FOREWORD

POTASSIUM HYDROXIDE

CAS N°: 1310-58-3
SIDS Initial Assessment Report

For

SIAM 13

Bern, Switzerland, 6-9 November 2001

1. Chemical Name: Potassium hydroxide
2. CAS Number: 1310-58-3
3. Sponsor Country: Belgium
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4. Shared Partnership with: ICCA (Tessenderlo Chemie NV)
5. Roles/Responsibilities of the Partners:
   • Name of industry sponsor /consortium
   • Process used
6. Sponsorship History
   • How was the chemical or category brought into the OECD HPV Chemicals Programme?
     In 2001, ICCA (Tessenderlo Chemie NV) had proposed sponsor and prepared draft documents(Dossier, SIAR, SIAP). It was submitted to the SIDS contact point of Belgium on May 2001. The draft documents were revised by Belgium after discussion with Tessenderlo Chemie NV. The revised draft was discussed in detail with Tessenderlo Chemie NV on June and July 2001. After agreement, the documents were finalized and the checklist was developed by jointly by Belgium and Tessenderlo Chemie NV
7. Review Process Prior to the SIAM:
8. Quality check process:
9. Date of Submission:
10. Date of last Update: February 2002
11. Comments: No testing
OECD SIDS
POTASSIUM HYDROXIDE

SIDS INITIAL ASSESSMENT PROFILE

<table>
<thead>
<tr>
<th>CAS No.</th>
<th>1310-58-3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chemical Name</td>
<td>Potassium hydroxide</td>
</tr>
<tr>
<td>Structural Formula</td>
<td>KOH</td>
</tr>
</tbody>
</table>

**RECOMMENDATIONS**

The chemical is currently of low priority for further work.

**SUMMARY CONCLUSIONS OF THE SIAR**

**Human Health**

Solid KOH is corrosive. Depending on the concentration, solutions of KOH are non-irritating, irritating or corrosive and they cause direct local effects on the skin, eyes and gastrointestinal tract. Systemic effects are not to be expected. Solutions with concentrations higher than 2% are corrosive, while concentrations of about 0.5 to about 2.0 % are irritating. No studies are available for repeated dose toxicity, *in vivo* genotoxicity, toxicity to reproduction and development.

The reported oral rat LD50 values are 365, 273 and 1230 mg/kg bw/day. Based on the data with other potassium compounds, it could be concluded that potassium has no or a negligible contribution to the toxicity at lethal dose levels of KOH. With KCl, the NOEL in rats for repeated dose toxicity is > 1820 mg/kg bw/day, and > 88-108 mg/kg bw/day in women, and for reproduction/developmental toxicity, > 235 and > 310 mg/kg bw/day for, respectively, mice and rats. With K2CO3, the teratogenic NOEL values could be established as > 290 mg/kg bw/day for mice, and > 180 mg/kg bw/day for rats. Under normal handling and use conditions (non-irritating) neither the concentration of potassium in the blood nor the pH of the blood will be increased above normal limits and therefore KOH is not expected to cause systemically toxic levels in the blood. The renal excretion of K can be elevated and the OH- ion is neutralised by the bicarbonate buffer system in the blood. It can also be stated that the substance will neither reach the foetus nor reach male and female reproductive organs in effective toxic concentrations. Therefore, no risk for reproductive toxicity is expected. An *in vitro* genetic toxicity test indicated no evidence for a mutagenic activity. No mutagenic activity was found for the related substances NaOH (both *in vitro* and *in vivo*) nor KCl and K2CO3 (*in vitro*).

Dust formation is unlikely because of the hygroscopic properties. Furthermore KOH has a negligible vapour pressure and is rapidly neutralized in air by carbon dioxide and therefore dust and vapour exposure are not expected.

Based on the available literature, there is a risk for accidental and intentional exposure to solid KOH or to irritating or corrosive solutions of KOH. Most of the ingestion accidents seem to be related with children and seem to occur at home. Accidental skin and eye exposure seems to be less frequently reported than ingestion in the medical literature.

**Environment**

The hazard of KOH for the environment is caused by the hydroxyl ion (pH effect). For this reason the effect of KOH on the organisms depends on the buffer capacity of the aquatic or terrestrial ecosystem. Also the variation in acute toxicity for aquatic organisms can be explained for a significant extent by the variation in buffer capacity of the test medium. The LC50 value of acute fish toxicity was in the order of 80 mg/l. It was 880 mg/l for KCl and ranged between 125-189 mg/l for NaOH. The LC50 values of acute invertebrate toxicity for KCl was 660 mg/l (*Daphnia magna*) and 630 mg/l (*Ceriodaphnia dubia*), and for NaOH 40 mg/l (*Ceriodaphnia dubia*). The EC50 algae value (*Nitschera linearis*) was 1337 mg/l for KCl.
Because the buffer capacity, the pH and the fluctuation of the pH are very specific for a certain ecosystem, it was not considered useful to derive a PNEC. If it is assumed that the upper pH limit for the protection of fish is 9 (according to Directive 78/659/EEC), this limit would be attained with 0.56, 0.86, 4.51 and 8.30 mg/l KOH in, respectively, distilled water, soft water (20 mg/l HCO$_3^-$), normal hardness water (106 mg/l HCO$_3^-$) and high hardness water (195 mg/l HCO$_3^-$). To assess the potential environmental effect of a KOH discharge, the pH change of the receiving water should be calculated or measured and compared with the natural variation of the receiving water. Based on this comparison it should be assessed which amount and pH of the effluent are acceptable under specific local situations.

Some few uses of KOH could result in an emission of KOH leading to a local increase of the pH in the aquatic environment. However, the pH of effluents is normally measured very frequently and can be adapted easily and therefore a significant increase of the pH of the receiving water is not expected. Generally the change in pH of the receiving water should stay within a tolerated range of the pH at the effluent side and for this reason no adverse effects on the aquatic environment are expected due to production or use of KOH, if emissions of waste water are controlled by appropriate pH limits and/or dilutions in relation to the natural pH and buffering capacity of the receiving water.

Aquatic potassium emissions originating from uses of KOH are probably small compared to other sources. It is clear that an environmental hazard assessment of potassium should not only evaluate all natural and anthropogenic sources of potassium but should also evaluate all other ecotoxicity studies (e.g. with potassium salts), which is beyond the scope of this report.

**Exposure**

Estimated world-wide demand of potassium hydroxide was higher than 1 million tons expressed as KOH 100% in 1994. The global demand is expected to grow with 4.0% per year. KOH is a white and deliquescent solid with a low vapour pressure. It is a strong alkaline substance that dissociates completely in water to potassium and hydroxyl ions.

KOH is commercialised as a solid or as solutions with varying concentrations. It has many industrial uses; less than 2% is for wide dispersive use. It is used in paint and varnish removers, drain cleaners, degreasing agents and dairy pipeline cleaners.

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**NATURE OF FURTHER WORK RECOMMENDED**

**Environment and Human Health:** no further work is recommended if sufficient control measures are in place to avoid significant human and environmental impact, including prevention of accidental exposure.

Due to the corrosivity of the substance, no further studies are required under SIDS programme.
SIDS Initial Assessment Report

1 IDENTIFY

1.1 Identification of the Substance

- CAS Number: 1310-58-3
- IUPAC Name: Potassium hydroxide
- Molecular Formula: KOH
- Structural Formula: KOH
- Molecular Weight: 56.11
- Synonyms: Caustic potash, Potassium hydrate, Potassium hydroxide, dry solid, flake, bead or granule, Potassium lye

1.2 Purity/Impurities/Additives

- Purity (industrial gr.): ca. 91% (ICCA-HPV KOH Consortium, 2001)
- Impurities: water ca. 8%, sodium hydroxide < 1%, potassium carbonate < 0.7%, potassium chloride < 0.01%, other impurities < 0.001%

1.3 Physico-Chemical properties

<table>
<thead>
<tr>
<th>Property</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Substance type</td>
<td>Inorganic</td>
<td></td>
</tr>
<tr>
<td>Physical state</td>
<td>White and deliquescent solid</td>
<td></td>
</tr>
<tr>
<td>Melting point</td>
<td>406°C</td>
<td>(Lide, 1995)</td>
</tr>
<tr>
<td>Boiling point</td>
<td>1327°C</td>
<td>(Lide, 1995)</td>
</tr>
<tr>
<td>Vapour pressure</td>
<td>1.3 hPa at 719°C</td>
<td>(Lide, 1995)</td>
</tr>
<tr>
<td>Water solubility</td>
<td>1100 g/l at 25°C</td>
<td>(Commission of the European Communities, 1993)</td>
</tr>
</tbody>
</table>

Strong alkaline substance that dissociates completely in water to K⁺ and OH⁻ ions. Strongly exothermic dissolution/dissociation in water (vigorous reaction when KOH is added to water).
2 GENERAL INFORMATION ON EXPOSURE

2.1 Production Volumes and Use Pattern

Estimated worldwide demand of potassium hydroxide is higher than 1 million tonnes expressed as KOH 100 % in 1994 (Tessenderlo, 2000). The global demand for KOH has been growing by 4.0 % per year (Brown, 1999).

KOH is produced via electrolysis of potassium chloride in some 25 production sites worldwide, which can be done via the mercury, membrane or diaphragm process. The mercury process is the preferred one for the high purity product obtained. KOH is commercialised as a solid (flakes, beads, granules) or as solutions with varying concentrations. The most important industrial concentration is 50 % (Ullmann, 1998).

KOH has mainly industrial uses. On a global level the main uses are (Occidental Chem. Corp., 2000):

- Potassium carbonate: 26 %
- Chemical manufacturing: 16 %
- Potassium chemicals: 12 %
- Fertilizers: 11 %
- Phosphates: 9 %
- Detergents: 8 %
- Agricultural chemicals: 7 %
- Alkaline batteries: 6 %
- All other: 5 %.

So, more than 95% of the KOH production is for non dispersive use, and is consumed by the industry, mainly by large enterprises. KOH is used in these applications as an intermediate and do not leave the plant where it is used. In these applications, KOH is consumed in a reaction and is no more present in the product that goes to the market. KOH is still present in the alkaline batteries, but here this substance is strictly confined in the battery screening and doesn’t come in contact with the consumer.

Less than 5% of the KOH production is for wide dispersive use and enters in the composition of consumer products (eventually to be consumed in small enterprises like garages or farms): paint and varnish removers (ICCA-HPV KOH Consortium, 2001), drain cleaners (Howell, 1991; Leape et al., 1971), degreasing agents (Swanson et al., 1995) and dairy pipeline cleaners (Edmonson, 1987).

Potential human exposure to KOH is thus for less than 5% of its total production. Without taking into account recycling of the alkaline batteries (these represent 6 % of the total production), which is normally done in many countries, the exposure to the environment is for less than 11% of the total production. Losses through production, through processes that use the compound and through disposal of the compound are minimized. The pH of effluents is controlled and these must be neutralized, this being normally linked to the agreement given to the plant by the authorities.

2.2 Environmental Exposure and Fate

The high water solubility and low vapour pressure indicate that KOH will be found predominantly in the aquatic environment. KOH is present in the environment as potassium and hydroxyl ions, which implies that it will not adsorb on particulate matter or surfaces and will not accumulate in living tissues. It is obvious that both potassium and hydroxyl ions have a wide natural occurrence (UNEP, 1995).
Atmospheric emissions as KOH aerosols should be rapidly neutralized by carbon dioxide, as occurs with NaOH (Cooper et al., 1979) or other acids and the salts (e.g. potassium carbonate) will be washed out by rain. For this reason potential atmospheric emissions of KOH are considered of no concern. Significant emissions to the terrestrial environment are not expected during normal handling and use of KOH. Small terrestrial emissions will be neutralized by the buffer capacity of the soil. For this reason the environmental assessment can be limited to the aquatic compartment.

Because KOH does occur in the environment as K⁺ and OH⁻ a separate environmental assessment of both the potassium and the hydroxyl ion is needed.

**Measured concentrations in ecosystems**

The concentration of hydroxyl ions in the environment has been determined very extensively via pH measurements. The pH is a very important parameter of aquatic ecosystems and it is a standard parameter of water quality monitoring programs. The most important freshwater aquatic ecosystems of the world revealed average annual pH values between 6.5 and 8.3 but lower and higher values have been measured in other aquatic ecosystems. The global median value was 7.7 and no strong variability was observed at individual stations. In aquatic ecosystems with dissolved organic acids a pH of less than 4.0 has been measured, while in waters with a high chlorophyll content the bicarbonate assimilation can result in pH values of higher than 9.0 at midday. The pH of an aquatic ecosystem is mainly determined by geochemical, hydrological and/or biological processes (UNEP, 1995).

Also potassium has been measured extensively in aquatic ecosystems. For example, UNEP (1995) reported the concentration for a total number of 75 rivers in North America, South-America, Asia, Africa, Europe and Oceania. The 10th - percentile, mean and 90th - percentile were 0.8 , 3.2 and 6.0 mg/l, respectively. The potassium concentration of topsoils is 0.2-3.3% (Chemical Economics Handbook, 1999), and that of seawater is 380 mg/l (Tait, 1980).

It should also be remembered that KCl is to be added to reconstituted fresh waters for toxicity tests with fishes, macro invertebrates and amphibians, from 0.5 to 16.0 mg KCl/l (0.3 to 8.4 mg K⁺/l), depending on the hardness of the water to be produced, from respectively very soft to very hard (ASTM, 1996).

**KOH addition and buffer capacity**

An addition of KOH to an aquatic ecosystem may increase the pH depending on the buffer capacity of the receiving water. In general the buffer capacity is regulated by the equilibrium between CO₂, HCO₃⁻ and CO₃²⁻:

\[
\text{CO}_2 + \text{H}_2\text{O} \leftrightarrow \text{HCO}_3^- + \text{H}^+ \quad (pK_{a1} = 6.35)
\]

\[
\text{HCO}_3^- \leftrightarrow \text{CO}_3^{2-} + \text{H}^+ \quad (pK_{a2} = 10.33)
\]

If the pH is between 7 and 9 then the bicarbonate ion is the most important species responsible for the buffer capacity of aquatic ecosystems. 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 were 20, 106 and 195 mg/l, respectively. To underline the importance of the buffer capacity, a table is included with the concentration of KOH needed to increase the pH to a value of 9.0 at different bicarbonate concentrations. It should be realised that the final pH could be slightly lower than 9.0 because at initial pH values below 8 there is some CO₂ available to buffer the pH.
Concentration of KOH needed to increase pH to a value of 9.0:

<table>
<thead>
<tr>
<th>Buffer capacity</th>
<th>Concentration KOH (mg/l)</th>
<th>Concentration K (mg/l)</th>
<th>Concentration KOH (mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 mg/l HCO₃⁻ (distilled water)</td>
<td>0.56</td>
<td>0.39</td>
<td>0.010</td>
</tr>
<tr>
<td>20 mg/l HCO₃⁻ (10th percentile 77 rivers)</td>
<td>0.86</td>
<td>0.60</td>
<td>0.015</td>
</tr>
<tr>
<td>106 mg/l HCO₃⁻ (mean value of 77 rivers)</td>
<td>4.51</td>
<td>3.14</td>
<td>0.080</td>
</tr>
<tr>
<td>195 mg/l HCO₃⁻ (90th percentile 77 rivers)</td>
<td>8.30</td>
<td>5.78</td>
<td>0.148</td>
</tr>
</tbody>
</table>

Use of KOH and anthropogenic exposure

The use of KOH could potentially result in an aquatic emission of KOH and it could locally increase the potassium and the pH in the aquatic environment.

The pH of effluents is normally measured very frequently, can be adapted easily and these effluents are commonly neutralised, and therefore a significant increase of the pH of the receiving water is not expected. However, in regions where the pH of effluents is not regulated, a KOH discharge might cause a significant increase in the pH of the receiving water.

Specific analytical data or other reliable data about the use of KOH and the related emissions of potassium are not available. However, it should be realised that emissions originating from the use of KOH are probably small compared to other sources of potassium. With a global potash production of 29 millions tons K₂O, or 24 millions tons K in 1989, and an estimated KOH production that not exceeded 0.800 million tons, or 0.560 million tons K (Ullmann, 1998), and taking into account that no more than 11% of the K from KOH could be discharged to the environment (see section 2 : uses), not more than 0.060 million tons K coming directly from KOH reaches the environment, or 0.25% of the total anthropogenic potassium. It is thus clear that an environmental hazard assessment of potassium should evaluate all the natural and anthropogenic sources.

2.3 Human Exposure

KOH has many industrial and some domestic uses and it is available to the general public in some few consumer products/formulations since a long time. For this reason accidental or intentional acute exposures (suicide) have been described in the medical literature. Some medical case reports and reviews of medical treatment methods of KOH burns are available. Of the great amount of publications related to caustic injury (concerning mostly NaOH), we selected those where KOH is specifically cited. Exposure via the environment is negligible, as the product is diluted in rivers and neutralized by the bicarbonate buffering capacity or acids.

Ingestion

Children may be accidentally exposed to commercial cleaning products containing KOH or NaOH, including, in the case of farm children, to high concentration (8 to 25%) dairy pipeline cleaners. In his study, Edmonson (1987) reported that 43 children were admitted from 1973 to 1983 to four rural Wisconsin (USA) hospitals, after ingestion of caustic products. Farm products constituted 23% of all products and 43% of all drain/pipe cleaners ingested.
In a retrospective clinical study with 168 children after alkaline substance ingestion, 9 children (5.3%) developed gastric outlet obstruction. After an appropriate treatment, all patients were without complaints (Ciftci, 1999).

In a study of liquid caustic ingestion on 31 patients in India, 3 ingested potassium hydroxide (9.7%) and 28 ingested sodium hydroxide (90.3%). The degree and extent of burns with respect to type of alkalis were not noticeably different. There was a poor correlation between the presence or absence or severity, of oropharyngeal burns on one hand and the presence or absence or severity of lesions in the UGIT (uro-gastro-intestinal tract) on the other hand. Eight patients (25.8%) who showed involvement of the UGIT had a normal oropharynx. Two patients had perforation on admission and in 3 more it occurred on the 9th, 11th and 14th days after ingestion. All patients suffered oesophageal injury, 29 (93.5%) gastric injury and 8 (25.8%) duodenum injury (Zargar, 1992).

Battery ingestions in the UK and the USA have been reviewed (Thompson et al., 1990). The potential for corrosive alkali injury from batteries is in fact dependent on their electrical properties, by the progressive electrolysis of the battery casing, in the area of the seal. Moreover, it is thought that burns to the oesophagus could be due to the low-voltage DC producing electrolysis with an increase of pH.

The hypothesis has been presented that burns produced by ingested button batteries with residual EMF (electromotive force), could be due, not by its KOH release, but by a hydrolysis process producing locally a high pH (Rauber, 1990).

Potassium hydroxide is a food additive, listed as E525 in Annex 1 of Directive 95/2/EU. This means that KOH is a general food additive to be used following the “quantum satis” principle: as much as necessary according to GMP (European Union, 1995).

The concentration of potassium is limited under the EU Directive on Drinking Water Quality 80/778/EEC. The potassium guide level is 10 mg/l and the maximum allowable concentration is 12 mg/l (European Economic Community, 1980). The taste threshold of KOH in water is reported to be 1 to 50 mg/l (Mc Kee et al., 1963).

The normal daily dietary intake of potassium in humans is approximately 2 - 4 g (FASEB, 1979), typically 2 – 6 g in the US diet (Saxena, 1989). The daily dietary intake of K is recommended to be approximately 2.4 g or more because this is associated with a reduced risk of stroke-related mortality (Burgess et al., 1999).

Skin and eyes

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

In an analysis of 2100 accidents and dangerous occurrences, which occurred in the UK chemical industry between January 1982 and March 1985, 32 involved caustic soda/potash (Robinson, 1987).

Inhalation

Dust formation is unlikely because of the hygroscopic properties. Furthermore KOH has a negligible vapour pressure and is rapidly neutralized in air by carbon dioxide and therefore dust and vapour exposure are not expected. For production and major uses of KOH, aerosols/mists do normally not occur. Different to NaOH, specific uses with a possibility of formation of aerosols are not known for KOH applications. In every case, it should be realised that aerosols of KOH are not stable. They are rapidly transformed due to an uptake of carbon dioxide from the atmosphere.
resulting in the formation of potassium bicarbonate and potassium carbonate. Cooper et al. (1979) reported that the transformation of respirable NaOH aerosols into carbonate aerosols could occur in seconds. Analytical measurements, to determine KOH concentrations in the air of working places during production and use, seem to be unavailable.

TLV (US), TWA (US), peak limitation (Australia), STEL 10 minutes (UK) and many other workplace exposure limit values are 2 mg / m³ (Hazard and Safety Data Bank, Potassium Hydroxide, 2000).

An epidemiological study about potash mining workers failed to correlate the exposure to potash to a number of diseases evaluated, including lung cancer (Waxweiler et al., 1973).
3 HUMAN HEALTH HAZARDS

3.1 Effects on Human Health

KOH has been commercialised for a long time and there exists 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 interesting to discuss the animal data and human data together where appropriate.

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

Potassium hydroxide causes direct local effects on the skin, eyes and gastrointestinal tract after direct exposure to sufficient concentrations. Inhalation of KOH dust does normally not occur. Most of the production of KOH is in the liquid form (mostly 50% solution). Potential dust formation during drying, pellets production and packing occurs normally in closed systems, which is essential for the product quality, to avoid moisture and carbon dioxide absorption. Moreover, as KOH is a deliquescent solid, the potential for dust formation is low. Nevertheless, direct manipulation of dry KOH should be done with an approved respirator (like European Standard EN-149), when necessary. Consumer uses of dry KOH are not known. If KOH aerosols/mists occur, they will cause direct local effects on respiratory tract (see also 2.2). According to Dick and Ahlers (1998), the irritant effects are reported as coughing, wheezing, conjunctivitis, tearing, irritation and alterations in general well being.

The local effects of potassium hydroxide are the result of the OH⁻ ion (pH change) rather than an effect of the potassium ion.

For the hazard assessment of potassium hydroxide, the use of data from sodium hydroxide, potassium chloride or potassium carbonate (“analogs or surrogates”) is useful. Indeed, NaOH is a strong base, a hydroxide that has a very similar effect regarding pH or corrosiveness. KCl and K₂CO₃ are soluble potassium salts, which have a very similar effect regarding the potassium toxicity of KOH.

3.1.1 Toxicokinetics, Metabolism and Distribution

As potassium hydroxide is dissociated in the body fluids, its systemic toxicity must be discussed for its constituting potassium and hydroxyl ions separately.

Potassium is an essential constituent of the body fluids. It is the principal intracellular cation (approximately 5.7 g/l) and it is necessary for the nervous and muscular cells function, as well as for several metabolic activities, among others the synthesis of proteins. Separation of the K⁺ and Na⁺ cations across the plasmatic membrane is assured by the ATP consuming K⁺/Na⁺ pumps, and allows membrane potentials necessary for nerve and muscle function (Marieb, 1992). Its normal plasmatic concentration is approximately 140 – 200 mg/l. The minimum toxicity level is under 250 mg/l. Between 250 and 310 mg/l, a moderate toxicity is observed, giving lassitude, fatigue and weakness. Severe toxic doses of over 310 mg/l lead to neuromuscular paralysis and, at 390-470 mg/l death from cardiac arrest, due to intraventricular conduction defects by depolarisation of cardiac muscle and subsequent increase in cardiac muscle excitability. Hyperkalemia can be produced by ingestion of 80 – 100 mg K⁺/kg bw, but cardiac effects predominate only after IV administration (Hazard and Safety Data Bank, Potassium Chloride, 2000).

Regulation of K⁺ concentration in blood is assured principally by renal excretion and reabsorption. The least increase of the K⁺ concentration in the extra cellular liquid stimulates strongly aldosterone liberation, which increases K⁺ excretion. This feedback regulation constitutes an efficient auto-
The systemic toxicity of hydroxyl ions confounds with an elevated blood pH. The normal pH of blood is 7.35 – 7.45 and the absolute range of pH is 7.0 – 7.8. Alkalosis causes hyperactivity of the central nervous system with, above pH 7.8, tetanus, extreme excitability, convulsions and respiratory stop. Blood pH is regulated by three distinct mechanisms. An immediate mechanism is the buffering capacity of bicarbonate (approximately 1.5 g/l), proteins and in a lesser extent phosphate. A short-term mechanism is the respiration compensation. Alkalosis will be decreased by a slow and superficial respiration, a low CO₂ expiration and an accumulation of HCO₃⁻ ions (higher than 1.7 g/l). A long-term mechanism is the renal compensation. Alkalosis will be decreased by an increase of the excretion of HCO₃⁻ ions (Marieb, 1992).

Interesting observations are also that alkalosis promotes renal excretion of K⁺, and that, for preventing hyperkalemia, extra cellular potassium is taken up by cells in exchange for hydrogen ions (Saxena, 1989). In other words, these compensating effects of K⁺ and OH⁻ would attenuate the systemic effect of KOH.

A systemic (non-acute) oral intoxication by KOH is not expected. Regarding the potassium toxicity, the LD50 value in rats of KCl, 3.020 g/kg bw/day (Boyd and Shanas, 1961) is much higher than that of KOH, in the range of 0.273 - 0.365 – 1.230 g KOH/kg bw/day (Bruce, 1987; Johnson et al., 1975; Smyth et al., 1969). This demonstrates that the acute toxicity of KOH is probably due to the corrosivity caused by the OH⁻ ion (pH value), and less to systemic toxic effects of the K⁺ ion. Under non-irritating conditions, the potassium doses are much lower than those used in acute toxicity studies, and therefore not relevant from the point of view of the systemic toxicity of potassium. Furthermore, the uptake of potassium, via exposure to potassium hydroxide, is much less than the oral uptake with therapeutic doses of KCl for treating potassium deficiency, of up to 10 g/day (USP XVII, 1970). Moreover, the oral uptake of potassium from food, from natural origin or from food additives, is likely to be also much higher. Potassium hydroxide, potassium chloride and other potassium salts are food additives listed in Annex 1 of Directive 95/2.EU, and these have no ADI specified (European Union, 1995).

In the case of an oral non-acute uptake of KOH in concentrations not irritant to the mucosa (at least not irritant to the skin or eyes, lower than 0.5%), the chance the hydroxyl ions to pass in the blood and to cause an alkalosis depends on the level of gastric HCl secretion, which is variable from one person to another and with the filling grade, and which is increased by an increasing pH. The eventual alkalosis due to the (non-acute) hydroxyl ion absorption is quickly regulated by the above-described mechanisms.

Conclusion

Both K⁺ and OH⁻ ions are normal constituents of the body fluids. K⁺ plays an essential role in the human physiology but starts to be toxic at levels exceeding 200 – 250 mg/l. Its concentration in the blood is regulated principally by renal excretion/reabsorption and controlled by an efficient feedback auto-regulation system. An excessive pH of the blood is prevented by the bicarbonate buffer system, respiration and renal compensation mechanisms.

3.1.2 Acute Toxicity

Studies in Animals

Several acute oral toxicity determinations have been done for KOH, with a good concordance for the two most recent.
In a first study, the LD50 (intubation) with male rats was 365 mg/kg (Johnson et al., 1975). Haemorrhaging of the stomach and intestine and adhesions of abdominal organs (stomach, pancreas, spleen, liver and small intestine) were seen following administration of both lethal and sub-lethal doses. Surviving animals showed evidence of hyper excitability, followed by apathy and weakness throughout the 14-day post-exposure period. Other clinical signs were increased respiration rate, ruffled fur, eye closing and bloody nasal exudate. All deaths occurred within 72 hours of dosing.

In another study, an LD50 (oral gavage) with male rats was 273 mg/kg (Bruce, 1987). Using the “Up and Down” method, without 14 day post-exposure period, the LD50 was 388 mg/kg.

A third study revealed an LD50 (gavage) with male rats of 1230 mg/kg (Smyth et al., 1969). The higher value found, compared to the first study, could be that non-fasted and younger rats were used (Johnson et al., 1975).

Neither acute inhalation nor dermal toxicity studies have been located.

Conclusion

KOH has a moderate acute oral toxicity, which is essentially due to its corrosivity. The observed systemic effects could be regarded as secondary effects.

3.1.3  Irritation and Corrosion

3.1.3.1  Oral human data

The only real effects of KOH ingestion are gastrointestinal burns. The mechanism of injury is one of liquefactive necrosis. Thrombosis of local blood vessels contributes to tissue damage. Tran mural necrosis can occur with frightening rapidity and injury often extrudes through the oesophagus to involve adjacent mediastinal and peritoneal structures. When alkali enters the stomach, there may be some neutralization by gastric acid, which can limit the injury to this organ. Perforation of the stomach can occur with peritonitis and caustic injury to the contiguous organs including the colon, pancreas, liver and spleen. If sufficient quantities of alkali pass through the pylorus, there may be substantial duodenal damage including perforation. Lye constitutes a greater danger than solid granules, which tend to adhere on contact to mucous membranes without travelling further. The severity of damage depends on concentration of the agent, but also on the quantity swallowed. Aspiration of the alkali into the airway can result in live-threatening injuries to the larynx, the tracheobronchial passages, and the lungs. There are three phases of injury and healing to the oesophagus. The acute phases, from about day 1 to 4, is that of liquefactive necrosis. During the sub acute phase, from day 4 to 14, there is sloughing of the necrotic area; the oesophageal wall appears thinnest and most vulnerable. About day 15 begins the cicatrisation phase with eventual oesophageal strictures resulting from collagen contraction. Reepithelialisation is complete by 4 weeks to 3 months. There is also a strong association between lye stricture of the oesophagus and oesophageal squamous cell carcinoma, with a long latent period of eventually several decades (Spechler, 1992).

The following publications concern caustic injury where KOH is specifically cited.

A young woman suffered severe burns in the oesophagus after consuming a soft drink contaminated by an industrial cleaning agent used to clean the non-disposable bottles. The “lemonade” had a pH of 13.3, a total alkalinity of 1.75 N, a sodium content of 1.75 mole/l and a potassium content of 1.15 mole/l (Stefanidou et al., 1997).

A woman who ingested 20 g of KOH in aqueous solution suffered glossopharyngalgia and oral pharyngeal burns (Cello et al., 1980).
The simultaneous admission of 9 youths in a medical centre following their ingestion of concentrated KOH, mistaken for wine, resulted in the following observations. Three patients with second-degree oral burns required no surgery. Six patients required laparotomy with gastrostomy and/or chimney feeding jejunostomy, one required immediate oesophagostrectomy, and 3 required immediate total or subtotal gastrectomy. There were no deaths. Three patients have required oesophageal replacement and 3 others have required repeated dilatations. At 2-year follow-up, all 9 maintain their nutritional status orally, and can phonate (Meredith and Thompson, 1987).

The fatal complications from an alkaline battery foreign body (containing potassium hydroxide 45%) in the oesophagus of a 2.5 year old male, resulting in corrosive burns of the oesophagus, necrosis, perforation, communication between the oesophagus and the trachea and subsequent death, is described (Blatnik et al., 1977).

### 3.1.3.2 Skin data

#### Animal data

In a classical rabbit Draize test with gauze covering, application of 0.5 ml of KOH 5% during 4 hours gave a PDII (primary dermal irritation indices) result of 4.8 (moderately irritating). A 10% solution was severely irritating (Nixon et al., 1990). With 19 mm diameter Hill Top Chamber pad covering during 1 or 4 hours and 0.2 ml applied, the 5 and 10% KOH solutions were qualified as severely irritating.

A rabbit Draize test with gauze covering and application of 0.1 ml during 24 hours qualified a 5% KOH solution as mildly irritating on intact skin and highly irritating on abraded skin (Johnson et al., 1975).

A 10% KOH solution was qualified as corrosive on both intact and abraded skin as the result of a Draize occlusive test on rabbits with 4 hours exposure to 0.5 ml of the solution. The results with guinea pigs were similar (Nixon et al., 1975).

In a Draize rabbit test (reliability 3) with gauze covering and application of 0.5 ml of KOH solutions during 4 hours, the 1% solution was not corrosive, whereas the 2% solution was corrosive. There was no post-exposure assessment of the lesion (Vernot et al., 1977).

In “in vitro” tests (reliability 3) with reconstructed human skin cultures Skin²ZS1301 and EpiDerm, and MTT vital dye metabolism, a 10% KOH solution was scored as corrosive (Perkins et al., 1996).

In a comparison study (reliability 3) of 4 “in vitro” methods, TER, Corrositex, Episkin and Skin²ZK1350, the 4 methods discriminated KOH 10% as highly corrosive or corrosive, while only the 3 first methods discriminated KOH 5% as highly corrosive or corrosive (Fentem et al., 1998).

#### Human data

The mechanism of injury by alkali skin burns is by saponification of fat, which causes fatty tissue to lose its function with increased damage due to heat reaction; extraction of considerable water from cells due to the hygroscopic nature of alkali; and dissolution of proteins, permitting deeper penetration of OH- ions and further chemical reactions (Milner et al., 1996).

A 2-year old male was found to have a third degree (full thickness) burn on his right thigh due to exposure to the contents of leaking alkaline batteries (Winek et al., 1999).

A 4-year old boy who had a button battery lodged in his nose for approx. 24 hrs had local tissue corrosion, with a small perforation, caused presumably by the 25% KOH electrolyte (Fernando, 1987).
Treatment of 32 children suffering of Molluscum contagiosum (a viral skin infection) with a topical 10% KOH aqueous solution, twice daily, during a period of 30 days, resulted in clearance of all lesions. The only side effects observed in 12 children were: severe stinging, transitory hypo pigmentation, persistent hypo and hyper pigmentation, hypertrophic scar and secondary infection (Romiti et al., 1999).

The skin corrosivity of KOH is extensively documented, and there is no need for further animal tests.

3.1.3.3 Eye data

Animal data

Several concentrations of KOH were tested by a Draize test on rabbits by instilling 0.1 ml, rinsing after 5 minutes or 24 hours of exposure and examining with the aid of fluorescein at 1, 24, 48 and 72 hours, 7 days, and eventually 14-21 days. The results were as follows:

5% / 5 min.: extremely irritant and corrosive.
1% / 5 min.: irritant; 1% / 24 hr.: irritant.
0.5% / 24 hr.: marginal.
0.1% / 24 hr.: negative (Johnson, 1975).

In an “in vitro” test (reliability 3) with human corneal endothelial cell cultures and cell viability quantification by a $^{51}$Cr-release assay, the ED50 result (50% maximal toxicity) of 0.073% was said to correlate with “severe irritating” in the Draize test (Douglas and Spilman, 1983).

Human data

Eye damage by alkali burns is most significant around pH 11 - 11.5. Alkali penetrates quickly, saponifies plasma membranes, denatures collagen, and causes vascular thromboses in the conjunctiva, the episclera, and even the anterior uvea. The sequelae of corneal burns include scarring and opacification of the cornea with resultant loss of visual acuity, corneal neovascularization, ulcer formation, and perforation. Other sequelae of untreated or very severe alkali burns include epithelial erosions, secondary glaucoma, progressive cicatrisation which occludes the ducts of main and accessory lachrymal glands and causes destruction of conjunctival goblet cells so as to cause dry eyes, cicatricial entropion, and trichiasis (Milner et al., 1996).

Conclusion

KOH is a corrosive substance at concentrations of about 2% and higher. Between about 0.5% and 2.0%, it is irritating. Case reports on human accidents or intentional exposure confirm that the risk posed by KOH for human health originates from its corrosive properties.

3.1.4 Sensitisation

An intracutaneous skin sensitisation test was performed with guinea pigs (Landsteiner and Jacobs method), by using 0.1 ml of 0.1% KOH induction injections and a 0.1 ml challenge injection. No allergic skin reactions were observed after 24, 48 and 72 hours following the challenge dose (Johnson, 1975).

Potassium hydroxide has been used extensively for many decades by the industry and by consumers. However, skin sensitisation has never been described secondary to skin irritation or
burns. Both the potassium and the hydroxide are ions, which are naturally, present in the body and for this reason it is very unlikely that they could cause skin sensitisation.

Conclusion

Based on the data available and on the above, it may be concluded that potassium hydroxide is not an allergen in humans.

3.1.5 Repeated Dose Toxicity

No studies were identified regarding the repeated dose toxicity of KOH in animals.

KOH in aqueous solutions is completely dissociated into K⁺ and OH⁻ ions. Due to the neutralization of OH⁻ by gastric HCl and the quick and efficient blood pH regulation mechanisms (buffer capacity of extra cellular body fluids, respiratory and renal compensation mechanisms), an alkalosis due to the OH⁻ ions after KOH oral dosage in non-irritating conditions is prevented.

Therefore, a possible systemic toxicity of KOH would be related to the K⁺ ion and studies with potassium salts in which the anion does not contribute significantly to toxicity could be used for KOH as well.

Groups of 50 male rats were fed KCl in the diet at levels of 0.25, 1 and 4% KCl (calculated as 110, 450 and 1820 mg/kg bw/day respectively) for 2 years. Only chronic gastritis and ulcer were found more in the experimental group than in the control group (an irritant effect). The survival rates were 64, 58 and 84% in, respectively, the 0.25, 1 and 4% KCl groups, and 48% in the control group (Imai et al., 1986). A NOEL > 1820 mg KCl/kg bw/day (> 955 mg K⁺/kg bw/day) can be suggested.

Giving a 2.5% KCl solution to rats (calculated as 5250 mg KCl/kg bw/day or 2751 mg K⁺/kg bw/day) as the sole source of fluid during 15 weeks induced an increase in the weight of the kidneys, a decrease in the weight of the hearth and adrenal glomeral zone hypertrophy, effects that were reversible on cessation of KCl flooding (Bacchus, 1951).

Two groups totalling 43 normotensive women received 80 mmol KCl/day (calculated as 108 mg KCl/kg bw/day) or placebo in a 2-period crossover study for the first 4-week treatment period. The treatments were reversed during the second 4-week period. Blood pressure, heart rate, urinary volume, electrolytes and creatinine were measured weekly during a screening period and the experimental periods (Barden et al., 1986). No adverse effects were seen and a NOEL > 108 mg KCl/kg bw/day (56.5 mg K⁺/kg bw/day) can be suggested.

Two groups totalling 32 hypertensive black women received 65 mmol KCl/day (calculated as 88 mg KCl/kg bw/day) or placebo in a 2-period crossover study for the first 6-week period. The treatments were reversed during the second 6-week period. Blood pressure, urinary electrolytes and creatinine were measured weekly. Plasma Na⁺ and K⁺ and serum albumin, Ca²⁺ and Mg²⁺ were measured at the 6th week (Matlou et al., 1996). A significant reduction in systolic and diastolic blood pressure was observed, but no adverse effects were seen and a NOEL > 88 mg KCl/kg bw/day (46 mg K⁺/kg bw/day) can be suggested.

Conclusion

Repeated dose studies with KOH are not available. Based on the results with KCl, it can be concluded that a chronic oral exposure to KOH in non-irritating concentrations/conditions would result in a low effect level of toxicity due to the K⁺ ion, similar to that of KCl, that is well documented.
3.1.6 Mutagenicity

Studies in Animals

In vitro Studies

The results of an Ames assay study with Salmonella typhimurium TA 97 and TA 102, with and without metabolic activation and up to 1 mg KOH/plate, were negative (Fujita et al., 1992).

The clastogenic activity of KOH 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 KOH concentrations of 0, 8 and 12 mM, which corresponded with initial pH values of 7.3, 9.8 and 10.4, respectively. In the presence of an activation system (S9 mix) a clastogenic activity was found with 12 mM KOH (pH 10.4). According to the authors, this genotoxic effect is due to the high non-physiological pH (same effect with NaOH at 16 mM, pH 10.8). At such high pH values, the clastogenic activity of S9 is increased, or new clastogens are induced by breakdown of the S9. Incubations at non-physiological pH might give false-positive responses, and this possibility must be considered in the evaluation of such results (Morita, 1989). Non-physiological environments can produce genotoxic effects in cultured mammalian cells (Brusick, 1986, 1987). The pH causality is further proven by the fact that the normal intracellular concentration of K+ is of the order of 10 times higher: 145 mM in human cells (Marieb, 1992). A high non-physiological pH is not relevant in human cells.

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 (reliability 3) of these tests NaOH was classified as non-genotoxic.

KCl has been classified as non-genotoxic in a bacterial reverse mutation assay with S. typhimurium TA 100, TA 1535, TA 1537 and TA 9, at 0, 100, 333, 1000, 3333 and 10000 µg/plate, with and without metabolic activation (Mortelmans et al., 1986). In a DNA damage and repair assay (SOS Chromotest Institut Pasteur) with E. coli PQ 37, at 1-100000 nM/ml, without metabolic activation, KCl was negative (Olivier and Marzin, 1987).

Two publications (Myhr and Caspary, 1988; Mitchell et al., 1988) report the genotoxic effect of KCl in a mammalian cell gene mutation assay with mouse lymphoma cell L5178Y, TK+/− heterozygote, at 0-5000 µg/ml, with and without metabolic activation (OECD guideline 476). The result was positive only at high KCl concentration with metabolic activation. This has been attributed by the authors to the changed physical environment of the cells (increased osmotic pressure; K+ effects on sequestering of Mg2+ ions required for chromatin integrity), rather than to a direct genotoxic effect. Several authors confirm this type of non-specific genotoxic effect (Brusick, 1986; Seeberg, 1988).

K2CO3 has been classified as non-genotoxic in an Ames test with Salmonella typhimurium strains TA 92, TA 94, TA 98, TA 100, TA 1535 and TA 1537, at up to 10 mg/plate, with metabolic activation (liver S-9 mix of Fischer rats) and pre-treatment with polychlorinated biphenyls (Ishidate et al., 1984).

Two publications (Myhr and Caspary, 1988; Mitchell et al., 1988) report the genotoxic effect of KCl in a mammalian cell gene mutation assay with mouse lymphoma cell L5178Y, TK+/− heterozygote, at 0-5000 µg/ml, with and without metabolic activation (OECD guideline 476). The result was positive only at high KCl concentration with metabolic activation. This has been attributed by the authors to the changed physical environment of the cells (increased osmotic pressure; K+ effects on sequestering of Mg2+ ions required for chromatin integrity), rather than to a direct genotoxic effect. Several authors confirm this type of non-specific genotoxic effect (Brusick, 1986; Seeberg, 1988).

K2CO3 has been classified as non-genotoxic in a cytogenetic assay with Chinese hamster fibroblasts (CHL cells) at up to 1 mg/ml, without metabolic activation (Ishidate et al., 1984).

In vivo Studies

No studies were identified regarding the “in vivo” genotoxicity.

NaOH was tested in 2 “in vivo” studies.
In a mouse bone micronucleus test (reliability 3) using 15 mM NaOH at a dose of 10 mg/kg bw, the test compound was administered as a single IP dose to treatment groups (5 males and 5 females) at 30, 48 and 72 h. No significant increase of nuclei was observed (Aaron et al., 1989).

Mouse oocytes were used (reliability 3) 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. No evidence of non-disjunction was found up to the 40 weeks tested.

**Conclusion**

With the sum of information of the studies on KOH, NaOH, KCl and K₂CO₃, apart from the artefacts due to high pH with KOH or NaOH, and high osmotic pressure with KCl, there is no evidence for a mutagenic activity. For the reasons explained in section 3.1.1, K⁺ and OH⁻ are not expected to be systemically available in the body over the normal limits, under non-irritating conditions. A genotoxic effect is also not very likely because both the K⁺ and OH⁻ ions are naturally present in the human body, K⁺ is present in the cells at much higher concentration than outside (30 – 40x) and an high concentration of OH⁻ is incompatible with cell life.

### 3.1.7 Carcinogenicity

Valid carcinogenicity studies with animals are not available for potassium hydroxide.

An old long-term study (reliability 3) of 25-46 weeks, consisting of painting 3-6% KOH solutions on mouse skin, has been performed (Narat, 1925). The results were ca. 15% occurrence of cancer at the application site. As discussed by Ingram and Grasso (1991), such a production of skin cancer is due to a non-genotoxic mechanism secondary to repeated application and prolonged inflammation, by indirect hyperplasia as a consequence of severe skin damage. Any kind of prolonged irritation possibly would have produced the same result. HCl solution painting produced also cancer in mice. Moreover, such an exposure causing repeated skin damage and increased cell proliferation to repair the chronic injury, is not relevant for man.

There is also a strong association between lye stricture of the oesophagus and oesophageal squamous cell carcinoma, with a long latent period of eventually several decades (Spechler, 1992).

**Conclusion**

There is no evidence KOH to be carcinogenic in exposure situations that are relevant for man.

### 3.1.8 Toxicity for Reproduction

No studies were identified regarding the reproduction/developmental toxicity.

For the reasons listed under section 3.1.5, KOH as such will not be available to the reproductive organs and the developing embryo or foetus. Possible effects are related to K⁺ ions of KOH, and studies conducted with KCl and K₂CO₃ could therefore be used for reproductive toxicity of KOH as well.

A one generation study with female mice and rats exists for KCl. Doses of 2.35 – 235 mg/kg bw/day in mice and 3.1 – 310 mg/kg bw/day in rats were administered to groups of 21-24 animals by single daily oral intubation. Body weights were recorded during 17 days for mice, with a post exposure period of 2 days and during 20 days for rats, with a post exposure period of 5 days. No significant effects were observed on mice and rat’s survival and reproductive organs, or on offspring survival, weight, sex ratio and congenital defects. The NOEL values for parental/maternal and F1 offspring could be established as > 235 mg KCl/kg bw/day (corresponding to > 123 mg
Reproduction toxicity studies with female mice and rats exist also for K₂CO₃. Doses of 2.9 – 290 mg/kg bw/day in mice and 1.8 – 180 mg/kg bw/day in rats were administered to 22 - 25 mice/group and 23 – 25 rats/group. The exposure period was from day 6 to 15 of gestation (10 days). No significant effects were observed on nidation or on maternal or foetal survival, or for abnormalities. The teratogenic NOEL values could be established as > 290 mg K₂CO₃/kg bw/day (corresponding to > 164 mg K⁺/kg bw/day) for mice, and > 180 mg K₂CO₃/kg bw/day (corresponding to > 102 mg K⁺/kg bw/day) for rats (NTIS, 1975).

Conclusion

Studies to the reproduction of KOH are not available. Based on the results of corresponding potassium salts like KCl and K₂CO₃, effects in non-irritating doses/concentrations to reproduction or development are not expected for KOH. The calculated NOAEL for the potassium ion is approx. 164 mg/kg bw.

3.2 Initial Assessment for Human Health

KOH is a corrosive substance. Solutions with concentrations of about 2% and higher are corrosive, while concentrations of about 0.5 to about 2.0 % are irritating. KOH is not considered to be a skin sensitiser. KOH has been used widely and for a long time, and no human cases of skin sensitisation have been reported. No studies are available for repeated dose toxicity, in vivo genotoxicity, toxicity to reproduction and development.

The reported oral rat LD50 values are 365, 273 and 1230 mg/kg bw/day. Based on the data with other potassium compounds, it could be concluded that potassium has no or a negligible contribution to the toxicity at lethal dose levels of KOH. With KCl, the NOEL in rats for repeated dose toxicity is > 1820 mg/kg bw/day, and > 88-108 mg/kg bw/day in women, and for reproduction/developmental toxicity, > 235 and > 310 mg/kg bw/day for, respectively, mice and rats. With K₂CO₃, the teratogenic NOEL values could be established as > 290 mg/kg bw/day for mice, and > 180 mg/kg bw/day for rats.

Under normal handling and use conditions (non-irritating) neither the concentration of potassium in the blood nor the pH of the blood will be increased above normal limits and therefore absorbed KOH is not expected to cause systemically toxic levels in the blood. The renal excretion of K⁺ can be elevated and the OH⁻ ion is neutralized by the bicarbonate buffer system in the blood. It can also be stated that the substance will neither reach the foetus nor reach male and female reproductive organs in effective toxic concentrations. Therefore, no risk for reproductive toxicity is to be expected. Two in vitro genetic toxicity tests (Ames test and mammalian cell gene mutation assay) indicated no evidence for a mutagenic activity. No mutagenic activity was found for the related substances NaOH (both in vitro and in vivo) nor KCl and K₂CO₃ (in vitro).

Accidental and intentional exposures to irritating or corrosive solutions of KOH have been described in the literature. The KOH quantity, its concentration, the exposure duration and the area of the tissue involved would mainly influence the severity of the effects. In general, accidental skin and eye exposures are less frequently reported than ingestion in the medical literature, are maybe less severe and therefore probably the number of admissions to hospital is less. Most of the ingestion accidents seem to be related with children and seem to occur at home. For adults, ingestion of KOH can be accidental but also intentional ingestion (suicide) does occur.

Dust formation is unlikely because of the hygroscopic properties. Furthermore KOH has a negligible vapour pressure and is rapidly neutralized in air by carbon dioxide and therefore dust and vapour exposure are not expected. There are no applications known where there is formation of
aerosols. If they occur, aerosols probably are neutralized by carbon dioxide before they reach the lungs.
4 HAZARDS TO THE ENVIRONMENT

4.1 Aquatic Effects

At concentrations reported in publications and study reports, the toxicity has been assumed to be due to hydroxide only, because at these effect concentrations the concentration of potassium is too low to explain the effects. However, it should be realised that the results of toxicity tests with KOH depend on the buffer capacity of the test medium. In a highly buffered test medium the hydroxyl ion will be neutralized and the observed toxicity will be low, while in a poorly buffered test medium the pH will increase rapidly and therefore the observed toxicity will be relatively high (see also section 2.1). Besides the direct effects (pH change) KOH could also have indirect effects. The pH change could influence the speciation of other chemicals and therefore increase and/or decrease the toxicity e.g. NH$_3$ is more toxic than NH$_4^+$. The available toxicity tests with KOH will be discussed below.

For the hazard assessment of potassium hydroxide, the use of data from sodium hydroxide or potassium chloride (“analogs or surrogates”) is useful. Indeed, NaOH is a strong base, a hydroxide that has a very similar effect regarding pH or corrosiveness. KCl is a soluble potassium salt, which has a very similar effect regarding the potassium toxicity of KOH.

Effects on fish

A 96-hour test (reliability 3) with Gambusia affinis (mosquito fish) revealed an LC50 of 80 mg/l. At 56 mg/l, no effects on the fish were observed. At the highest concentration tested (100 mg/l) the pH was increased up to 10.3 (Wallen et al, 1957). In an old 24-hour test (reliability 3) with brook trout, Salvelinus fontinalis, a minimum lethal dose of 50 mg/l is given (Belding, 1927). A 24-hour LC50 value of 165 mg/l has been published (reliability 3) for guppy, Poecilia reticulata (Yarzhombek, 1991).

A short review (reliability 4) of the toxicity of KOH for fish is given by McKee et al. (1963). Concentrations between 29 and 140 mg/l of KOH seemed to have killed fish but on the other hand a concentration of 28 mg/l have not harmed fish.

A TLm 24 hr for Gambusia affinis (mosquito fish) of 80 ppm in fresh water is given in the U.S. (U.S. Coast Guard, Department of Transportation, 1984).

Several publications with NaOH (reliability 3 or 4) give an indication on the OH$^-$ effect.

A 96-hour test with Gambusia affinis (mosquito fish) revealed an LC50 of 125 mg/l. At 84 mg/l, no effects on the fish were observed. The pH was 9 at 100 mg/l (Wallen et al, 1957). A 24-hour toxicity test (Jensen, 1978) with Carassius auratus (goldfish) revealed a NaOH LC50 of 160 mg/l (equivalent KOH: 224 mg/l). At 100 mg/l (equivalent KOH: 140 mg/l), pH 9.8, no mortality was observed. A toxicity test (Juhnke et al., 1978) with a related species, Leuciscus idus melanotus, revealed an LC50 (48-hour) of 189 mg/l (equivalent KOH: 265 mg/l). Solutions of NaOH in pond water started to be toxic to the fry of Lucioperca lucioperca L (pike perch) at NaOH concentrations of 35 mg/l (equivalent KOH: 49 mg/l) and higher (Stangenberg, 1975).

The chronic effect of NaOH on guppies (Lebistes reticulatus) has been tested (Rustamova, 1977; reliability 3) at 25, 50, 75 and 100 mg/l (equivalent KOH: 35, 70, 105 and 140 mg/l respectively). An adverse effect on the survival rate, growth and fecundity, as well as on the quality of the progeny was found. Upon prolonged exposure concentrations of 25-100 mg/l (equivalent KOH: 35-140 mg/l) produced significant changes in the biology of the fish.

Regarding the KCl toxicity, in a study carried out under national guidelines and with a reliable procedure description, a 96h – LC50 value of 880 mg/l (±15) with Pimephales promelas has been published (Mount et al., 1997).
Effects on invertebrates

In an abstract (reliability 4), Matisoff et al., (1991) report that KOH concentrations exceeding 10 ppm induce complete mortality of zebra mussels (*Dreissenia polymorpha*).

Toxicity tests with invertebrates like *Daphnia magna* have not been found. Publications exist in the case of NaOH and KCl. These data are useful for the evaluation of KOH, as their effects are probably caused by the hydroxyl ion and the potassium ion, respectively.

The LC50 after 48 hours of exposure was 40 mg NaOH/l (equivalent KOH: 56 mg/l) for the freshwater cladoceran *Ceriodaphnia dubia* (Warne et al., 1999).

A short review (reliability 4) of the toxicity of NaOH for invertebrates is given by McKee et al. (1963). The toxicity threshold concentration of NaOH for *Daphnia magna* was reported to range from 40 to 240 mg/l (equivalents KOH: 56 and 337 mg/l). Concentrations of 125 to 1000 mg/l were reported to be lethal to insect larvae (equivalents KOH: 175 and 1403 mg/l).

The following studies (reliability 3) give additional information.

The lethal concentration of NaOH to the vector snails *Biomphalaria a. alexandrina*, *Bulinus truncatus* and *Lymnaea caillaudi* (Gohar et al., 1961) was 150, 150 and 450 mg/l (equivalents KOH: 210, 210 and 637 mg/l respectively). The LC50 after 48 hours of exposure was 33-100 mg/l (equivalents KOH: 46 and 140 mg/l) for the marine polychaete *Ophryotrocha diadema* (Parker, 1984).

Regarding the KCl toxicity, in a study carried out under national guidelines and with a reliable procedure description, 48h – LC50 values of 660 mg/l (±7.5) with *Daphnia magna* and 630 mg/l (±14) with *Cerodaphnia dubia* have been published (Mount et al., 1997).

Effects in aquatic plants / algae

Toxicity tests with algae or aquatic plants have not been found (nor for sodium hydroxide).

A KCl study reports a 120 h – EC50 (growth rate) value of 1337 mg/l with the alga *Nitscheria linearis* (Patrick et al., 1968).

Effects on micro organisms

The inhibition of the bioluminescence of the bacterium *Photobacterium phosphoreum* by KOH has been measured with the Microtox system (Bulich et al., 1990). The 15 minutes-EC50 was 22 mg/l. The test medium was 2 % NaCl which means that the medium was not buffered.

The effect of NaOH on motility of the protozoan *Tetrahymena thermophila* was studied by microscope (Silverman et al., 1987). When 1% NaOH was diluted 62 times the motility was higher than 90 % of control cell motility (highest tolerated dose, HTD). This would be equal to a NaOH concentration of 161 mg/l (equivalent KOH: 226 mg/l).

The following table summarizes the aquatic organisms toxicity.

<table>
<thead>
<tr>
<th>Substance</th>
<th>Toxicity type</th>
<th>Value (mg/l)</th>
<th>Organism</th>
<th>Species</th>
<th>Reference</th>
<th>Reliability</th>
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<tbody>
<tr>
<td>KOH</td>
<td>NOEC</td>
<td>28</td>
<td>Fish acute</td>
<td>56 (96 h)</td>
<td>Wallen (1957)</td>
<td>3a</td>
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<tr>
<td></td>
<td>Lethal</td>
<td>50 (24 h)</td>
<td>Fish acute</td>
<td>50 (24 h)</td>
<td>Belding (1927)</td>
<td>3a</td>
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<tr>
<td></td>
<td></td>
<td>29-140</td>
<td>Fish acute</td>
<td>50 (24 h)</td>
<td>McKee (1963)</td>
<td>4b</td>
</tr>
<tr>
<td></td>
<td>LC50/EC50</td>
<td>80 (96 h)</td>
<td>Fish acute</td>
<td>80 (96 h)</td>
<td>Wallen (1957)</td>
<td>3a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>165 (24 h)</td>
<td>Fish acute</td>
<td>165 (24 h)</td>
<td>Yarzhombek (1991)</td>
<td>3a</td>
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<tr>
<td></td>
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<td>22 (15 min.)</td>
<td>Micro-organisme</td>
<td>22 (15 min.)</td>
<td>Bulich (1990)</td>
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<tr>
<td></td>
<td></td>
<td>80 (24 h)</td>
<td>Fish acute</td>
<td>80 (24 h)</td>
<td>US Coast Guard</td>
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OECD SIDS | POTASSIUM HYDROXIDE

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<th>Invertebrate acute</th>
<th>Dreissena polymorpha</th>
<th>Matisoff (1991)</th>
<th>4a</th>
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<tbody>
<tr>
<td>NaOH NOEC</td>
<td>84</td>
<td>(96 h)</td>
<td>Fish acute</td>
<td>Gambusia affinis</td>
<td>Wallen (1957)</td>
<td>3a</td>
</tr>
<tr>
<td>Toxic</td>
<td>161</td>
<td>(2 min.)</td>
<td>Micro-organisme</td>
<td>Tetrahymena thermophila</td>
<td>Silverman (1987)</td>
<td>3a</td>
</tr>
<tr>
<td></td>
<td>35</td>
<td>(24 h)</td>
<td>Fish acute</td>
<td>Lucioperca lucioperca L.</td>
<td>Stangenberg (1975)</td>
<td>3a</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>100</td>
<td>Fish chronic</td>
<td>Lepistes reticulatus</td>
<td>Rustamova (1977)</td>
<td>3a</td>
</tr>
<tr>
<td></td>
<td>40-240</td>
<td>Invertebrate acute</td>
<td>Daphnia magna</td>
<td>Mc Kee (1963)</td>
<td>4b</td>
<td></td>
</tr>
<tr>
<td>Lethal</td>
<td>125</td>
<td>1000</td>
<td>Invertebrate acute</td>
<td>Insect larvae</td>
<td>Mc Kee (1963)</td>
<td>4b</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td></td>
<td>Invertebrate acute</td>
<td>Biomphalaria a. alexandrina</td>
<td>Mc Kee (1963)</td>
<td>3a</td>
</tr>
<tr>
<td></td>
<td>150</td>
<td></td>
<td>Invertebrate acute</td>
<td>Bulinus truncatus</td>
<td>Gohar (1961)</td>
<td>3a</td>
</tr>
<tr>
<td></td>
<td>450</td>
<td></td>
<td>Invertebrate acute</td>
<td>Lymnaea caullaindi</td>
<td>Gohar (1961)</td>
<td>3a</td>
</tr>
<tr>
<td>LC50/EC50</td>
<td>160</td>
<td>(24 h)</td>
<td>Fish acute</td>
<td>Carassius auratus</td>
<td>Jensen (1978)</td>
<td>3a</td>
</tr>
<tr>
<td></td>
<td>189</td>
<td>(48 h)</td>
<td>Fish acute</td>
<td>Leuciscus idus melanotus</td>
<td>Juhnke (1978)</td>
<td>4e</td>
</tr>
<tr>
<td></td>
<td>125</td>
<td>(96 h)</td>
<td>Fish acute</td>
<td>Gambusia affinis</td>
<td>Wallen (1957)</td>
<td>3a</td>
</tr>
<tr>
<td></td>
<td>40</td>
<td>(48 h)</td>
<td>Invertebrate acute</td>
<td>Cerodaphnia dubia</td>
<td>Warner (1999)</td>
<td>2d</td>
</tr>
<tr>
<td></td>
<td>33-100</td>
<td>(48 h)</td>
<td>Invertebrate acute</td>
<td>Ophyatrocho diadema</td>
<td>Parker (1984)</td>
<td>3a</td>
</tr>
<tr>
<td>KCl LC50/EC50</td>
<td>880</td>
<td>(96 h)</td>
<td>Fish acute</td>
<td>Pimephales promelas</td>
<td>Mount (1997)</td>
<td>1b</td>
</tr>
<tr>
<td></td>
<td>660</td>
<td>(48 h)</td>
<td>Invertebrate acute</td>
<td>Daphnia magna</td>
<td>Mount (1997)</td>
<td>1b</td>
</tr>
<tr>
<td></td>
<td>630</td>
<td>(48 h)</td>
<td>Invertebrate acute</td>
<td>Cerodaphnia dubia</td>
<td>Mount (1997)</td>
<td>1b</td>
</tr>
<tr>
<td></td>
<td>1337</td>
<td>(120h)</td>
<td>Algae</td>
<td>Nitscheria linearis</td>
<td>Patrick (1968)</td>
<td>2c</td>
</tr>
</tbody>
</table>

**Conclusion**

The aquatic life toxicity values for KOH/NaOH fluctuate, probably because of the different buffer capacities of the test systems. The toxicity values for KCl are much higher, indicating that the toxicity of KOH/NaOH is probably to be attributed to a pH effect.

**PNEC derivation**

With a few exceptions, the available toxicity studies with KOH or NaOH were not conducted according to current standard guidelines. In many cases pH, buffer capacity and/or medium composition were not discussed in the publications, although this is essential information for toxicity tests with KOH. The observed variation in LC50 values can be explained by the buffer capacity of the testing water, which was used.

Although high quality acute ecotoxicity tests and chronic ecotoxicity tests with KOH or NaOH are not available there is no need for additional testing with KOH. Aquatic ecosystems are characterized by an alkalinity/pH and the organisms of the ecosystem are adapted to these specific natural conditions. Based on the natural alkalinity of waters, organisms will have different optimum pH conditions, ranging from poorly buffered waters with a pH of 6 to very hard waters with pH values up to 9. 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.

Based on the available data it is not considered useful to derive a PNEC or a PNEC<sub>added</sub> for KOH because:

- The natural pH of aquatic ecosystems can vary significantly between aquatic ecosystems.
  Also the sensitivity of the aquatic ecosystems to a change of the pH can vary significantly between aquatic ecosystems.

- The change in pH due to an anthropogenic KOH addition is influenced significantly by the buffer capacity of the receiving water.
Although a PNEC or a PNEC\textsubscript{added} was not calculated for KOH there is a need to assess the environmental effect of a KOH (alkaline) discharge. Based on the pH and buffer capacity of effluent and receiving water and the dilution factor of the effluent, the pH of the receiving water after the discharge can be calculated. Of course the pH change can be measured also very easily via a laboratory experiment or by conducting field measurements. The change in pH should be compared with the natural variation in pH of the receiving water and based on this comparison it should be assessed if the pH change is acceptable.

To illustrate the procedure and to get an idea about the order of magnitude for acceptable anthropogenic additions, the acceptable KOH addition will be calculated for 2 representative cases. According to Directive 78/659/EEC (European Economic Community, 1978), the pH of surface water for the protection of fish should be between 6 and 9. In section 2.1 it was mentioned that the 10\textsuperscript{th} -percentile and the 90\textsuperscript{th} -percentile of the bicarbonate concentrations of 77 rivers of the world were 20 and 195 mg/l, respectively. If it is assumed that only bicarbonate is responsible for the buffer capacity of the ecosystem and if it is assumed that an increase of the pH to a value of 9.0 would be the maximum accepted value, then the acceptable anthropogenic addition of KOH would be 0.86 and 8.30 mg/l for bicarbonate concentrations of 20 and 195 mg/l, respectively. This gives an indication of the acceptable amount of KOH that could be discharged to an aquatic ecosystem if there was an emission of a pure KOH solution.

KOH concentrations of 0.86 and 8.30 mg/l are equivalent with K concentrations of 0.60 and 5.78 mg/l, respectively. These concentrations are close to measured concentrations of potassium in rivers of the world (see section 2.1), and stay far behind levels, which are economic (there is approximately a factor 100 between these values and the values of KCl toxicity; see section 4.1 Aquatic effects). Furthermore concentrations of 0.60 – 5.78 mg/l do not exceed the levels to be added to reconstituted fresh water, which is used for toxicity testing. According to ASTM (1996) the potassium concentration of reconstituted fresh water is 0.3 – 8.4 mg/l depending on the hardness of the water to be prepared. This confirms that the hazard of KOH (not neutralized) is caused by the hydroxyl ion (pH effect).

4.2 Terrestrial Effects

The EC50 after 90 days of KOH for \textit{Enchytraeus sp.} (>95\% \textit{Cognetia sphagnetorum}), soil dwelling organisms, has been evaluated by Heungens (1984) as 850 mg / l litter (artificial soil). The effect of KOH on the enchytraeid population was not correlated to pH, but to an increase of conductivity.

It is remembered that potassium is one of the three major nutrients in terrestrial plants and is important for the osmotic and ionic regulation, control of water, protein synthesis and photosynthesis (Marschner, 1995).

Conclusion

The only study in this compartment indicates a low level of toxicity, probably due to the buffering capacity of the soil used. In general, the results of terrestrial toxicity tests will depend strongly on the buffer capacity of the soil and can probably be predicted based on the buffer capacity of the soil.

4.3 Other Environmental Effects

No other environmental effects are expected.

4.4 Initial Assessment for the Environment

The hazard of KOH for the environment is caused by the hydroxyl ion (pH effect). For this reason the effect of KOH on the organisms depends on the buffer capacity of the aquatic or terrestrial
ecosystem. Also the variation in acute toxicity for aquatic organisms can be explained for a significant extent by the variation in buffer capacity of the test medium. The LC50 value of acute fish toxicity was in the order of 80 mg/l. It was 880 mg/l for KCl and ranged between 125-189 mg/l for NaOH. The LC50 values of acute invertebrate toxicity for KCl was 660 mg/l (Daphnia magna) and 630 mg/l (Ceriodaphnia dubia), and for NaOH 40 mg/l (Ceriodaphnia dubia). The EC50 algae value (Nitscheria linearis) was 1337 mg/l for KCl.

Because of the buffer capacity, the pH and the fluctuation of the pH are very specific for a certain ecosystem, it was not considered useful to derive a PNEC. If it is assumed that the upper pH limit for the protection of fish is 9 (according to Directive 78/659/EEC), this limit would be attained with 0.56, 0.86, 4.51 and 8.30 mg/l KOH in, respectively, distilled water, soft water (20 mg/l HCO₃⁻), normal hardness water (106 mg/l HCO₃⁻) and high hardness water (195 mg/l HCO₃⁻). To assess the potential environmental effect of a KOH discharge, the pH change of the receiving water should be calculated or measured and compared with the natural variation of the receiving water. Based on this comparison it should be assessed which amount and pH of the effluent are acceptable under specific local situations.

Some few uses of KOH could result in an emission of KOH leading to a local increase of the pH in the aquatic environment. However, the pH of effluents is normally measured very frequently and can be adapted easily and therefore a significant increase of the pH of the receiving water is not expected. Generally the change in pH of the receiving water should stay within a tolerated range of the pH at the effluent site, and for these reason adverse effects on the aquatic environment are not expected due to production or use of KOH, if emissions of waste water are controlled by appropriate pH limits and/or dilutions in relation to the natural pH and buffering capacity of the receiving water.

Aquatic potassium emissions originating from uses of KOH are probably small compared to other sources. It is clear that an environmental hazard assessment of potassium should not only evaluate all natural and anthropogenic sources of potassium but should also evaluate all other ecotoxicity studies (e.g. with potassium salts), which is beyond the scope of this report.
5 RECOMMENDATIONS

Environment

The risk that KOH poses for the environment is essentially restricted to a pH increase of the aquatic compartment, which is dependent on the hardness of the waters. This effect is well known, as are the ways to control it. Therefore, no further testing is required.

Human health

The risk that KOH poses for human health originates from its corrosive properties essentially. These are sufficiently documented in order to take the indicated precautions of use. Therefore no further testing is required.
6 REFERENCES

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OECD SIDS  


NTIS (National Technical Information Service), (1975), Report PB 242 522, Food and Drug Research Laboratories, Inc.


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Romiti, R. et al., (1999), Pediatric Dermatology, 16(3), 228-231.


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Wallen, I.E. et al., (1957), Sewage Ind. Wastes, 29(6), 695-711.

OECD SIDS POTASSIUM HYDROXIDE


Yarzhombek, A.A. et al., Vopr. Ikhtiol., 31(3), 496-502 (as reported in EPA-ECOTOX).

I U C L I D D a t a S e t

Existing Chemical ID: 1310-58-3
CAS No. 1310-58-3
EINECS Name potassium hydroxide
EINECS No. 215-181-3
TSCA Name Potassium hydroxide (K(OH))
Molecular Formula HKO

Producer Related Part
Company: Tessenderlo Chemie
Creation date: 10-JAN-2001

Substance Related Part
Company: Tessenderlo Chemie
Creation date: 10-JAN-2001

Printing date: 30-JAN-2002
Revision date: 
Date of last Update: 30-JAN-2002

Number of Pages: 74

Chapter (profile): Chapter: 1, 2, 3, 4, 5, 7
Reliability (profile): Reliability: without reliability, 1, 2, 3, 4
1.0.1 OECD and Company Information

Type: cooperating company
Name: Aragonesas SA
Country: Spain
07-MAY-2001

Type: cooperating company
Name: Asahi Glass Co
Country: Japan
07-MAY-2001

Type: cooperating company
Name: BASF AG
Country: Germany
07-MAY-2001

Type: cooperating company
Name: Daiso Co
Country: Japan
07-MAY-2001

Type: cooperating company
Name: Degussa AG
Country: Germany
26-JUL-2001

Type: cooperating company
Name: Enichem Spa
Country: Italy
07-MAY-2001

Type: cooperating company
Name: Ineos Ltd
Country: United Kingdom
07-MAY-2001

Type: cooperating company
Name: SPC Harbonnières SA
Country: France
07-MAY-2001

Type: lead organisation
Name: Tessenderlo Chemie NV
Partner: Date: 10-JAN-2001
Street: Stationstraat
Town: 3980 Tessenderlo
Country: Belgium
Phone: 32 13 61 22 11
Telefax: 32 13 67 23 43
1.0.2 Location of Production Site

1.0.3 Identity of Recipients

Name of recip.: Guy Muyldermans
Street: Stationstraat
Town: B-3980 Tessenderlo
Country: Belgium
Phone: 32 13 61 26 46
Telefax: 32 13 67 23 43

1.1 General Substance Information

Substance type: inorganic
Physical status: solid
Purity: ca. 91 % w/w

1.1.0 Details on Template

1.1.1 Spectra

1.2 Synonyms

Caustic Potash
Potassium hydrate
Potassium hydroxide, dry solid, flake, bead or granule
Potassium lye

1.3 Impurities

CAS-No: 7732-18-5
EINECS-No: 231-791-2
EINECS-Name: water
Contents: ca. 8 % w/w

27-MAR-2001 (35)
# 1. GENERAL INFORMATION

<table>
<thead>
<tr>
<th>ID: 1310-58-3</th>
<th>DATE: 30-JAN-2002</th>
</tr>
</thead>
</table>

**OECD SIDS POTASSIUM HYDROXIDE**

## 1.4 Additives

**CAS-No:**

**EINECS-No:**

**EINECS-Name:**

**Remark:**

<table>
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</table>

**No additives**

## 1.5 Quantity

**Production during the last 12 months:** yes

**Import during the last 12 months:** yes

**Quantity produced:**

<table>
<thead>
<tr>
<th>more than 1 000 000 tonnes in 1994</th>
</tr>
</thead>
</table>

---

**Contents:**

**< 1 % w/w**

**CAS-No:**

**EINECS-No:**

**EINECS-Name:** sodium hydroxide

**27-MAR-2001**

---

**Contents:**

**< .7 % w/w**

**CAS-No:**

**EINECS-No:**

**EINECS-Name:** potassium carbonate

**12-SEP-2001**

---

**Contents:**

**< .01 % w/w**

**CAS-No:**

**EINECS-No:**

**EINECS-Name:** potassium chloride

**Remark:**

For the major process in use, the mercury cells process.

**27-MAR-2001**

---

**Contents:**

**< .0001 % w/w**

**CAS-No:**

**EINECS-No:**

**EINECS-Name:** potassium bromide

**11-APR-2001**

---

**Contents:**

**< .001 % w/w**

**CAS-No:**

**EINECS-No:**

**EINECS-Name:** potassium chlorate

**27-MAR-2001**

---

**Contents:**

**< .0005 % w/w**

**CAS-No:**

**EINECS-No:**

**EINECS-Name:** potassium bromate

**11-APR-2001**

---

**Contents:**

**< .0001 % w/w**

**CAS-No:**

**EINECS-No:**

**EINECS-Name:** mercury

**Remark:**

Mercury cells process (< 0.00005%).

**26-JUL-2001**

---

**CAS-No:**

**EINECS-No:**

**EINECS-Name:**

**Remark:**

No additives

**17-JAN-2001**

---

**CAS-No:**

**EINECS-No:**

**EINECS-Name:**

**Remark:**

Mercury cells process (< 0.00005%).

**26-JUL-2001**

---

**CAS-No:**

**EINECS-No:**

**EINECS-Name:**

**Remark:**

No additives

**17-JAN-2001**

---

**CAS-No:**

**EINECS-No:**

**EINECS-Name:**

**Remark:**

Mercury cells process (< 0.00005%).

**26-JUL-2001**

---

**CAS-No:**

**EINECS-No:**

**EINECS-Name:**

**Remark:**

No additives

**17-JAN-2001**

---

**CAS-No:**

**EINECS-No:**

**EINECS-Name:**

**Remark:**

Mercury cells process (< 0.00005%).

**26-JUL-2001**

---

1.6.1 Labelling

Labelling: as in Directive 67/548/EEC
Symbols: C
Specific limits: yes
R-Phrases:
(22) Harmful if swallowed
(35) Causes severe burns
S-Phrases:
(1/2) Keep locked up and out of reach of children
(26) In case of contact with eyes, rinse immediately with plenty of water and seek medical advice
(36/37/39) Wear suitable protective clothing, gloves and eye/face protection
(45) In case of accident or if you feel unwell, seek medical advice immediately (show the label where possible)
Specific limits:
C>=25%: C; R22-35
5%<=C<25%: C; R35
2%<=C<5%: C; R34
0.5%<=C<2%: Xi; R36/38.

UN Hazard Class: 8
UN n° 1813 (dry material)
UN n° 1814 (aqueous solutions)
UN Packing Group: II.

1.6.2 Classification

Classification: as in Directive 67/548/EEC
Class of danger: corrosive
R-Phrases: (35) Causes severe burns
Remark: Xn; R22
C; R35

1.7 Use Pattern

Type: type
Category: Non dispersive use
Result: > 95% of the global market.
28-MAR-2001

Type: type
Category: Wide dispersive use
Result: < 5% of the global market.
OECD SIDS

POTASSIUM HYDROXIDE

1. GENERAL INFORMATION

DATE: 30-JAN-2002
ID: 1310-58-3

28-MAR-2001

Type: industrial
Category: Agricultural industry
Remark: Non dispersive use.
Result: 18% of the global market.

28-MAR-2001

Type: industrial
Category: Basic industry: basic chemicals
Remark: Non dispersive use.
Result: 47% of the global market.

28-MAR-2001

Type: industrial
Category: Chemical industry: used in synthesis
Remark: Non dispersive use.
Result: 24% of the global market.

28-MAR-2001

Type: industrial
Category: Electrical/electronic engineering industry
Remark: Non dispersive use.
Result: 6% of the global market.

28-MAR-2001

Type: industrial
Category: other
Remark: Non dispersive use.
Result: All others: < 5% of the global market.

28-MAR-2001

Type: use
Category: other
Remark: Non dispersive use.
Result: Potassium carbonate: 26% of the global market.

28-MAR-2001

Type: use
Category: other
Remark: Non dispersive use.
Result: Chemical manufacturing: 16% of the global market.

11-APR-2001

Type: use
Category: other
Remark: Non dispersive use.
Result: Potassium chemicals: 12% of the global market.

28-MAR-2001

Type: use
Category: Fertilizers
Remark: Non dispersive use.
Result: 11% of the global market.
Type: use
Category: other
Remark: Non dispersive use.
Result: Phosphates: 9% of the global market.
28-MAR-2001

Type: use
Category: other
Remark: Non dispersive use.
Result: Detergents: 8% of the global market.
11-APR-2001

Type: use
Category: other
Remark: Non dispersive use.
Result: Agricultural chemicals: 7% of the global market.
28-MAR-2001

Type: use
Category: other
Remark: Non dispersive use.
Result: Alkaline batteries: 6% of the global market.
11-APR-2001

Type: use
Category: other
Remark: Non dispersive use.
Result: All other: < 5% of the global market.
28-MAR-2001

Type: industrial
Category: Paints, lacquers and varnishes industry
Remark: Wide dispersive use.
Result: < 5% of the global market.
28-MAR-2001

Type: industrial
Category: Personal and domestic use
Remark: Wide dispersive use.
Result: < 5% of the global market.
28-MAR-2001

Type: use
Category: Cleaning/washing agents and disinfectants
Remark: Wide dispersive use.
Result: Paint and varnish removers: < 5% of the global market.
28-MAR-2001

Type: use
Category: Cleaning/washing agents and disinfectants
Remark: Wide dispersive use.
Result: Drain cleaners: < 5% of the global market.
11-APR-2001
### 1. General Information

**Type:** use

**Category:** Cleaning/washing agents and disinfectants

**Remark:** Wide dispersive use.

**Result:** Degreasing agents: < 5% of the global market.

28-MAR-2001

**Type:** use

**Category:** Cleaning/washing agents and disinfectants

**Remark:** Wide dispersive use.

**Result:** Dairy pipeline cleaners: < 5% of the global market.

11-APR-2001

**Type:** use

**Category:** Cleaning/washing agents and disinfectants

**Remark:** Wide dispersive use.

**Result:** Liquid drain cleaners containing 4 - 4.5% KOH.

11-APR-2001

**Type:** use

**Category:** Cleaning/washing agents and disinfectants

**Remark:** Wide dispersive use.

**Result:** Liquid drain cleaners containing 25 - 36.5% KOH and a solid drain cleaner with 71% KOH.

11-APR-2001

**Type:** use

**Category:** Cleaning/washing agents and disinfectants

**Remark:** Wide dispersive use.

**Result:** Liquid degreasing agents containing 0.75 - 1.50% KOH.

11-APR-2001

**Type:** use

**Category:** Cleaning/washing agents and disinfectants

**Remark:** Wide dispersive use.

**Result:** Liquid dairy pipeline cleaners containing 8 - 25% KOH.

11-APR-2001

**Type:** use

**Category:** Cleaning/washing agents and disinfectants

**Remark:** Non dispersive use.

**Result:** A battery electrolyte containing 25% KOH.

11-APR-2001

**Type:** use

**Category:** Cleaning/washing agents and disinfectants

**Remark:** Non dispersive use.

**Result:** Battery electrolytes containing 7 - 12% KOH.

11-APR-2001

### 1.7.1 Technology Production/Use

**Type:** Production

**Remark:** KOH (together with chlorine) is manufactured by KCl electrolysis. The 3 existing electrolysis processes, mercury, diaphragm and membrane processes, can be applied. With the diaphragm process, an high chloride content cannot be avoided. With the membrane process, the chloride content is lower. The mercury process is still preferred, for the high product purity obtained.
1.8 Occupational Exposure Limit Values

Type of limit: TLV (US)
Limit value: 2 mg/m³
Reliability: (1) valid without restriction
02-JAN-2002

Limit value:
Short term expos.
Limit value: 2 mg/m³
Schedule: 10 hour(s)
Reliability: (1) valid without restriction
02-JAN-2002

Type of limit: other: peak limitation Australia, 1990.
Limit value:
Short term expos.
Limit value: 2 mg/m³
Reliability: (1) valid without restriction
02-JAN-2002

Limit value:
Short term expos.
Limit value: 2 mg/m³
Schedule: 10 minute(s)
Reliability: (1) valid without restriction
02-JAN-2002

Type of limit: other: Dk limit value ambient air.
Limit value: 0.005 mg/m³
Reliability: (1) valid without restriction
02-JAN-2002

1.9 Source of Exposure

Memo: K in rivers, worldwide, <5 mg/l; >12 mg/l downstream of potash mines. Monitoring values worldwide. pH annual average mostly 6.5-8.3; global median value 7.7. No strong variability at individual stations.

Remark: Potassium statistics for 75 world rivers:
mean: 3.22 mg/l
10th percentile: 0.80 mg/l
90th percentile: 5.96 mg/l
range: 41.90 mg/l

Potassium statistics for 11 rivers from North America:
mean: 2.35 mg/l
10th percentile: 0.80 mg/l
90th percentile: 5.00 mg/l
range: 7.20 mg/l
Potassium statistics for 5 rivers from South America:
mean: 1.58 mg/l
10th percentile: 0.74 mg/l
90th percentile: 2.30 mg/l
range: 1.80 mg/l

Potassium statistics for 25 rivers from Asia:
mean: 2.17 mg/l
10th percentile: 0.84 mg/l
90th percentile: 3.94 mg/l
range: 6.80 mg/l

Potassium statistics for 7 rivers from Africa:
mean: 1.84 mg/l
10th percentile: 1.16 mg/l
90th percentile: 2.66 mg/l
range: 2.10 mg/l

Potassium statistics for 21 rivers from Europe:
mean: 5.88 mg/l
10th percentile: 0.80 mg/l
90th percentile: 7.00 mg/l
range: 41.40 mg/l

Potassium statistics for 6 rivers from Oceania:
mean: 2.87 mg/l
10th percentile: 0.40 mg/l
90th percentile: 5.00 mg/l
range: 5.60 mg/l

Levels of concern for water quality from anthropogenic exposure, predominantly fertilizers, are never reached (12 mg/l WHO guideline for drinking water), except downstream from potash mines (Weser: 42.0 mg/l; Elbe: 26.1 mg/l).

Acidity/alkalinity is measured as pH which is a key parameter in water quality. pH is closely linked to biological productivity in aquatic systems. With dissolved organic acids from soil leaching (Amazonia), a pH of less than 4.0 has been measured. In waters with a high chlorophyll content, the bicarbonate assimilation can result in pH values exceeding quiet commonly 8.5 and even 9.0 at midday (Loire).

Attached doc.:
UNEPbicarbK010718.xls
UNEP Bicarbonate and Potassium in Rivers Worldwide.

Reliability:
(1) valid without restriction
27-JUL-2001

Memo:
K coming directly from KOH reaching the environment: <0.25% of the total anthropogenic potassium.

Remark:
With a global potash production of 29.341.000 tons K2O, or 24.357.391 tons K in 1989, and an estimated KOH production that not exceeded 800.000 tons that year, or 557.476 tons K (Ullmann, 1998), and taking into account that no more than 11% of the K from KOH could be discharged to the environment (see section 1.7 Use Pattern: 5% wide dispersive use + 6% in alkaline batteries; the rest is transformed in industrial uses; Occidental Chem. Corp., 2000), not more than 61.322 tons K coming directly from KOH reaches the environment, or 0.25% of the total anthropogenic potassium.

Reliability:
(1) valid without restriction
27-JUL-2001
1.0.1 Recommendations/Precautionary Measures

1.0.2 Emergency Measures

1.11 Packaging

1.12 Possib. of Rendering Subst. Harmless

1.13 Statements Concerning Waste

1.14.1 Water Pollution

1.14.2 Major Accident Hazards

1.14.3 Air Pollution

1.15 Additional Remarks

Memo: Electrodialysis can be used for the purification and recovery of spent KOH containing streams from alkaline storage batteries recycling.

17-JAN-2001 (97)

Memo: Solvent extraction with Kelex 100 (Witco) can be used for the treatment of spent electrolyte solutions generated in nickel-cadmium battery manufacturing.

17-JAN-2001 (83)
KOH solutions can be used to inactivate fecal coliforms in sewage sludge by storage at pH 10.5 during 14 days, the total bacteria and fungal microflora being only slightly affected.

11-APR-2001 (2)

KOH solutions should be considered for neutralizing plant acid wastes.

17-JAN-2001 (90)

Soils contaminated by KOH can be permanently treated by "in situ" vitrification (ISV).

17-JAN-2001 (23)
2.1 Melting Point

Value: = 406 degree C
Decomposition: no
Sublimation: no
GLP: no data
Test substance: no data
Remark: The melting point varies widely with the water content.
Reliability: (2) valid with restrictions
Flag: Critical study for SIDS endpoint
19-DEC-2001

Value: = 250 degree C
Decomposition: no
Sublimation: no
GLP: no data
Test substance: as prescribed by 1.1 - 1.4
Remark: The melting point varies widely with the water content.
Reliability: (2) valid with restrictions
Flag: Critical study for SIDS endpoint
19-DEC-2001

2.2 Boiling Point

Value: = 1327 degree C at 1013 hPa
Decomposition: no
GLP: no data
Test substance: no data
Reliability: (2) valid with restrictions
Flag: Critical study for SIDS endpoint
19-DEC-2001

Value: = 1324 degree C at 1013 hPa
Decomposition: no
GLP: no data
Test substance: no data
Reliability: (2) valid with restrictions
Flag: Critical study for SIDS endpoint
19-DEC-2001

2.3 Density

Type: relative density
Value: = 2.044 g/cm3 at 20 degree C
GLP: no data
Test substance: no data
Reliability: (2) valid with restrictions
Flag: Critical study for SIDS endpoint
02-JAN-2002

Type: relative density
Value: = 2.04 g/cm3 at 20 degree C
GLP: no data
2. PHYSICO CHEMICAL DATA

Testsubstance: no data
Reliability: (2) valid with restrictions
(2g) Data from handbook or collection of data.
19-DEC-2001

2.3.1 Granulometry

2.4 Vapour Pressure

Value: = 1.3 hPa at 719 degree C
Decomposition: no
GLP: no data
Testsubstance: no data
Reliability: (2) valid with restrictions
(2g) Data from handbook or collection of data.
Flag: Critical study for SIDS endpoint
19-DEC-2001

Value: = 1.3 hPa at 714 degree C
Decomposition: no
GLP: no data
Testsubstance: no data
Reliability: (2) valid with restrictions
(2g) Data from handbook or collection of data.
19-DEC-2001

2.5 Partition Coefficient

log Pow:
Method:
Year:
Remark: Not applicable when the species undergo dissociation in either phase.
10-JAN-2001

2.6.1 Water Solubility

Value: = 1100 g/l at 25 degree C
Qualitative: of very high solubility
GLP: no data
Testsubstance: no data
Reliability: (2) valid with restrictions
(2g) Data from handbook or collection of data.
Flag: Critical study for SIDS endpoint
19-DEC-2001

Value: = 1120 g/l at 20 degree C
Qualitative: of very high solubility
GLP: no data
Testsubstance: no data
Reliability: (2) valid with restrictions
(2g) Data from handbook or collection of data.
02-JAN-2002

pH: ca. 13.5 at 5.611 g/l and 25 degree C
GLP: no data
Testsubstance: no data
Remark: From a theoretical point of view, the pH of a 0.1M solution should be 13. pKa is not applicable (strong base, totally dissociated).
Reliability: (2) valid with restrictions
(2g) Data from handbook or collection of data.
19-DEC-2001

2.6.2 Surface Tension

2.7 Flash Point

Value:
Type:
Method:
Year:
Remark: not applicable
10-JAN-2001

2.8 Auto Flammability

Value:
Remark: Not applicable.
10-JAN-2001

2.9 Flammability

Result: non flammable
GLP: no data
Testsubstance: no data
07-MAY-2001

2.10 Explosive Properties

Result:
Remark: Not applicable.
10-JAN-2001

2.11 Oxidizing Properties

Result:
Remark: Not applicable.
10-JAN-2001

2.12 Additional Remarks

Memo: When dissolved in water or alcohol or when the solution is treated with acid, much heat is generated.
11-APR-2001
Memo: Enthalpy of solution
KOH: -57.61 kJ/mol.
KOH.H2O: -14.64 kJ/mol.
KOH.1.5H2O: -10.46 kJ/mol.
11-APR-2001

Memo: Readily absorbs moisture and carbon dioxide from air and deliquesces.
11-APR-2001

Memo: When heated to decomposition it emits toxic fumes of K2O.
11-APR-2001

Memo: When wet, attacks metals such as aluminium, tin, lead and zinc to produce flammable hydrogen gas.
11-APR-2001

Memo: Aggregate stability and saturated hydraulic conductivity Ks decreases with increasing hydroxide concentration.
11-APR-2001

Memo: Hydroxide anions increase (concentration dependent) the negative charge (CEC) of soils. Higher exchangeable potassium percentage (EPP) values are measured, compared with chloride.
11-APR-2001
3. ENVIRONMENTAL FATE AND PATHWAYS

3.1 Photodegradation

Type: 
Method: 
Year: GLP: 
Test substance: 
Remark: Not applicable. 10-JAN-2001

3.1.2 Stability in Water

Type: 
Method: 
Year: GLP: 
Test substance: 
Remark: Not applicable. 10-JAN-2001

3.1.3 Stability in Soil

3.2 Monitoring Data (Environment)

Type of measurement: background concentration
Medium: surface water
Method: Concentration
Remark: Potassium statistics for 75 world rivers:
mean: 3.22 mg/l
10th percentile: 0.80 mg/l
90th percentile: 5.96 mg/l
range: 41.90 mg/l

Potassium statistics for 11 rivers from North America:
mean: 2.35 mg/l
10th percentile: 0.80 mg/l
90th percentile: 5.00 mg/l
range: 7.20 mg/l

Potassium statistics for 5 rivers from South America:
mean: 1.58 mg/l
10th percentile: 0.74 mg/l
90th percentile: 2.30 mg/l
range: 1.80 mg/l

Potassium statistics for 25 rivers from Asia:
mean: 2.17 mg/l
10th percentile: 0.84 mg/l
90th percentile: 3.94 mg/l
range: 6.80 mg/l
Potassium statistics for 7 rivers from Africa:
mean: 1.84 mg/l
10th percentile: 1.16 mg/l
90th percentile: 2.66 mg/l
range: 2.10 mg/l

Potassium statistics for 21 rivers from Europe:
mean: 5.88 mg/l
10th percentile: 0.80 mg/l
90th percentile: 7.00 mg/l
range: 41.40 mg/l

Potassium statistics for 6 rivers from Oceania:
mean: 2.87 mg/l
10th percentile: 0.40 mg/l
90th percentile: 5.00 mg/l
range: 5.60 mg/l

Levels of concern for water quality from anthropogenic exposure, predominantly fertilizers, are never reached (12 mg/l WHO guideline for drinking water), except downstream from potash mines (Weser: 42.0 mg/l; Elbe: 26.1 mg/l).

Acidity/alkalinity is measured as pH which is a key parameter in water quality. pH is closely linked to biological productivity in aquatic systems. With dissolved organic acids from soil leaching (Amazonia), a pH of less than 4.0 has been measured. In waters with a high chlorophyll content, the bicarbonate assimilation can result in pH values exceeding quiet commonly 8.5, and even 9.0 at midday (Loire).

Result:
K in rivers, worldwide, < 5 mg/l; > 12 mg/l downstream of potash mines. Monitoring values worldwide.
pH annual average 6.5 - 8.3; global median value 7.7. No strong variability at individual stations.

Attached doc.: UNEPbiscarbK010718.xls
UNEP bicarbonate and potassium in rivers worldwide

Reliability:
(1) valid without restriction
(1d) Test procedure in accordance with generally accepted scientific standards and described in sufficient detail.

Flag:
Critical study for SIDS endpoint
19-DEC-2001

Type of measurement:
Medium:
Method:

Concentration
Remark:
With a global potash production of 29.341.000 tons K2O, or 24.357.391 tons K in 1989, and an estimated KOH production that not exceeded 800.000 tons that year, or 557.476 tons K (Ullmann, 1998), and taking into account that no more than 11% of the K from KOH could be discharged to the environment (see section 1.7 Use Pattern: 5% wide dispersive use + 6% in alkaline batteries; the rest is transformed in industrial
uses; Occidental Chem. Corp., 2000), not more than 61.322 tons K coming directly from KOH reaches the environment, or 0.25% of the total anthropogenic potassium.

Reliability:
(2) valid with restrictions
(2e, 2g) Study well documented, meets generally accepted scientific principles, acceptable for assessment. Data from handbook or collection of data.

Flag:
Critical study for SIDS endpoint

19-DEC-2001

Type of measurement: background concentration
Medium: surface water
Method: Concentration
Result:
Potassium at Rijn Lobith 1998: min. 4.4 mg/ml; max. 6.8; mean 5.6; n=13.
pH at Rijn Lobith 1998: min. 7.42; max. 7.87; mean 7.7; n=304.

Reliability:
(2) valid with restrictions
(2e) Study well documented, meets generally accepted scientific principles, acceptable for assessment.

19-DEC-2001

Type of measurement: background concentration
Medium: surface water
Method: Concentration
Result:
Potassium Maas Tailfer 1998: min. 2.2 mg/ml; max. 3.0; mean 2.7; n=13.
pH Maas Tailfer 1998: min. 7.92; max. 8.36; mean 8.18; n=13.

Reliability:
(2) valid with restrictions
(2e) Study well documented, meets generally accepted scientific principles, acceptable for assessment.

19-DEC-2001

Type of measurement: background concentration
Medium: surface water
Method: Concentration
Result:
Potassium Maas Eijsden 1998: min. 2.5; max. 5.0; mean 3.5; n=13.
pH Maas Eijsden 1998: min. 7.08; max. 8.17; mean 7.65; n=311.

Reliability:
(2) valid with restrictions
(2e) Study well documented, meets generally accepted scientific principles, acceptable for assessment.

19-DEC-2001

Type of measurement: background concentration
Medium: surface water
Method: Concentration
Result:
Potassium Maas Keizersveer 1998: min. 3.6; max. 8.1; mean 5.5; n=27.
pH Maas Keizersveer 1998: min. 7.61; max. 8.08; mean 7.84; n=14.
3. ENVIRONMENTAL FATE AND PATHWAYS

3.1 Transport between Environmental Compartments

Type: (2) valid with restrictions
Media:
Air (Level I):
Water (Level I):
Soil (Level I):
Biota (L.II/III):
Soil (L.II/III):
Method:
Year:
Remark: Very high solubility in water; no transport to air (see physico-chemical data).
10-JAN-2001

3.2 Distribution

Media:
Method:
Year:
Remark: Not applicable.
10-JAN-2001

3.3 Mode of Degradation in Actual Use

Remark: Not applicable.
10-JAN-2001

3.4 Biodegradation

Type:
Inoculum:
Method: GLP:
Year:
Test substance:
Remark: Not applicable.
10-JAN-2001

3.5 BOD5, COD or BOD5/COD Ratio

Remark: Not applicable.
10-JAN-2001

3.6 Bioaccumulation

Species:
Exposure period:
Concentration:
BCF:
Elimination:
Method:
Year: GLP:
Test substance: 
Remark: Not applicable.
10-JAN-2001

3.8 Additional Remarks

Memo: KOH solutions can be used to inactivate fecal coliforms in sewage sludge by storage at pH 10.5 during 14 days, the total bacteria and fungal microflora being only slightly affected.
17-JAN-2001 (2)

Memo: KOH solutions should be considered for neutralizing plant acid waste.
11-APR-2001 (91)

Memo: Neutralization and discharge to sewer: carefully dissolve in water and neutralize with dilute acetic acid. Flush to sewer with lots of water, regulations permitting or dispose of through a licensed contractor.
11-APR-2001 (91)
AQUATIC ORGANISMS

4.1 Acute/Prolonged Toxicity to Fish

Type: static  
Species: Gambusia affinis (Fish, fresh water)  
Exposure period: 96 hour(s)  
Unit: mg/l  
Analytical monitoring: no  
NOEC: m = 56  
LC50: c = 80  
Method: other  
Year: 1956  
Test substance: other TS  
Method:  
- Type: static with checks after 24, 48, 72 and 96 hrs.  
- GLP: N.  
- Species/Strain/Supplier: Gambusia affinis (mosquitofish).  
- Analytical monitoring: temperature, turbidity and pH, measured just after the chemical was added and daily throughout the experiment.  
- Exposure period: 96 hrs.  
- Statistical methods: no data.

Result:  
TLm at 24 hrs. = 85 mg/l.  
TLm at 48 hrs. = 80 mg/l.  
TLm at 96 hrs. = 80 mg/l.  
All fish were normal at 56 mg/l and less. At 100 mg/l, 7 fish died in 24 hrs., another died in 48 hrs. and the remaining 2 seemed normal at 96 hrs.

Test condition:  
- Test fish:  
  * Wild caught (Stillwater Creek in Payne County, Oklahoma).  
  * Age/sex/weight: adult females.  
  * Feeding: locally collected detritus and plankton; during the test the fish were not fed.  
  * Pretreatment: 2-3 weeks acclimatization in laboratory.  
- Test conditions:  
  * Dilution water: 2 local farm ponds with turbid water.  
  * Dilution water chemistry: pH 7.8-8.3; alkalinity < 100 mg/l.  
  * Concentrations: 10, 18, 32, 56 and 100 mg/l.  
  * Exposure vessel type: cylindric pyrex jars 12 in. high and 12 in. diameter containing 15 liters.  
  * Number of replicates/fish per replicate: 1/10.  
  * Aeration: yes.  
  * pH: 7.3-10.3 (100 mg/l).  
  * Oxygen content: no data.  
  * Turbidity: 117 mg/l (init.), 55 mg/l (fin.).  
  * Photoperiod: no data.  
- Test temperature range: 18-19°C.  
- Test parameter: mortality.  
- Method of calculating: median tolerance limit (TLm) plotted on logarithmic paper.

Test substance: Chemically pure
### Reliability: invalid

(3a) Documentation insufficient for assessment. The dilution water was turbid, which could influence the buffer (neutralization) capacity of the water. This is a significant methodological deficiency.

19-DEC-2001

| Type: static |
| Species: Salvelinus fontinalis (Fish, estuary, fresh water) |
| Exposure period: 24 hour(s) |
| Unit: mg/l |
| Minimum lethal dose = 5 |
| Analytical monitoring: no data |
| Method: other |
| Year: 19-DEC-2001 |
| Test substance: high grade purity |

#### Result:
Symptoms at 2x the minimum lethal dose were slow onset, restlessness, secretion of mucus, sluggish movements, irregular movements, late loss of equilibrium, decreased respiration and oxygen hunger.

| Test substance: high grade purity |
| Reliability: invalid |

19-DEC-2001

| Type: static |
| Species: Poecilia reticulata (Fish, fresh water) |
| Exposure period: 24 hour(s) |
| Unit: mg/l |
| LC50: = 165 |
| Analytical monitoring: no data |
| Method: other |
| Year: 19-DEC-2001 |
| Test substance: no data |

#### Method:
5 fishes per sample in glass desiccators.

| Reliability: invalid |

19-DEC-2001

In a review/abstract, the authors report the following concentrations that killed fish:

- Cyprinodon sp. (minnows): 28.6 mg/l, 24 hrs.
- Salvelinus fontinalis (trout): 50 mg/l, 24 hrs.
- Bluegills: 56 mg/l, 24 hrs.
- Bluegills: 56 mg/l, 4 hrs 30, distilled water.
- Gambusia affinis (mosquito-fish): 80-85 mg/l, 24 & 96 hrs TLm, turbid water.
- Carrassius auratus (goldfish): 140 mg/l, 24 hrs.
The following concentrations were not harmful to fish.
Bluegills: 28 mg/l, 24 hrs.
Bluegills: 28 mg/l, 24 hrs, distilled water.

Reliability:
(4) not assignable
(4b) Secondary literature.

03-JAN-2002

Type: static
Species: Gambusia affinis (Fish, fresh water)
Exposure period: 96 hour(s)
Unit: mg/l
Analytical monitoring: no
NOEC: m = 84
LC50: c = 125
Method: other

Result:
- All fish were normal at 84 mg/l and lower. At 100 mg/l one
  fish died in 24 hours and another died in 48 hours. At 180
  mg/l and higher all fish died. The median tolerance limit
  (TLm) after 24, 48 and 96 hours was 125 mg/l.

Test condition:
TEST ORGANISMS
- Wild caught: Stillwater Creek in Payne County, Okla.
- Age/sex/weight: adult females.
- Feeding: locally collected detritus and plankton; during the
  test the fish were not fed.
- Pretreatment: 2-3 weeks acclimatization in laboratory.

DILUTION WATER
- Source: two local farm ponds with turbid water.
- pH: between 7.8 and 8.3.
- Alkalinity: < 100 mg/l.

TEST SYSTEM
- Test conc.: 10; 18; 32; 56; 100; 180; 320; 560; 1000 mg/l.
- Exposure vessel type: cylindric pyrex jars 12 in. high and
  12 in. diameter containing 15 liters.
- Number of replicates/fish per replicate: 1/10.
- Aeration: yes.
- pH: 8.3-9.0 (100 mg/l).
- Test temperature: 22-24°C.
- Oxygen content: not described.
- Turbidity: 1000 mg/l (init.), 550 mg/l (final).
- Photoperiod: not described.

Test substance: NaOH (SODIUM HYDROXIDE) STUDY!

Reliability:
(3) invalid
(3a) Documentation insufficient for assessment.
The dilution water was turbid, which could influence the
buffer (neutralization) capacity of the water. This is a
significant methodological deficiency. The pH was not measured
at all concentrations.
**OECD SIDS**

**POTASSIUM HYDROXIDE**

**4. ECOTOXICITY**

**DATE:** 30-JAN-2002

**ID:** 1310-58-3

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**Type:** static  
**Species:** Carassius auratus (Fish, fresh water)  
**Exposure period:** 24 hour(s)  
**Unit:** mg/l  
**Analytical monitoring:** no

**NOEC:** m = 100  
**LC50:** m = 160  
**Method:** other

**Test substance:** no data  
**Result:**

500 ppm: both fish expired in 15 minutes.  
160 ppm: median tolerance limit (TLm) 24 hours.  
100 ppm: both fish survived 24 hours.  
50 ppm: both fish survived 24 hours.  
25 ppm: both fish survived 24 hours.

**RESULTS CONTROL**  
No data.

**Test condition:** DILUTION WATER  
- Source: Louisville city water.

**TEST SYSTEM**  
- Concentrations: 25, 50, 100, 160 and 500 mg/l.  
- Exposure vessel type/test volume: 16 liters.  
- Number of replicates/fish per replicate: 1/2.  
- pH: 9.8 (100 mg/l).

**TEST PARAMETER**  
- Mortality.

**Reliability:** (3) invalid  
(3a) Documentation insufficient for assessment, several test conditions not described.

**28-DEC-2001**

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**Type:** static  
**Species:** other: Lucioperca Lucioperca L. (pike perch)  
**Exposure period:** 24 hour(s)  
**Unit:** mg/l  
**Analytical monitoring:**

**LOEC :** m = 35  
**Method:** other

**Test substance:** no data  
**Result:**

Solutions of NaOH in pond water started to be toxic to the fry of Lucioperca Lucioperca L. (pike perch) at NaOH concentrations of 35 mg/l (pH 8.2) and higher. At a concentration of 52 mg/l, 40% of the total fry died within 24 hours. Thus a pH over 8.2 appeared to be dangerous to the pike perch fry.

**Test condition:** TEST ORGANISMS  
- Wild caught: lake Goplo.  
- Age/size/weight: fry, 11.5-16 mm.

DILUTION WATER  
- Source: pond water.  
- Total hardness: 130 mg/l CaCO3.

**TEST SYSTEM**  
- Exposure vessel type: glass aquariums.

**TEST PARAMETER**  
- Mortality.

**Test substance:** NaOH (SODIUM HYDROXIDE) STUDY!
Reliability: (3) invalid
   (3a) Documentation insufficient for assessment.
A pH of 8.2 is a very normal pH for aquatic ecosystems and for
this reason it is doubtful if a pH of 8.2 is really toxic for
the fry of pike perch.

28-DEC-2001                                                                 (80)

Type: other
Species: Leuciscus idus melanotus  (Fish, fresh water)
Exposure period: 48 hour(s)
Unit: mg/l  Analytical monitoring: no data
LC0: = 157
LC50: = 189
LC100: = 213
Year: 1975  GLP: no
Test substance: no data
Test substance: NaOH (SODIUM HYDROXIDE) STUDY!
Reliability: (4) not assignable
   (4e) Documentation insufficient for assessment.
Several test conditions not described.

28-DEC-2001                                                                 (41)

Type: static
Species: Pimephales promelas  (Fish, fresh water)
Exposure period: 96 hour(s)
Unit: mg/l  Analytical monitoring: yes
LC50: c = 880
Method: other: EPA/600/4-90/027
Year: GLP: no
Test substance: other TS
Remark: All organisms, 1 to 7 days old, were obtained from in-house
cultures. Brood stock were cultured at 20-25°C in tap water
pretreated with activated carbon. Larva were fed Artemia sp.
twice daily until they were used in testing.

The toxicity tests followed the general guidance of the U.S.
Environmental Protection Agency. All tests were conducted in
30 ml plastic beakers containing 10 ml test solution and five
organisms per chamber. Tests were conducted under a 16 h/8 h
light-dark photoperiod at 25°C. Dilution/control water for all
tests was moderately hard reconstituted water (MHRW). Exposure
period was 96 hours, with daily observations of mortality. The
criteria for death were no visible movement and no response to
prodding. 100 µl of concentrated brine shrimp nauplii was
added after 48 hours of exposure.

Test solutions were prepared by dissolving 10,000 mg/l KCl
salt in MHRW, and diluting with MHRW to a series of 5 test
concentrations spaced on 0.5 dilution factor. As testing
proceeded, test concentrations were spaced much more closely
to better define responses near the effect threshold.

All ion concentrations measured in stock solutions were
comparable to nominal values. If measured concentration
differed from the nominal value by more than 20%, the actual
measured concentrations were substituted for the nominal
concentrations. Background KCl content in the dilution water
MHRW were added to the calculated contributions from the stock
solutions.
Dissolved oxygen (DO) and pH were measured in selected test solutions during actual toxicity testing: measured DO concentrations were always > 40% saturation; measured pH was between 7.5 - 9.0.

3 replicate tests were conducted, and average LC50 values were calculated as the arithmetic mean of the values; SD = 15, range = 1.4 - 62.

Result:
LC50 24 hrs: 950 mg/l
LC50 48 hrs: 910 mg/l
LC50 96 hrs: 880 mg/l

Test substance: KCl (POTASSIUM CHLORIDE) STUDY!
Reagent grade.

Reliability:
(1) valid without restriction
(1b) Comparable to guideline study.

03-JAN-2002

### 4.2 Acute Toxicity to Aquatic Invertebrates

<table>
<thead>
<tr>
<th>Type</th>
<th>other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Species</td>
<td>other aquatic mollusc</td>
</tr>
<tr>
<td>Exposure period:</td>
<td>mg/l</td>
</tr>
<tr>
<td>Analytical monitoring:</td>
<td>no data</td>
</tr>
<tr>
<td>EC0:</td>
<td>&lt; 1</td>
</tr>
<tr>
<td>EC100:</td>
<td>&gt; 10</td>
</tr>
<tr>
<td>Method:</td>
<td>other</td>
</tr>
<tr>
<td>Year:</td>
<td>GLP: no data</td>
</tr>
</tbody>
</table>

Test substance: no data

Method:
Exposure periods up to several days.
Species: Dreissena polymorpha (zebra mussels).
Reliability:
(4) not assignable
(4a) Abstract.

19-DEC-2001

<table>
<thead>
<tr>
<th>Type</th>
<th>static</th>
</tr>
</thead>
<tbody>
<tr>
<td>Species</td>
<td>Daphnia magna (Crustacea)</td>
</tr>
<tr>
<td>Exposure period:</td>
<td>48 hour(s)</td>
</tr>
<tr>
<td>Unit:</td>
<td>mg/l</td>
</tr>
<tr>
<td>Analytical monitoring:</td>
<td>yes</td>
</tr>
<tr>
<td>EC50:</td>
<td>c = 660</td>
</tr>
<tr>
<td>Method:</td>
<td>other: EPA/600/4-90/027</td>
</tr>
<tr>
<td>Year:</td>
<td>GLP: no</td>
</tr>
</tbody>
</table>

Test substance: other TS

Remark:
All organisms were obtained from in-house cultures. D. magna were cultured in hard reconstituted water at 20°C. Test organisms were less than 24 hours old at test initiation.

The toxicity tests followed the general guidance of the US Environmental Protection Agency. All tests were conducted in 30 ml plastic beakers containing 10 ml test solution and five organisms per chamber. Tests were conducted under 16 hr/8 hr light-dark photoperiod at 20°C. Dilution/control water for all tests was moderately hard reconstituted water (MHRW). Exposure period was 48 hours, with daily observations of mortality. 100 µl of a 1:1 mix of YCT and algal suspension was added to the test chambers at test initiation. The criteria for death were no visible movement and no response to prodding.
Test solutions were prepared by dissolving 10,000 mg/l KCl salt in MHRW, and diluting with MHRW to a series of 5 test concentrations spaced on 0.5 dilution factor. As testing proceeded, test concentrations were spaced much more closely to better define responses near the effect threshold.

All ion concentrations measured in stock solutions were compared to nominal values. If measured concentrations differed from the nominal value by more than 20%, the actual measured concentrations were substituted for the nominal concentrations. Background KCl content in the dilution water MHRW were added to the calculated contributions from the stock solutions.

Dissolved oxygen (DO) and pH were measured in selected test solutions during actual toxicity testing: measured DO concentrations were always > 40% saturation; measured pH was between 7.5 - 9.0.

5 replicate tests were conducted, and average LC50 values were calculated as the arithmetic mean of the values; SD = 7.5; range 4.8 - 31.

Result:
LC50 24 hrs: 740 mg/l
LC50 48 hrs: 660 mg/l.

Test substance: KCl (POTASSIUM CHLORIDE) STUDY!
Reagent grade.

Reliability: (1) valid without restriction
(1b) Comparable to guideline study.

28-DEC-2001 (56)

Type: Species: Daphnia magna (Crustacea)
Exposure period: mg/l Analytical monitoring: no data
Toxicity threshold: = 40 - 240
Method: other: invertebrate toxicity test
Year: GLP: no
Test substance: no data
Test substance: NaOH (SODIUM HYDROXIDE) STUDY!
Reliability: (4) not assignable
(4b) Secondary literature.

03-JAN-2002 (50)

Type: Species: other aquatic arthropod: freshwater insect larvae
Exposure period: mg/l Analytical monitoring: no data
Lethal : = 125 - 1000
Method: other: invertebrate toxicity test
Year: GLP: no
Test substance: no data
Test substance: NaOH (SODIUM HYDROXIDE) STUDY!
Reliability: (4) not assignable
(4b) Secondary literature.

03-JAN-2002 (50)
OECD SIDS

4. ECOTOXICITY

ID: 1310-58-3

ECOTOXICITY

UNEP PUBLICATIONS

DATE: 30-JAN-2002

IDE: 1310-58-3

**Type:**

**Species:** Ceriodaphnia sp. (Crustacea)

**Exposure period:** 48 hour(s)

**Unit:** mg/l

**Analytical monitoring:** no

**EC50:** c = 40.4

**Method:** other

**Year:** 1999

**GLP:** no

**Test substance:** other TS

**Method:** METHOD FOLLOWED
- Acute 48-h immobilization test according to the NSW Environment Protection Authority.

**STATISTICAL METHODS**
- Trimmed Spearman-Karber.

**Test condition:**

**TEST ORGANISMS**
- Source/supplier: not described.
- Feeding: S. capricornutum and Ankistrodesmus sp., no feeding during the test.

**STOCK AND TEST SOLUTION**
- Vehicle, solvent: no solvent used.
- Source: dechlorinated and filtered Sydney mains water, aged 1 month and adjusted to 500 µS/cm with seawater.
- Hardness: not described.

**DILUTION WATER**
- Source: dechlorinated and filtered Sydney mains water, aged 1 month and adjusted to 500 µS/cm with seawater.
- Hardness: not described.

**TEST SYSTEM**
- Test concentrations: five concentrations in a geometric series, plus a control.
- Exposure vessel type: 200 ml test solution in a 250 ml glass beaker.
- Number of replicates/individuals per replicate: 3/5.
- Test temperature: 23 +/- 1°C.
- Dissolved oxygen: measured, but not described.
- pH: measured, but not described.
- Intensity of radiation: < 1000 lx.
- Photoperiod: 16h:8h light-dark cycle.

**TEST PARAMETER**
- Immobility.

**Test substance:** NaOH (SODIUM HYDROXIDE) STUDY!

**Reliability:**

(2) valid with restrictions
(2d) Test procedure in accordance with national standard methods with acceptable restrictions.

02-JAN-2002

**Type:**

**Species:** other aquatic mollusc: Vectro snail

**Exposure period:**

**Unit:** Analytical monitoring: no

**Method:** other

**Year:** 1961

**GLP:** no

**Test substance:** other TS

**Method:** METHOD FOLLOWED
- 96 h test.

**STATISTICAL METHOD**
- Not described.

**Result:**

RESULTS: EXPOSED
- The results showed that Biomphalaria a. alexandrina tolerated a concentration of 400 mg/l NaOH. Bulinus truncatus and Lymnaea caillaudi tolerated a 100 mg/l NaOH solution. The lethal concentration of NaOH to Biomphalaria a. alexandrina, Bulinus truncatus and Lymnaea caillaudi was
450, 150 and 150 mg/l respectively.

RESULTS: CONTROLS
- Not described.

Test condition: TEST ORGANISMS
- Wild caught: river Nile.
- Age/size/weight: full grown snails.
- Feeding: not described.
- Pretreatment: 3 days acclimatization in laboratory.

DILUTION WATER
- Source: cleared Nile water.
- Alcalinity: not described.

TEST SYSTEM
- Test conc.: series of concentrations varying 50 mg/l.
- Exposure vessel type: 200 ml test solution in a 250 ml jar.
- Number of replicates, snails per replicate: 1/20.
- pH: not described.
- Test temperature: 27°C.
- Oxygen content: not described.

TEST PARAMETER
- Mortality.

Test substance: NaOH (SODIUM HYDROXIDE) STUDY!

Reliability: (3) invalid
(3a) Documentation insufficient for assessment. Test procedure is not a standard method.

02-JAN-2002 (30)

Type: other: Ophryotrocha diadema
Species: other TS
Exposure period: 48 hour(s)
Unit: mg/l Analytical monitoring: no
LC50 : = 33 - 100
Method: other
Year: 1983 GLP: no
Test substance: other TS
Method: METHOD FOLLOWED
- Acute 48-h toxicity test.
STATISTICAL METHODS
- Not described.

Test condition: TEST ORGANISM
- Source/supplier: University of Gothenburg, Sweden.
- Feeding: fragmented spinach. No feeding during the test.

STOCK AND TEST SOLUTION
- Vehicle, solvent: no solvent used.

DILUTION WATER
- Source: filtered sea water.
- Hardness: not described.

TEST SYSTEM
- Test concentrations: a half-logarithmic series of concentrations and one control.
- Exposure vessel type: 50 ml test solution.
- Number of replicates/individuals per replicate: 2/10.
- Test temperature: not described.
- Dissolved oxygen: not described.
- pH: not described.

TEST PARAMETER
- Mortality.

Test substance: NaOH (SODIUM HYDROXIDE) STUDY!
4.3 Toxicity to Aquatic Plants e.g. Algae

<table>
<thead>
<tr>
<th>Species: Nitscheria linearis</th>
<th>(Algae)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endpoint: growth rate</td>
<td></td>
</tr>
<tr>
<td>Exposure period: 120 hour(s)</td>
<td></td>
</tr>
<tr>
<td>Unit: mg/l</td>
<td></td>
</tr>
<tr>
<td>EC50: m = 1337</td>
<td></td>
</tr>
<tr>
<td>Method: other</td>
<td></td>
</tr>
<tr>
<td>Year: 1968</td>
<td></td>
</tr>
<tr>
<td>Test substance: other TS</td>
<td></td>
</tr>
<tr>
<td>Remark: Diatoms were cultured in synthetic dilution water under controlled conditions. Tests were made by putting standard inoculum of cells in each of the 150 ml Erlenmeyer flasks, and comparing the number of cells produced at the end of a 5-day period. A graded series of concentrations was used.</td>
<td></td>
</tr>
<tr>
<td>Test substance: KCl (POTASSIUM CHLORIDE) STUDY!</td>
<td></td>
</tr>
<tr>
<td>Reliability: (2) valid with restrictions</td>
<td></td>
</tr>
<tr>
<td>GLP: no data</td>
<td></td>
</tr>
<tr>
<td>02-JAN-2002 (68)</td>
<td></td>
</tr>
</tbody>
</table>

4.4 Toxicity to Microorganisms e.g. Bacteria

| Type: aquatic |
| Species: Photobacterium phosphoreum  | (Bacteria) |
| Exposure period: 15 minute(s)        |         |
| Unit: mg/l                           |         |
| EC50: c = 22                         |         |
| Method: other                        |         |
| Year: GLP: no data                  |         |
| Test substance: no data              |         |
| Method: Microtox Toxicity Analyzer System, Microbics Corporation (Carlsbad CA). |
| Aliquots of 10 µl of the cell suspension are transferred to test vials containing 2% NaCl equilibrated to 15°C using a temperature-controlled photometer. Light readings are taken for each vial before and 15 minutes after sample addition. The amount of light lost per sample dilution is proportional to the toxicity of that sample. EC50 values were used as the measure of toxicity. The log of the gamma values from four dilutions of the sample were plotted against the log of the sample concentration. That concentration of sample causing a 50% reduction in light (gamma of 1.0) after 15 minutes exposure was designated the EC50 value (expressed in mg/ml) for the sample. |
| Reliability: (2) valid with restrictions |
| GLP: no data |
| Flag: Critical study for SIDS endpoint |
| 19-DEC-2001 (12) |
Type: Tetrahymena sp.  (Protozoa)
Species: 
Exposure period: 2 minute(s)  Analytical monitoring: no
Unit: 
Method: other  
Year: 1987  GLP: no
Test substance: other TS
Method: METHOD
Test in which the motility pattern of Tetrahymena was observed, evaluated and quantified. The positive control in this test was 1.0% sodium hydroxide.

Result:
When 1% NaOH was diluted 62 times, the motility was higher than 90% of control cell motility (highest tolerated dose, HTD). This would be equal to a NaOH concentration of 161 mg/l.

Test condition:
- Strain: Tetrahymena thermophila (30377).
- Source/supplier: ATCC, Rockville, MD.
- Feeding: liver powder, 0.1%; S. cerevisiae, 0.1%; soy lecithin, 0.001%.

DILUTION WATER
- Source: filtered MM2 medium.

TEST SYSTEM
- Test concentrations: not described.
- Exposure vessel type: 50 µl diluted chemical and 50 µl T. thermophila suspension is placed on a microscope coverglass.
- Test temperature: 30°C.

TEST PARAMETER
- Motility pattern.

Test substance: NaOH (SODIUM HYDROXIDE) STUDY!
Reliability: (3) invalid
(3a) Documentation insufficient for assessment. Several test conditions not described.

02-JAN-2002  (77)

4.5 Chronic Toxicity to Aquatic Organisms

4.5.1 Chronic Toxicity to Fish
Species: Lebistes reticulatus  (Fish, fresh water)
Endpoint: other
Exposure period: mg/l  Analytical monitoring: no
Unit: 
Method: other  
Year: 1977  GLP: no
Test substance: other TS
Method: METHOD FOLLOWED

- Two tests were run. In the first, fry of 1 to 2 days old were tested. In the second, sexually mature females were exposed together with males to solutions with NaOH.

STATISTICAL METHODS
- Not described.

Result:
The presence of NaOH had an adverse effect on the survival rate, growth and fecundity, as well as the quality of the progeny of the guppy. Upon prolonged exposure concentrations of 25 to 100 mg/l produced significant changes in the biology of the fish.
Test condition: TEST ORGANIMS
- Strain Lebistes reticulatus (guppy);
- Pretreatment: not described.
DILUTION WATER
- Not described.
TEST SYSTEM
- Concentrations: 25, 50, 75 and 100 mg/l and one control.
- Exposure vessel Type/test volume: glass aquaria with 3 ind/liter.
- Renewal of test solutions: daily.
- Test temperature: 20-25°C.
- Oxygen content: not described.
TEST PARAMETER
- Survival rate, growth, maturation time, fecundity.

Test substance: NaOH (SODIUM HYDROXIDE) STUDY!

Reliability: (3) invalid
(3a) Documentation insufficient for assessment.

02-JAN-2002 (75)

4.5.2 Chronic Toxicity to Aquatic Invertebrates
TERRESTRIAL ORGANISMS

4.6.1 Toxicity to Soil Dwelling Organisms

Type: artificial soil
Species: Enchytraeus sp. (Worm (Annelida), soil dwelling)
Endpoint: mortality
Exposure period: 90 day
Unit: other
LC50: c = 850
LC92 : c = 3000
Method: KOH is mixed with a fresh pine substrate (pH 4.4) at 3 g/l litter, with 4 replicates (other bases, acids and salts were also tested). The humidity was adjusted weekly with rainwater. pH and conductivity was measured during 6 months. At regular times the enchytraeids are counted both in the controls and in the test samples. After 3 months, the conductivity of the controls and the KOH treated samples (means) were respectively 185 and 680 µS/cm. pH of the KOH treated samples passed from initially 7.3 to 6.3 (CO2 absorption). The conclusion of the study was that the decrease of enchytraeid population was not correlated to pH but to an increase of conductivity. Regardless to the acid, base or salt, the following regression was calculated:
\[ \log(y+1) = -0.002092x + 2.2409, \quad R = -0.8161, \] where \( y \) = population/100 ml and \( x \) = conductivity (µS/cm).

Remark: The values of LC50 and LC92 are calculated using the regression given in the method.
The enchytraeid population was >95% Cognetia sphagnetorum.

Result: LC50 = 850 mg/l
LC92 = 3000 mg/l
Reliability: (2) valid with restrictions
(2c) Comparable to guideline study with acceptable restrictions.
Flag: Critical study for SIDS endpoint

4.6.2 Toxicity to Terrestrial Plants

4.6.3 Toxicity to other Non-Mamm. Terrestrial Species

4.7 Biological Effects Monitoring

4.8 Biotransformation and Kinetics

4.9 Additional Remarks

Memo: TLm Mosquito fish = 80 ppm / 24 hr, fresh water.
Memo: Allowable Tolerances (ATOL), when used as a neutralizer in pesticides formulations for growing crops, raw agricultural commodities or animal applications: exempted.
18-JAN-2001 (33)

Memo: KOH, as well as nine potassium salts, at concentrations of 20 mM promoted growth of spiroplasmas cultures when supplemented with NaH2PO4.H2O.
Reliability: (3) invalid
(3b) Significant methodological deficiencies. Very high concentration of KOH was used (20 mM). Probably the pH was adapted.
18-JAN-2001 (15)

Memo: The ability of Cochliobolus sativa conidia to germinate is impaired in salt solutions or soils adjusted to a pH up to 9 with KOH or K2CO3. This is paralleled with a loss of endogenous carbon.
Reliability: (4) not assignable
(4a) Abstract.
19-DEC-2001 (34)

Memo: The minimum inhibitory concentration (MIC) of KOH ranged from 0.15 to 0.24% in bacterial cultures and from 0.10 to 0.24% in yeast cultures.
Reliability: (4) not assignable
(4a) Abstract.
19-DEC-2001 (38)

Memo: The minimum effective concentration of KOH with an exposure time of 10-20 minutes for killing specific bacteria was: Coccus sp.: 6%; Bacillus sotto: 2%; Serratia marcescens: 2%. Streptococcus sp. was resistant to 6%.
Reliability: (3) invalid
(3c) Unsuitable test system. Efficacy test, can not be used for environmental assessment.
18-JAN-2001 (5)

Memo: The light of a culture of the luminous bacteria Vibrio harveyi was reduced below 50% within 20 s after exposure to 150 ppb KOH.
Reliability: (3) invalid
(3a) Documentation insufficient for assessment. Species unknown.
18-JAN-2001 (70)

Memo: In a static test with sea urchin (Paracentrotus lividus) embryos, with an exposure period of 48 hr., the no effect pH of sea water alkalinized with KOH was 8.7.
Remark: KOH-alkalinized sea water, for an initial pH of 8.5-8.7, definitely improved the cultures, as compared to larvae being reared at the initial normal pH.
Reliability: (3) invalid
(3b) Significant methodological deficiencies. The pH is not constant during exposure. Results for KOH are poorly reported. It is also very strange that KOH improved cultures, while NaOH resulted in toxicity within the same pH interval!. Na concentration in seawater is very high.
19-DEC-2001 (65)
Memo: In a static test with sea urchin (Paracentrotus lividus) sperm (30 minutes exposure) and embryos (48 hr exposure), the no effect pH of sea water alkalinized with KOH was 8.85.

Reliability:
(3) invalid

(3b) Significant methodological deficiencies. The pH is not constant during exposure. Results for KOH are poorly reported. No monotone dose-response. Effects could be due to enhancement of the activity of trace-level genotoxicants. No direct pH effect!

18-JAN-2001
5.1 Acute Toxicity

5.1.1 Acute Oral Toxicity

Type: LD50
Species: rat
Strain: other
Sex: male
Number of Animals: 54
Vehicle: water
Value: = 365 mg/kg bw
Method: other
Year: GLP: no
Test substance: other TS

Method:
- Method/guideline: the 14-day survival population LD50, and their 95% confidence limits were calculated by the probit analysis of Finney (1971).
- Type: LD50.
- GLP: N.
- Year: no data.
- Species/strain: rat/Charles River albino.
- Sex: male.
- No. of animals per dose: 9.
- Vehicle: deionized-distilled water.
- Route of administration: gastric intubation.

Remark:
- Time of death: within 72 hrs. after dosing.
- Clinical signs: hyperexcitability followed by apathy and weakness, increased respiration rate, ruffled fur, eye closing and huddling, bloody nasal exudate.
- Necropsy findings: stomach and intestinal hemorrhage and adhesions of abdominal organs, blockage of the gastrointestinal tract, leakage of bloody fluid exudates into the peritoneal cavity.
- Potential target organs: stomach, pancreas, spleen, liver, small intestine.

Result:
- Value reported on the basis of 100% purity: LD50 = 365 mg/kg bw.
  95% confidence interval: 310-429 mg/kg bw.
- Number of deaths at each dose level: no data.

Test condition:
- Age: no data.
- Weight: 175-250 g.
- Rats were cesarean-derived, fasted overnight.
- Doses: 6 dosage levels, not further specified.
- Doses per time period: single dose.
- Volume administered or concentration: no data.
- Post dose observation period: 14 days.

Test substance: 85% purity.
Reliability: (2) valid with restrictions
(2d) Test procedure in accordance with national standard methods with acceptable restrictions.
Flag: Critical study for SIDS endpoint

19-DEC-2001 (40)
**OECD SIDS**

**POTASSIUM HYDROXIDE**

**DATE:** 30-JAN-2002

**ID:** 1310-58-3

---

**Type:** LD50  
**Species:** rat  
**Strain:** Sprague-Dawley  
**Sex:** male  
**Number of Animals:** 50  
**Vehicle:** no data  
**Value:** = 273 mg/kg bw  
**Method:** other  
**Year:** GLP: no data

**Test substance:** no data  
**Method:** - Method/guideline: 14-day survival population conventional LD50; data were analyzed by the probit procedure of the SAS system (SAS Institute Inc., 1985).  
- Type: LD50.  
- GLP: no data.  
- Year: no data.  
- Species/strain: rat/Sprague-Dawley.  
- Sex: male.  
- No. of animals per dose: 10.  
- Vehicle: no data.  
- Route of administration: oral gavage.

**Remark:**  
- Time of deaths: some deaths observed during the second week after dosing.  
- Clinical signs: no data.  
- Necropsy findings: no data.  
- Potential target organs: no data.  
- Weight variation of rats is greater than the +/- 20% limit stated in OECD guideline 401.  
- LD50 = 273 mg/kg bw.  
- 95% confidence interval: 214-324 mg/kg.  
- LD50 calculated taking deaths over first 7 days was 333 mg/kg and, using an "Up and Down" method, 388 mg/kg (only 7 days post-exposure period).

**Test condition:**  
- Age: no data.  
- Weight: 190-300g.  
- Rats were fasted, 18-20 hr prior to dosing.  
- Doses: 5 doses, with a geometric progression of 1.4.  
- Doses per time period: single dose.  
- Volume administered or concentration: no data.  
- Post dose observation period: 14 days.

**Reliability:** (2) valid with restrictions  
(2d) Test procedure in accordance with national standard methods with acceptable restrictions.

**Flag:**  
Critical study for SIDS endpoint

---

**Type:** LD50  
**Species:** rat  
**Strain:** other  
**Sex:** male  
**Number of Animals:**  
**Vehicle:** water  
**Value:** = 1230 mg/kg bw  
**Method:** other  
**Year:** GLP: no
OECD SIDS POTASSIUM HYDROXIDE
5. TOXICITY
DATE: 30-JAN-2002
ID: 1310-58-3

Test substance: no data
Method: Groups of 5 male rats were given doses of 0.1 gm/ml solution of KOH by intubation, the doses being increased in logarithmic fashion by a factor of 2. 14 day post-exposure period.
Remark: Range-finding data; 0.80-1.89 g/kg bw.
Strain: Carwoth - Wistar.
Reliability: (3) invalid
(3a) Documentation insufficient for assessment. Rats were not fasted prior to dosing.

5.1.2 Acute Inhalation Toxicity

5.1.3 Acute Dermal Toxicity

5.1.4 Acute Toxicity, other Routes

5.2 Corrosiveness and Irritation

5.2.1 Skin Irritation

Species: rabbit
Concentration: 5 %
Exposure: Occlusive
Exposure Time: 4 hour(s)
Number of Animals: 6
PDII: 4.8
Result: moderately irritating
EC classification: highly corrosive (causes severe burns)
Method: Draize Test
Year: GLP: no data
Test substance: no data
The test substance was tested as 5% and 10% solutions with covering, either as a 19 mm diameter Hill Top Chamber pad, or as a classical gauze. Covering time was 1 or 4 hr. with the chamber and 4 hr. with the gauze. Groups of rabbits were subgroups of 3/treatment for the chamber, and 6 for the gauze. Volumes applied were 0.2 ml for the chamber and 0.5 ml for the gauze. Sites were graded 30 min., 24, 48 and 72 hr. after removal of the patches using the Draize grading scale.
Primary irritation indices (PII) were calculated by adding the average of all erythema scores and the average of all edema scores.
Result:
- Chamber/5%/1hr/sub-group A. PII=6.8; response exceeds scale on 2 of 3 sites, excluded from average; severely irritating.
- Chamber/5%/1hr/sub-group B. Response exceeds scale on 3 of 3 sites; severely irritating.
- Chamber/5%/4hr/sub-group A. Response exceeds scale on 3 of 3 sites; severely irritating.
- Chamber/5%/4hr/sub-group B. PII=4.7; response exceeds scale on 1 of 3 sites, excluded from average; severely irritating.
irritating.
- Chamber/10%/1 and 4hr/sub-groups A and B. Response exceeds scale on 3 of 3 sites; severely irritating.
- Gauze/5%/4hr. PII=4.8; moderately irritating.
- Gauze/10%/4hr. Response exceeds scale on 3 of 3 sites; severely irritating.

**Test condition:** 4 DOT patches (2 per side) per rabbit.

**Reliability:** (2) valid with restrictions
(2e) Study well documented, meets generally accepted scientific principles, acceptable for assessment.

**Flag:** Critical study for SIDS endpoint

---

**Species:** rabbit
**Concentration:** 5%

**Exposure:** Occlusive
**Exposure Time:** 24 hour(s)
**Number of Animals:** 6
**PDII:** Result: moderately irritating
**EC classificat.:** irritating
**Method:** Draize Test
**Year:** GLP: no

**Test substance:** The test material (0.1 ml) was tested as a 5% solution on 6 albino rats with abraded and intact skin. Covering was with a 20 mm² gauze patch. Evaluation was after 24 and 48 hr. Irritation indices were scored according to a modified Draize technique.

**Result:** Intact skin: mild irritant. Abraded skin: extreme irritant.

**Test condition:** Controls: distilled water (negative) and HCl 5% (positive).

---

**Species:** rabbit
**Concentration:** 10%

**Exposure:** Occlusive
**Exposure Time:** 4 hour(s)
**Number of Animals:** 6
**PDII:** Result: corrosive
**EC classificat.:** highly corrosive (causes severe burns)
**Method:** Draize Test
**Year:** GLP: no data

**Test substance:** no data

**Method:** According to FDA (USA), Federal Hazardous Substance Act, 1972. The test substance (0.5ml of a 10% aqueous solution; 50mg) was applied to both intact and abraded skin of 6 rabbits, kept under occlusion for 4 hr. Sites were evaluated for erythema and edema after 4, 24 and 48 hr, and were scored according to the above mentioned procedure. Primary
irritation indices were calculated by averaging the scores of all sites. Tests were also performed on guinea pigs with the same protocol.

**Result:**

**Rabbits:**
Mean scores: intact >6.9; abraded >7.0.
PIII: >6.9.
Tissue destruction: intact 6/6; abraded 6/6.

**Guinea pigs:**
Mean scores: intact >7.6; abraded >7.6.
PIII: >7.6.
Tissue destruction: intact 6/6; abraded 6/6.

**Test condition:**
Area of exposure: 4 sites/rabbit and 2 sites/guinea pig.
Scoring system: according to the descriptive scales in the proposed revision of the test procedure (FHSA, FDA, 1972).

**Reliability:**
(2) valid with restrictions
(2d) Test procedure in accordance with national standard methods with acceptable restrictions.

**Flag:**
Critical study for SIDS endpoint

---

Species: rabbit
Concentration: 2 %

**Exposure:**
Occlusive
**Exposure Time:** 4 hour(s)
**Number of Animals:** 6

**PDII:** corrosive
**Result:** corrosive (causes burns)
**EC classificat.:** corrosive (causes burns)
**Method:** Draize Test

---

Species: other
Concentration: 10 %

**Exposure:**
**Exposure Time:** 3 minute(s)
**Number of Animals:**

**PDII:**
**Result:** corrosive
**EC classificat.:** highly corrosive (causes severe burns)
**Method:** In-vitro test
**Year:** GLP: no data
**Test substance:** other TS
**Method:** KOH 10% were applied topically to the stratum corneum surface of reconstructed human skin cultures Skin²ZS1301 (15µl) and EpiDerm (25µl). Skin culture damage or cytotoxicity was measured as decreased 3-(4,5-dimethylthiazol-2-yl)2,5-diphenyltetrazolium bromide (MTT) vital dye metabolism. In time-course experiments, the time (minutes) of test material exposure eliciting 50% reduction of MTT metabolism was calculated (t50). Corrosive substances were found to have a t50 < 3 minutes. After a 3 minutes treatment with the test substance, cell viability was determined by measurement of MTT metabolism. Treatment with corrosive chemicals reduced cell viability to <50% of control levels. A comparison with the histological damage was done.

**Result:**
- t50 was 1.5 (+- 0.98 SD).
- %Viability after 3 minutes:
  - Skin²ZS1301: 4.2%
  - EpiDerm: 14.8% +6.6
- Histological grade with Skin²ZS1301: most severe.

**Test substance:** Aldrich Chemical Co. or Sigma Chemical Co.
**Reliability:**
- (3) invalid
  - (3c) Unsuitable test system.

19-DEC-2001

**Species:** other
**Concentration:** 10 %

**Exposure:**

**Exposure Time:**

**Number of Animals:**

**PDII:** corrosive
**Result:**
**EC classificat.:** highly corrosive (causes severe burns)
**Method:** In-vitro test
**Year:** GLP: no data
**Test substance:** no data
**Method:** 4 "in vitro" methods were compared in an international validation study with, among others, KOH 10% and 5%:
- The rat skin TER assay.
- Corrositex.
- The Skin²zk1350 corrosivity test.
- Episkin TM.

**Result:**
- The 4 methods discriminated KOH 10% as highly corrosive (R35) or corrosive (R34).
- TER, Corrositex and Episkin discriminated KOH 5% as highly corrosive (R35) or corrosive (R34). Skin² did not.

**Reliability:**
- (3) invalid
  - (3c) Unsuitable test system.

19-DEC-2001
5.2.2 Eye Irritation

Species: rabbit
Concentration: 5 %
Dose: .1 ml
Exposure Time: 5 minute(s)
Comment: rinsed after (see exposure time)
Number of Animals: 1
Result: highly corrosive
EC classification: risk of serious damage to eyes
Method: Draize Test

Test substance: other TS
Method: 0.1ml of the test substance was administered at concentrations of 0.1, 0.5, 1.0 and 5.0%. Eyes were washed with 300 ml distilled water either 5 min. or 24 hr after instillation. The eyes were examined with the aid of fluorescein at 1, 24, 48 and 72 hr and at 7 days, and eventually 14 and 21 days. Irritation indices were scored according to a modified Draize method.

Result: 5%/5 min.: extremely irritant and corrosive (1 rabbit)
1%/5 min.: irritant; 1%/24 hr: irritant (3 rabbits)
0.5%/24 hr: marginal (3 rabbits)
0.1%/24 hr: negative (3 rabbits).

Test condition: Strain: albino
Weight at study initiation: 2-3 kg
Vehicle: water
Test substance: 85% pure.
Reliability: (2) valid with restrictions
(2e) Study well documented, meets generally accepted scientific principles, acceptable for assessment.
Flag: Critical study for SIDS endpoint

Species: other
Concentration: .1 %
Dose: 
Exposure Time: 
Comment: 
Number of Animals: 
Result: highly irritating
EC classification: 
Method: other
Year: 

Test substance: no data
Method: Corneal endothelial cell cultures are obtained from human endothelial cell sheets of 2 mm² in 3 ml of RPMI 1640 tissue culture medium with 10% calf serum. After 2 weeks the endothelial cell outgrowth is ready to be transferred to multiwell dishes in which the toxicity tests are performed. The cell viability is quantified by a 51Cr-release assay. The cells are preincubated with 0.5 µCi per ml of 51Cr, as
sodium chromate, for one hr, during which time the cells take up 60000 to 200000 DPM of the isotope. Each monolayer is then washed 3 times. When cells are killed by the test substance, the isotopes release from the monolayer and distribute to the culture medium, which is measured in a gamma counter. A dose is estimated resulting in 50% maximal toxicity (ED50).

**Result:**
The ED50 of 0.013M (0.073%) found is said to correlate with "severe irritating" in the Draize test.

**Reliability:**
(3) invalid
(3c) Unsuitable test system.

19-DEC-2001 (22)

### 5.3 Sensitization

**Type:** Intracutaneous test  
**Species:** guinea pig  
**Concentration:**  
Induction .1 % intracutaneous  
Challenge .1 % intracutaneous  
**Number of Animals:** 15  
**Vehicle:** water  
**Result:** not sensitizing  
**Classification:** other  
**Method:** GLP: no  
**Year:**  
**Test substance:** other TS  
**Method:** A volume of 0.1 ml of a 0.1% solution of the test substance was injected intracutaneously to separate skin sites of the animals (5 males; 10 males used as controls), 3 times weekly for a total of 9 treatments. Two weeks after the last injection, a challenge dose of 0.1 ml was administered to both test and control animals in the same manner. Skin reactions were examined at 24, 48 and 72 hr following the challenge dose.  
**Test condition:** Strain: albino.  
Weight at study initiation: 300-400 g.  
Study type: non-adjuvant.  
**Test substance:** 85% pure  
**Reliability:**  
(2) valid with restrictions  
(2d) Test procedure in accordance with national standard methods with acceptable restrictions.

02-JAN-2002 (40)

### 5.4 Repeated Dose Toxicity

**Species:** rat  
**Sex:** male  
**Strain:** other  
**Route of admin.:** oral feed  
**Exposure period:** 2 years  
**Frequency of treatment:** daily  
**Post. obs. period:** none  
**Doses:** 0.25, 1 and 4% KCl; 110, 450 and 1800 mg/kg bw/day  
**Control Group:** yes, concurrent no treatment  
**NOAEL:** > 1800 mg/kg bw
Method: other  
Year:  
GLP: no data  
Test substance: no data  
Remark: Groups of 50 rats were exposed to KCl administered through the food. Strain of the rats: F344/Scl.  
Result: The only treatment related effect seen in rats fed about 110-1800 mg/kg bw/day was gastritis, an irritant effect. At the end of the 2 years experimental period, the survival rates were 48%, 64%, 58%, 84% for respectively the 0%, 0.25%, 1% and 4% KCl groups.  

Test substance: KCl (POTASSIUM CHLORIDE) STUDY!  
Reliability: (2) valid with restrictions  
(2e), Study well documented, meets generally accepted scientific principles, acceptable for assessment.  
21-DEC-2001  
Species: rat  
Sex: female  
Strain: Wistar  
Route of admin.: drinking water  
Exposure period: 105 days  
Frequency of treatment: daily  
Post. obs. period: 1 month  
Doses: 5250 mg/kg bw/day administrated as a 2.5% aqueous solution of KCl.  
Control Group: yes, concurrent no treatment  
Method: other  
Year:  
GLP: no  
Test substance: no data  
Remark: 14 experimental animals and 6 control rats were fed ad libitum on a diet (Purina laboratory chow) and a 2.5% aqueous solution of KCl as the sole source of fluid. At the end of the exposure period, 10 animals were terminated, the remaining 4 animals were supplied with tap water in place of KCl for one further month. At termination of the study, the hearth and kidneys were weighted. The adrenals and kidneys were prepared for histological studies which included detection of ascorbic acid, cholesterol and other lipids.  
Result: Mean hearth weight was significantly less and mean kidney weight was significantly higher than the control animals. Histological studies of the adrenals indicated hypertrophy in the glomerular zone. All changes seen were reversible in the control animals.  

Test substance: KCl (POTASSIUM CHLORIDE) STUDY!  
Reliability: (2) valid with restrictions  
(2e) Study well documented, meets generally accepted scientific principles, acceptable for assessment.  
21-DEC-2001  
Species: human  
Sex: female  
Strain:  
Route of admin.: oral feed  
Exposure period: 4 week placebo controlled crossover study.  
Frequency of treatment: daily.  
Post. obs.
**OECD SIDS POTASSIUM HYDROXIDE**

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**period:** 1 month  
**Doses:** 108 mg/kg bw/day (80 mmol KCl/day).  
**Control Group:** yes, concurrent no treatment  
**NOAEL:** > 108 mg/kg bw  
**Method:** other  
**Year:** GLP: no data  
**Test substance:** other TS  
**Remark:** 44 females aged 18-55 years were selected for the study on the basis of lower prevailing potassium intake. They were randomly allocated to one of two groups who took either 80 mmol/day of KCl (Slow-K, Ciba Geigy), or matching placebo for the first two 4-week treatment periods. The treatments were reversed during the second 4-week period. Blood pressure, heart rate, urinary volume, electrolytes and creatinine, were measured weekly during a screening period and the two 4-weeks treatment periods.

**Result:** No significant changes in blood pressure, hearth rate and body weight. Significant increases in both urinary and plasma potassium.

**Test substance:** KCl (POTASSIUM CHLORIDE) STUDY!  
**Reliability:** (2) valid with restrictions  
(2e) Study well documented, meets generally accepted scientific principles, acceptable for assessment.

**21-DEC-2001** (4)

**Species:** human  
**Sex:** female  
**Strain:**  
**Route of admin.:** oral feed  
**Exposure period:** 6 week placebo controlled crossover study.  
**Frequency of treatment:** daily  
**Post. obs. period:** 1 month  
**Doses:** 88 mg/kg bw/day (65 mmol KCl/day).  
**Control Group:** yes, concurrent no treatment  
**NOAEL:** > 88 mg/kg bw  
**Method:** other  
**Year:** GLP: no data  
**Test substance:** no data  
**Remark:** 32 hypertensive females aged 34-62 years were selected for the study. They were randomly allocated to one of two groups who took either 65 mmol/day KCl, or matching placebo, for a 6 weeks treatment period. The treatments were reversed after the 6 weeks period. Blood pressure, and urinary electrolytes (Na and K) and creatinine were measured weekly during the treatment period. Plasma Na and K and serum albumin, calcium and magnesium were measured at the 6th week only.

**Result:** Significant reduction in systolic and diastolic blood pressure, from 153/104 to 146/101. Significant increase in serum and urine potassium. Changes in blood pressure did not correlate with changes in serum or urine electrolytes. Analysis of the 95% confidence intervals in this and five other studies suggests that KCl supplementation lowers blood pressure, but that the change is small and within the confidence levels of all 6 trials.

**Test substance:** KCl (POTASSIUM CHLORIDE) STUDY!  
**Reliability:** (2) valid with restrictions  
(2e) Study well documented, meets generally accepted scientific principles, acceptable for assessment.
5.5 Genetic Toxicity 'in Vitro'

Type: Mammalian cell gene mutation assay
System of testing: Chinese hamster ovary cells CHO-K1
Concentration: 12 mM (pH 10.4)
Cytotoxic Conc.: With metabolic activation: 16 mM (pH 10.9); without metabolic activation: 20 mM (pH 11.5)
Metabolic activation: with and without
Result: ambiguous
Year: no data
GLP: no data
Test substance: no data
- Type: Mammalian cell gene mutation assay.
- System of testing: non bacterial.
- GLP: no.
- Year: no data.
- Cell type: Chinese hamster ovary cells CHO-K1.
- Metabolic activation: with and without.
  * Species and cell type: S9 mix derived from rat livers.
  * Quantity: 5% final.
  * Induction: pretreatment with phenobarbital and 5,6-benzoflavone.
- Concentrations tested: 0, 4, 8, 12, 16 and 20 mM.
- Statistical methods: no data.
Remark:
According to the authors, this genotoxic effect is due to the high non-physiological pH (same effect with NaOH at 16 mM, pH 10.8). At such high pH values, the clastogenic activity of S9 is increased, or new clastogens are induced by breakdown of the S9. Incubations at non-physiological pH might give false-positive responses, and this possibility must be considered in the evaluation of such results (Morita, 1989). Non-physiological environments can produce genotoxic effects in cultured mammalian cells (Brusick, D., 1986, Env. Mutagenesis, 8, 879-886; Brusick, D., 1987, Mutation Res., 189, 1-6). The pH causality is further proven by the fact that the normal intracellular concentration of potassium is of the order of 10 times higher: 145 mM in human cells (Marieb, E.N., 1992, Human Anatomy and Physiology, The Benjalin/Cummings Publishing Company Inc.). An high non-physiological pH is not relevant in human cells.
Result: - Result: ambiguous.
- Cytotoxic concentration:
  * With metabolic activation: 16 mM (pH 10.9).
  * Without metabolic activation: 20 mM (pH 11.5).
- Genotoxic effects:
  * With metabolic activation: positive (12 mM, pH 10.4).
  * Aberrant cells: 6% (all structural aberrations except gaps).
  For 200 cells scored:
  1 chromatid gap
  5 chromatid breaks
  1 chromosome break
  9 chromatid exchanges
  1 chromosome exchange (including dicentric and ring chromosomes).
  * Without metabolic activation: negative.
Test condition:  
- Test design  
  * Number of replicates: 2 (except 4 mM: no replication).  
  * Frequency of dosing: no data.  
  * Positive and negative control groups and treatment: only negative control groups.  
  * Number of metaphases analyzed: 100/group.  
- Solvent: no data.  
- Description of follow-up repeat study: no data.  
- Criteria for evaluating results: number of cells with chromosomal aberrations and type of aberrations.  

Reliability:  
(2) valid with restrictions  
(2b) Guideline study with acceptable restrictions.  

Flag:  
Critical study for SIDS endpoint

19-DEC-2001

Type:  
Ames test

System of testing:  
Salmonella typhimurium TA97 and TA102

Concentration:  
0, 0.01, 0.05, 0.1, 0.5 and 1 mg/plate

Cytotoxic Conc.:  
No data

Metabolic activation:  
with and without

Result:  
negative

Method:  
other

Year:  
GLP: no data

Test substance:  
no data

Method:  
- Method/guideline: no data.  
- Type: Ames test.  
- System of testing: bacterial.  
- GLP: no data.  
- Year: no data.  
- Species/strain: Salmonella typhimurium TA97 and TA102.  
- Metabolic activation:  
  * Species and cell type: S9 mix prepared from the liver homogenate of male SD rats (Jcl:SD).  
  * Quantity: S9 mix containing 10% S9 fraction, 50µl/plate.  
  * Induced or not induced: Aroclor 1254 induced rats.  
- Concentrations tested: 0.01, 0.05, 0.1, 0.5, 1 mg/plate.  
- Statistical methods: Kruskal-Wallis test and regression analysis.  

Result:  
- Cytotoxic concentration:  
  * With metabolic activation: no data.  
  * Without metabolic activation: no data.  
- Genotoxic effects:  
  With and without metabolic activation: negative.  
  No. revertants/plate in function of dose mg/plate:  
  mg/TA97-S9/TA97+S9/TA102-S9/TA102+S9  
  0/162/211/227/378  
  0.01/148/215/250/350  
  0.05/141/193/248/359  
  0.1/153/201/239/351  
  0.5/122/200/256/390  
  1/159/200/243/354

Test condition:  
- Test design:  
  * Number of replicates: 3.  
  * Frequency of dosing: no data.  
  * Negative control group: distilled water.  
  Positive control groups: 9-aminoacridine (DMSO), mitomycin C (DMSO) and 2-aminoanthracene (DMSO).  
  * Number of metaphases analyzed: no data.
- Solvent: distilled water.
- Description of follow up repeat study: no data.
- Criteria for evaluating results: No. of revertants/plate.

**Reliability:**

(2) valid with restrictions
(2b) Guideline study with acceptable restrictions.

**30-JAN-2002**

**Type:** Bacterial reverse mutation assay

**System of testing:** E. coli B/Sd - 4/1,3,4,5 and B/Sd - 4/3,4

**Concentration:** 0.00945 - 0.019%

**Cytotoxic Conc.:**

**Metabolic activation:** without

**Result:** ambiguous

**Method:** other

**Year:** GLP: no

**Test substance:** no data

**Method:** Test system with E. coli based on back mutation from streptomycin dependence to non-dependence. A sample of a bacterial suspension is added to the aqueous solution of the chemical and incubated at 37°C for either 3 or 24 hr. A suitable dilution is assayed by plating on streptomycin-agar plates and on streptomycin-free plates, which are incubated for 48 hr or 6 days respectively, and frequency of mutants is calculated.

**Remark:** No dose/response behaviour.

**Test condition:** Number of replicates: treated 8-10; control 4-6.

Negative control: distilled water.

Pre-incubation time: 24 hr at 37°C.

**Reliability:**

(3) invalid

(3b) Significant methodological deficiencies.

**07-JAN-2002**

**Type:** Ames test

**System of testing:** Salmonella typhimurium TA 1535, TA 1537, TA 1538, TA 98, TA 100.

**Concentration:**

**Cytotoxic Conc.:**

**Metabolic activation:** no data

**Result:** negative

**Method:** other

**Year:** GLP: no

**Test substance:** no data

**Test substance:** NaOH (SODIUM HYDROXIDE) STUDY !

**Reliability:**

(3) invalid

(3a) Documentation insufficient for assessment.

**28-DEC-2001**

**Type:** Bacterial reverse mutation assay

**System of testing:** S. typhimurium TA 100, TA 1535, TA 1537, TA 9.

**Concentration:** 0, 100, 333, 1000, 3333, 10000 µg/plate.

**Cytotoxic Conc.:**

**Metabolic activation:** with and without
## OECD SIDS POTASSIUM HYDROXIDE

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<table>
<thead>
<tr>
<th>Date</th>
<th>ID: 1310-58-3</th>
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</tr>
</thead>
</table>

**Result:** negative  
**Method:** other  
**Year:** no data  
**Test substance:** no data  
**Remark:** Salmonella preincubation assay (NTP modified standard plate incorporation assay).  
Activation system: S-9 fraction from the liver of Arochlor 254-induced male SD rats with a NADPH-generating system.  
No. replicates: 3 plates per dose level.

### DNA damage and repair assay

**Type:** DNA damage and repair assay  
**System of testing:** E. coli PQ37  
**Concentration:** 1 - 100,000 nM/ml  
**Cytotoxic Conc.:**  
**Metabolic activation:** without  
**Result:** negative  
**Method:** other  
**Year:** no data  
**Test substance:** no data  
**Remark:** SOS Chromotest (Institut Pasteur).  
No. replicates: 3 plates per dose level.

### Mammalian cell gene mutation assay

**Type:** Mammalian cell gene mutation assay  
**System of testing:** Mouse lymphoma cell (L5178Y), TK+/− heterozygote.  
**Concentration:** 0 - 5000 µg/ml.  
**Cytotoxic Conc.:**  
**Metabolic activation:** with and without  
**Result:** ambiguous  
**Method:** OECD Guide-line 476 "Genetic Toxicology: In vitro Mammalian Cell Gene Mutation Tests"  
**Year:** yes  
**Test substance:** no data  
**Remark:** Media: Fischer's liquid medium for leuemic cells supplemented with 10% horse serum.  
Result: Without metabolic activation: negative.  
With metabolic activation: positive.  
The positive results are seen only at high concentrations and have been attributed by the authors to the changed physical environment of the cells (increased osmotic pressure; K+ effects on sequestering of Mg++ ions required for chromatin integrity), rather than to a direct genotoxic effect.

**Test substance:** KC1 (POTASSIUM CHLORIDE) STUDY!  
**Reliability:** (1) valid without restriction  
(1a) GLP guideline study.

28-DEC-2001

(55)
| Type: Mammalian cell gene mutation assay |
| System of testing: Mouse lymphoma cell (L5178Y), TK+/- heterozygote. |
| Concentration: 0 - 5000 µg/ml. |
| Cytotoxic Conc.: |
| Metabolic activation: with and without |
| Result: ambiguous |
| Method: OECD Guide-line 476 "Genetic Toxicology: In vitro Mammalian Cell Gene Mutation Tests" |
| Year: GLP: yes |
| Test substance: no data |
| Remark: Media: Fischer's liquid medium for leucemic cells supplemented with 10% horse serum. |
| Result: Without metabolic activation: negative. |
| With metabolic activation: positive. |
| The positive results are seen only at high concentrations and have been attributed by the authors to the changed physical environment of the cells (increased osmotic pressure; K+ effects on sequestering of Mg++ ions required for chromatin integrity), rather than to a direct genotoxic effect. |
| Test substance: KCl (POTASSIUM CHLORIDE) STUDY! |
| Reliability: (1) valid without restriction |
| (1a) GLP guideline study. |

---

| Type: Mammalian cell gene mutation assay |
| System of testing: Chinese Hamster Ovary cells (CHO). |
| Concentration: 0, 70, 80, 90 mM. |
| Cytotoxic Conc.: |
| Metabolic activation: without |
| Result: ambiguous |
| Method: other |
| Year: GLP: no data |
| Test substance: no data |
| Result: A positive result was obtained at high concentration and has been attributed by the author to the high osmolality. |
| Test substance: KCl (POTASSIUM CHLORIDE) STUDY! |
| Reliability: (4) not assignable |
| (4b) Secondary literature. |

---

| Type: Mammalian cell gene mutation assay |
| System of testing: Chinese hamster V79 cells, Chinese hamster ovary cells (CHO). |
| Concentration: 0 - 300 mM. |
| Cytotoxic Conc.: Without metabolic activation: 100 mM (V79), 75 mM (CHO). |
| With metabolic activation: 37.5 mM (V79), 150 mM (CHO). |
| Metabolic activation: with and without |
| Result: ambiguous |
| Method: other |
| Year: GLP: no data |
| Test substance: other TS |
| Remark: No. replicates: at least 2 replicates per experiment. |
Result: Without metabolic activation: positive. With metabolic activation: positive. Following the authors, artifacts are to be considered in the explanation of the positive results.

Test substance: KCl (POTASSIUM CHLORIDE) STUDY! General purpose reagent grade from BDH, Milano, Italia, 99.5% pure.

Reliability: (2) valid with restrictions (2e) Study well documented, meets generally accepted scientific principles, acceptable for assessment.

28-DEC-2001

Type: Ames test
System of testing: Salmonella typhimurium TA 92, TA 94, TA 98, TA 100, TA 1535, TA 1537.
Concentration: Up to 10 mg/plate.
Cytotoxic Conc.: with
Metabolic activation: with
Result: negative
Method: other
Year: 28-DEC-2001
Test substance: other TS
Remark: Solvent: phosphate buffer. Metabolic activation: liver S-9 mix of Fischer rats, pretreated with polychlorinated biphenyls. No increase in the number of revertant colonies were detected in any S. typhimurium strain up to the maximum dose of 10 mg/plate.

Test substance: K2CO3 (POTASSIUM CARBONATE) STUDY.
Reliability: (2) valid with restrictions (2e) Study well documented, meets generally accepted scientific principles, acceptable for assessment.

28-DEC-2001

Type: Cytogenetic assay
System of testing: Chinese hamster fibroblasts (CHL cells).
Concentration: up to 1 mg/ml.
Cytotoxic Conc.: without
Metabolic activation: without
Result: negative
Method: other
Year: 28-DEC-2001
Test substance: other TS
Remark: Solvent: physiological saline. No increase in the incidence of chromosomal aberrations was observed after 24 or 48 hr of treatment.

Test substance: K2CO3 (POTASSIUM CARBONATE) STUDY!
Reliability: (2) valid with restrictions (2e) Study well documented, meets generally accepted scientific principles, acceptable for assessment.

28-DEC-2001
### 5.6 Genetic Toxicity 'in Vivo'

<table>
<thead>
<tr>
<th>Type</th>
<th>Micronucleus assay</th>
</tr>
</thead>
<tbody>
<tr>
<td>Species</td>
<td>mouse</td>
</tr>
<tr>
<td>Sex</td>
<td>male/female</td>
</tr>
<tr>
<td>Strain</td>
<td>CD-1</td>
</tr>
<tr>
<td>Route of admin.</td>
<td>i.p.</td>
</tr>
<tr>
<td>Doses</td>
<td>10 mg/kg of 15 mM NaOH</td>
</tr>
<tr>
<td>Result</td>
<td>negative</td>
</tr>
<tr>
<td>Method</td>
<td>other</td>
</tr>
<tr>
<td>Year</td>
<td>28-DEC-2001</td>
</tr>
<tr>
<td>GLP</td>
<td>no</td>
</tr>
<tr>
<td>Test substance</td>
<td>other TS</td>
</tr>
<tr>
<td>Method</td>
<td>The test compound was administered as</td>
</tr>
<tr>
<td></td>
<td>a single i.p. dose to treatment groups</td>
</tr>
<tr>
<td></td>
<td>(5 males and 5 females) for sacrifice</td>
</tr>
<tr>
<td></td>
<td>at 30, 48 and 72 hours. NaOH was used</td>
</tr>
<tr>
<td></td>
<td>as control substance.</td>
</tr>
<tr>
<td>Result</td>
<td>No significant increase of nuclei was</td>
</tr>
<tr>
<td></td>
<td>observed.</td>
</tr>
</tbody>
</table>

**Test substance:** NaOH (SODIUM HYDROXIDE) STUDY!

**Reliability:**
- (3) invalid
- (3a) Documentation insufficient for assessment.

### 5.7 Carcinogenicity

<table>
<thead>
<tr>
<th>Species</th>
<th>mouse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>male/female</td>
</tr>
<tr>
<td>Strain</td>
<td>no data</td>
</tr>
<tr>
<td>Route of admin.</td>
<td>dermal</td>
</tr>
<tr>
<td>Exposure period</td>
<td>25-46 weeks</td>
</tr>
<tr>
<td>Frequency of treatment</td>
<td>Initially: every 1-2 days; thereafter: twice weekly.</td>
</tr>
<tr>
<td>Doses</td>
<td>3-6% in water</td>
</tr>
<tr>
<td>Result</td>
<td>positive</td>
</tr>
<tr>
<td>Control Group</td>
<td>other</td>
</tr>
<tr>
<td>Method</td>
<td>other</td>
</tr>
</tbody>
</table>
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<table>
<thead>
<tr>
<th>Year:</th>
<th>GLP: no</th>
</tr>
</thead>
<tbody>
<tr>
<td>Test substance:</td>
<td>no data</td>
</tr>
<tr>
<td>Method:</td>
<td>The test substance was applied to the skin of four strains of mice (strains not reported), every 1-2 days until the first development of lesions and thereafter approx. twice weekly for 4-6 weeks from the time of the first appearance of papillary growths. The total period of application ranged from 25 to 46 weeks.</td>
</tr>
<tr>
<td>Remark:</td>
<td>Skin cancer is produced by long term KOH solution painting, by a non-genotoxic mechanism, secondary to repeated application and prolonged inflammation. This is a result of indirect hyperplasia as a consequence of severe skin damage (Ingram, A.J., and Grasso, P., (1991), Mutation Res., 248, 333-340). Any kind of prolonged irritation possibly would have produced the same result. No cancer nor sarcoma was developed in rats. HCl solution painting produced also cancer in mice.</td>
</tr>
<tr>
<td>Result:</td>
<td>Occurrence of cancer in KOH treated mice in 14% of males (4 out of 29) and 15% of females (8 out of 52). Tar control: 36% of males (5 out of 11) and 40% of females (4 out of 10).</td>
</tr>
<tr>
<td>Test condition:</td>
<td>Age: 1 to 6 months; Number of animals: 29 males and 52 females. Type of exposure: dorsum in the sacral region, without epilation. Clinical signs: alopecia in 2-4 weeks, pacydermia in ca. 10 weeks, sessile papillomatous growths in 4-5 months, horn masses, tumors in ca. 15% of the cases, no metastases, emaciation and cachexia.</td>
</tr>
<tr>
<td>Reliability:</td>
<td>(3) invalid</td>
</tr>
<tr>
<td></td>
<td>(3b) Significant methodological deficiencies.</td>
</tr>
<tr>
<td>19-DEC-2001</td>
<td>(58)</td>
</tr>
</tbody>
</table>

5.8 Toxicity to Reproduction

<table>
<thead>
<tr>
<th>Type:</th>
<th>One generation study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Species:</td>
<td>mouse</td>
</tr>
<tr>
<td>Sex:</td>
<td>female</td>
</tr>
<tr>
<td>Strain:</td>
<td>CD-1</td>
</tr>
<tr>
<td>Route of admin.:</td>
<td>gavage</td>
</tr>
<tr>
<td>Exposure Period:</td>
<td>10 days (day 6 to 15 of gestation).</td>
</tr>
<tr>
<td>Frequency of treatment:</td>
<td>single daily (intubation).</td>
</tr>
<tr>
<td>Premating Exposure Period</td>
<td></td>
</tr>
<tr>
<td>male:</td>
<td>none</td>
</tr>
<tr>
<td>female:</td>
<td>none</td>
</tr>
<tr>
<td>Duration of test:</td>
<td>17 days</td>
</tr>
<tr>
<td>Doses:</td>
<td>2.35, 10.9, 50.6, 235 mg/kg bw.</td>
</tr>
<tr>
<td>Control Group:</td>
<td>yes</td>
</tr>
<tr>
<td>NOAEL Parental:</td>
<td>&gt; 235 mg/kg bw</td>
</tr>
<tr>
<td>NOAEL F1 Offspr.:</td>
<td>&gt; 235 mg/kg bw</td>
</tr>
<tr>
<td>Method:</td>
<td>other</td>
</tr>
<tr>
<td>Year:</td>
<td>GLP: no</td>
</tr>
<tr>
<td>Test substance:</td>
<td>no data</td>
</tr>
<tr>
<td>Remark:</td>
<td>Groups of 21-24 experimental animals were used (virgin adult albino CD-1 outbred). Body weights were recorded on days 0, 6, 11, 15 and 17 of gestation. The mice were subjected to Caesarean section on gestation day 17. Post exposure observation period: 2 days.</td>
</tr>
</tbody>
</table>
Result: The administration of up to 235 mg/kg bw of KCl to pregnant mice for 10 consecutive days had no effect on nidation or on maternal or offspring survival. The number of abnormalities seen in either soft or skeletal tissues of the test group did not differ from the controls.

General parental/maternal toxicity: No effects seen in survival, total number of corpora lutea, implant sites, resorptions, soft tissue observations (urogenital tract), or live offspring.

Toxicity to offspring: No effects seen in survival, sex ratio, average offspring weight, external congenital abnormalities, soft tissue defects (cleft palate), or skeletal defects (sternebrae, ribs, vertebrae, skull and extremities).

Test substance: KCl (POTASSIUM CHLORIDE) STUDY!

Reliability: (2) valid with restrictions

Test procedure in accordance with national standard methods with acceptable restrictions.

Type: One generation study
Species: rat
Sex: female
Strain: Wistar
Route of admin.: gavage
Exposure Period: ten days (day 6 to 15 of gestation).
Frequency of treatment: single daily (oral intubation).
Premating Exposure Period
male: none
female: none
Duration of test: 20 days
Doses: 3.1, 14.4, 66.8, 310 mg/kg bw.
Control Group: yes
NOAEL Parental: > 310 mg/kg bw
NOAEL F1 Offspr.: > 310 mg/kg bw
Method: other

Year: GLP: no

Test substance: no data
Remark: Groups of 21-24 experimental animals were used (Wistar rats). Body weights were recorded on days 0, 6, 11, 15 and 20 of gestation. The rats were subjected to Caesarean section on gestation day 20. Post exposure observation period: 5 days.

Result: The administration of up to 310 mg/kg bw of KCl to pregnant rats for 10 consecutive days had no clear discernible effects on nidation or on maternal or offspring survival. The number of abnormalities seen in either soft or skeletal tissues of the test group did not differ from the controls.

General parental toxicity: No effects seen in survival, total number of corpora lutea, implant sites, resorptions, soft tissue observations (urogenital tract), or live offspring.

Toxicity to offspring: No effects seen in survival, sex ratio, average offspring weight, external congenital abnormalities, soft tissue defects (cleft palate), or skeletal defects (sternebrae, ribs, vertebrae, skull and extremities).
Test substance: KCl (POTASSIUM CHLORIDE) STUDY!
Reliability: (2) valid with restrictions
           (2d) Test procedure in accordance with national standard
                methods with acceptable restrictions.

21-DEC-2001

Type: One generation study
Species: mouse Sex: female
Strain: CD-1
Route of admin.: gavage
Exposure Period: day 6 to 15 of gestation.
Frequency of treatment: once daily.
Duration of test: 10 days.
Doses: 2.9, 13.5, 62.5 or 290.0 mg/kg bw.
Control Group: other: water.
other: NOAEL Teratogen. :
> 290 mg/kg bw
Method: other
Year: 1975 GLP: no data
Test substance: no data
Result: 22-25 mice/group.
One group of mice received the vehicle (water) as negative control, another group of mice received 150 mg/kg acetylsalicylic acid as positive control. The administration of up to 290 mg/kg of the test substance to pregnant mice for 10 consecutive days had no clearly discernible effect on nidation or on maternal or fetal survival. The number of abnormalities seen in either soft or skeletal tissues of the test groups did not differ from the number occurring spontaneously in vehicle treated controls. No treatment related effects in the positive control group (treated with acetylsalicylic acid) were observed.

Test substance: K2CO3 (POTASSIUM CARBONATE) STUDY!
Reliability: (2) valid with restrictions
           (2d) Test procedure in accordance with national standard
                methods with acceptable restrictions.

03-JAN-2002

Type: One generation study
Species: rat Sex: female
Strain: Wistar
Route of admin.: gavage
Exposure Period: day 6 to 15 of gestation.
Frequency of treatment: once daily.
Duration of test: 10 days.
Doses: 1.8, 8.4, 38.8 or 180.0 mg/kg.
Control Group: other: water.
other: NOAEL teratogen. :
> 180 mg/kg bw
Method: other
Year: 1975 GLP: no data
Test substance: no data
Result: 23-25 rats/group.
One group of rats received the vehicle (water) as negative control, another group of rats received 250 mg/kg acetylsalicylic acid as positive control. The administration of up to 180 mg/kg of the test substance to pregnant rats for 10 consecutive days had no clearly discernible effect on
nidadation or on maternal or fetal survival. The number of abnormalities seen in either soft or skeletal tissues of the test groups did not differ from the number occurring spontaneously in the vehicle treated controls. In the positive control group (treated with acetylsalicylic acid) treatment related effects (increased number of resorptions, decreased average fetus weight, incomplete ossification, missing sternebrae, incomplete closure of the skull, etc...) were observed.

Test substance: K$_2$CO$_3$ (POTASSIUM CARBONATE) STUDY!
Reliability: (2) valid with restrictions
(2d) Test procedure in accordance with national standard methods with acceptable restrictions.

5.9 Developmental Toxicity/Teratogenicity

5.10 Other Relevant Information

Type: other
Result: Skin and ocular burns; chemical injury.

The mechanism of injury by alkali is by saponification of fat, which causes fatty tissue to lose its function with increased damage due to heat reaction; extraction of considerable water from cells due to the hygroscopic nature of the alkali; and dissolution of proteins, permitting so deeper penetration of OH- ions and further chemical reactions. Ocular damage is most significant around pH 11-11.5. The alkali penetrates quickly, saponify plasma membranes, denatures collagen, and causes vascular thromboses in the conjunctiva, the episclera, and even the anterior uvea. The sequelae of corneal burns include scarring and opacification of the cornea with resultant loss of visual acuity, corneal neovascularization, ulcer formation, and perforation. Other sequelae of untreated or very severe alkali burns include epithelial erosions, secondary glaucoma, progressive cicatrization which occludes the ducts of main and accessory lacrimal glands and causes destruction of conjunctival goblet cells so as to cause dry eyes, cicatricial entropion, and trichiasis.

Type: other
Result: Gastrointestinal burns; chemical injury.

The mechanism of injury is one of liquefactive necrosis. Thrombosis of local blood vessels contributes to tissue damage. Transmural necrosis can occur with frightening rapidity and injury often extrudes through the esophagus to involve adjacent mediastinal and peritoneal structures. When the alkali enters the stomach, there may be some neutralization by gastric acid, which can limit the injury to this organ. Perforation of the stomach can occur with peritonitis and caustic injury to the contiguous organs including the colon, pancreas, liver and spleen. If sufficient quantities of alkali pass through the pylorus, there may be substantial duodenal damage including perforation. Lye constitutes a greater danger than solid granules, which tend to adhere on contact to mucous
membranes without traveling further. The severity of damage depends on concentration of the agent, but also on the quantity swallowed. Aspiration of the alkali into the airway can result in live-threatening injuries to the larynx, the tracheobronchial passages, and the lungs. There are three phases of injury and healing to the esophagus in animals. The acute phases, from about day 1 to 4, is that of liquefactive necrosis. During the subacute phase, from day 4 to 14, there is sloughing of the necrotic area; the esophageal wall appears thinnest and most vulnerable. About day 15 begins the cicatrization phase with eventual esophageal striitures resulting from collagen contraction. Reepithelialization is complete by 4 weeks to 3 months.

07-MAY-2001

Type: other
Result: Sensory irritation.
The irritant effects are listed as coughing, wheezing, conjunctivitis, tearing, irritation and alterations in general well being, which is relevant because workers distracted by primary irritants are more likely to have accidents and endanger themselves and others.

07-MAY-2001

5.11 Experience with Human Exposure

Memo: A three year survey of accidents and dangerous occurrences in the UK chemical industry.
Result: In an analysis of 2100 accidents and dangerous occurrences which occurred in the UK chemical industry between January 1982 and March 1985, 32 involved caustic soda/potash.

23-JAN-2001

Memo: Acute poisoning due to alkalis used during industrial cleaning of soft drinks' glass containers: a case report.
Result: A young woman suffered severe burns in the esophagus after consuming a soft drink contaminated by an industrial cleaning agent used to clean the non-disposable bottles. The "lemonade" had a pH of 13.3, a total alkalinity of 1.57N, a sodium content of 1.75 mole/l and a potassium content of 1.15 mole/l.

23-JAN-2001

Memo: Button batteries: letting the skeleton out of our closet.
Result: The hypothesis is presented that burns produced by ingested button batteries with residual EMF, could be due, not by its KOH release, but by an hydrolysis process producing locally a high pH (12).

23-JAN-2001

Memo: Button battery ingestion: a review.
Result: The potential for corrosive alkali injury from batteries is in fact dependent on their electrical properties, by the progressive electrolysis of the battery casing, in the area of the seal. Moreover, it is thought that burns to the esophagus could be due to the low-voltage DC producing an electrolysis with an increase of pH. Battery ingestions in the UK and the USA are reviewed.
Caustic alkali ingestions by farm children.

Children are particularly exposed to commercial cleaning products containing KOH or NaOH, including, in the case of farm children, to high concentration dairy pipeline cleaners (8 to 25%). In this study, 43 children were admitted from 1973 to 1983 to four rural Wisconsin (USA) hospitals, after ingestion of caustic products. Farm products constituted 23% of all products and 43% of all drain/pipe cleaners ingested.

Chemical burn from alkaline batteries - a case report.

A 2-year old male was found to have a third degree (full thickness) burn on his right thigh due to exposure to the contents of leaking alkaline batteries.


KOH is "GRAS" (Generally Recognized As Safe) as stated by the FDA (USA).

Potassium hydroxide is a food additive, listed as E525 in Annex 1 of Directive 95/2/EU. This means that KOH is a general food additive to be used following the "quantum satis" principle (as much as necessary according to GMP).

Fatal complication from an alkaline battery foreign body in the esophagus.

The fatal complications from an alkaline battery foreign body (containing potassium hydroxide 45%) in the esophagus of a 2.5 year old male, resulting in corrosive burns of the esophagus, necrosis, perforation, communication between the esophagus and the trachea and subsequent death, is described.

Gastric outlet obstruction due to corrosive ingestion: incidence and outcome.

In a retrospective clinical study with 168 children after alkaline substance ingestion, 9 children (5.3%) developed gastric outlet obstruction. After an appropriate treatment, all patients are alive without any complaints.

Ingestion of strong corrosive alkalis: spectrum of injury to upper gastrointestinal tract and natural history.

In a study of liquid caustic ingestion on 31 patients in India, 3 ingested potassium hydroxide (9.7%) and 28 ingested sodium hydroxide (90.3%). The degree and extent of burns with respect to type of alkalis were not noticeably different. There was a poor correlation between the presence or absence or severity, of oropharyngeal burns on one hand and the presence or absence or severity of lesions in the UGIT on the other hand. Eight patients (25.8%) who showed involvement of the UGIT had a normal oropharynx. Two patients had perforation on admission and in three more it occurred on the 9th, 11th and 14th days after ingestion. All patients suffered esophageal injury, 29 (93.5%) gastric injury and 8 (25.8%) duodenal injury.
23-JAN-2001

Memo: Liquid caustic ingestion; spectrum of injury.
Result: A woman who ingested 20 g of KOH in aqueous solution suffered glossopharyngalgia and oral pharyngeal burns.

23-JAN-2001

Memo: New approach to management of esophageal injuries caused by ingestion of potent liquid alkali.
Result: The simultaneous admission of 9 youths in a medical center following their ingestion of concentrated KOH, mistaken for wine, resulted in the following observations. Three patients with second-degree oral burns required no surgery. Six patients required laparotomy with gastrostomy and/or chimney feeding jejunostomy, one required immediate esophagogastrctomy, and 3 required immediate total or subtotal gastrectomy. There were no deaths. Three patients have required esophageal replacement and 3 others have required repeated dilatations. At 2-year follow-up, all 9 maintain their nutritional status orally, and all can phonate.

23-JAN-2001

Memo: Perforation of nasal septum due to button battery lodging in nose.
Result: A 4-year old boy who had a button battery lodged in his nose for appr. 24 hrs had local tissue corrosion, with a small perforation, caused presumably by the 25% KOH electrolyte.

23-JAN-2001

Memo: Prompt irrigation of chemical eye injuries may avert severe damage.
Result: Potassium hydroxide is an infrequent cause of injury, but can result in severe damage. Potassium hydroxide penetrates eye tissue nearly as rapidly as sodium hydroxide and thus causes injuries of the same severity.

23-JAN-2001

Result: Treatment of 32 children suffering of Molluscum contagiosum (a viral skin infection) with a topical 10% KOH aqueous solution, twice daily, during a period of 30 days, resulted in clearance of all lesions. The only side effects observed in 12 children were: severe stinging, transitory hypopigmentation, persistent hypo and hyperpigmentation, hypertrophic scar and secondary infection.
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7.1 End Point Summary

7.2 Hazard Summary

7.3 Risk Assessment