - local	**Acute**	in mg/m3		
	Long term	in mg/m3		
Inhalation - systemic	**Acute**	= Inhalation-local in mg/m3		
	Long term			
Combined routes	**Acute**			
	Long term			

10.1.1.2 Consumers

Table 103: (Semi) Quantitative risk characterisation for consumers

Route		ES 1- exposure concentrations (EC)	Leading toxic end point / Critical effect	DN(M)EL[21]	Risk characterisation ratio [22]
Dermal- local	**Acute**	In mg/cm^2			
	Long term	In mg/cm2			
Dermal- systemic	**Acute**	in mg/kg bw/d			
	Long term	in mg/kg bw/d			
Inhalation - local	**Acute**	in mg/m3			
	Long term	in mg/m3			
Inhalation - systemic	**Acute**	= Inhalation-local in mg/m3			
	Long term				
Oral (systemic)	**Acute**	in mg/kg bw/d			
	Long term	in mg/kg bw/d			
Combined routes	**Acute**				RCR Inhalation-systemic + RCR Dermal- systemic
	Long term				

[21] The 8 D(M)NELs relevant here can be extracted from IUCLID 5 and are already reported in Table 28

[22] Equal to the ratio of the relevant EC (reported in column 3) to the relevant D(M)NEL (reported in column 5)

Table 104: Qualitative risk characterisation for consumers

Route		ES 1- exposure concentrations (EC)	Leading toxic end point / Critical effect	Qualitative risk characterisation
Dermal- local	**Acute**	In mg/cm^2		
	Long term	In mg/cm2		
Dermal- systemic	**Acute**	in mg/kg bw/d		
	Long term	in mg/kg bw/d		
Inhalation - local	**Acute**	in mg/m3		
	Long term	in mg/m3		
Inhalation - systemic	**Acute**	= Inhalation-local in mg/m3		
	Long term			
Oral systemic	**Acute**	in mg/kg bw/d		
	Long term	in mg/kg bw/d		
Combined routes	**Acute**			
	Long term			

10.1.1.3 Indirect exposure of humans via the environment

Table 105: (Semi) Quantitative risk characterisation for humans exposed via the environment

Route	ES 1- exposure concentrations (EC)	Leading toxic end point / Critical effect	DN(M)EL[23]	Risk characterisation ratio[24]
Dermal- systemic[25] (acute or long term)	in mg/kg bw/d			
Inhalation- systemic (long term)	in mg/m3 (from **Fehler! Verweisquelle konnte nicht gefunden werden.**)			
Oral- systemic (long term)	in mg/kg bw/d (from **Fehler! Verweisquelle konnte nicht gefunden werden.**)			
Combined routes				RCR Inhalation-systemic + RCR Oral-systemic

23 The 8 D(M)NELs relevant here can be extracted from IUCLID 5 and are already reported in Table 28

24 Equal to the ratio of the relevant EC (reported in column 3) to the relevant D(M)NEL (reported in column 5)

25 Dermal exposure is rarely relevant for exposure of man via the environment (bathing waters)

Table 106: Qualitative risk characterisation for humans exposed via the environment

Route	ES 1- exposure concentrations (EC)	Leading toxic end point / Critical effect	Qualitative risk characterisation
Dermal- systemic[26] (acute or long term)	in mg/kg bw/d		
Inhalation- systemic (long term)	in mg/m3 (from **Fehler! Verweisquelle konnte nicht gefunden werden.**)		
Oral- systemic (long term)	in mg/kg bw/d (from **Fehler! Verweisquelle konnte nicht gefunden werden.**)		
Combined routes			RCR Inhalation- systemic + RCR Oral-systemic

10.1.2 Environment

10.1.2.1 Aquatic compartment (including sediment and secondary poisoning)

Table 107: Risk characterisation for the aquatic compartment

Compartments	PEC	PNEC	PEC/PNEC	Discussion
Freshwater	*in mg/l (from **Fehler! Verweisquelle konnte nicht gefunden werden.**)*	*in mg/l (from Table 37)*		
Marine water	*idem*	*idem*		
Sediment	*in mg/kg (from **Fehler! Verweisquelle konnte nicht gefunden werden.**)*	*in mg/kg (from Table 38)*		
Aquatic freshwater food chain	*in mg/kg (from **Fehler! Verweisquelle konnte***	*in mg/kg food (from Table 48)*		

[26] Dermal exposure is rarely relevant for exposure of man via the environment (bathing waters)

	nicht gefunden werden.)			
Aquatic marine water food chain	*idem*	*idem*		

10.1.2.2 Terrestrial compartment (including secondary poisoning)

Table 108: Risk characterisation for the terrestrial compartment

Compartments	PEC	PNEC	PEC/PNEC	Discussion
Agricultural soil	*in mg/kg (from* ***Fehler! Verweisquelle konnte nicht gefunden werden.)***	*in mg/kg (from Table 43)*		
Grassland	*idem*	*idem*		
Terrestrial food chain	*in mg/kg (from* ***Fehler! Verweisquelle konnte nicht gefunden werden.)***	*in mg/kg food (from Table 48)*		

10.1.2.3 Atmospheric compartment

10.1.2.4 Microbiological activity in sewage treatment systems

Compartments	PEC	PNEC	PEC/PNEC	Discussion
STP	*in mg/l (from* ***Fehler! Verweisquelle konnte nicht gefunden werden.)***	*in mg/l (from Table 45)*		

10.2 (Title of exposure scenario 2)

10.3 Overall exposure (combined for all relevant emission/release sources)

10.3.1 Human health (combined for all exposure routes)

Table 109: Identification of relevant combination of exposure scenarios

Exposure scenarios	Combination 1	Combination 2		
ES 1				
ES 2				
ES 3				

Table 110: Risk characterisation for combined relevant emission

Relevant combination of exposure scenario	Risk characterisation ratio
Combination 1	
Combination 2	

10.3.2 Environment (combined for all emission sources)

REFERENCES

ANNEX

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