# Lithium hydroxide: Human health tier II assessment

**13 February 2015**



## Chemicals in this assessment

| Chemical Name in the Inventory | CAS Number |
|---|---|
| **Lithium hydroxide (Li(OH))** | 1310-65-2 |
| **Lithium hydroxide, monohydrate** | 1310-66-3 |

## Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted and published separately, using information available at the time, and may be undertaken at different tiers.

This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

For more detail on this program please visit:**www.nicnas.gov.au**

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ACRONYMS & ABBREVIATIONS

# Grouping Rationale

The chemicals in this group are lithium compounds containing an alkali metal cation (lithium) and the hydroxide anion (HO-) and are a class of 'alkali metal hydroxides'. The group contains two chemicals: lithium hydroxide anhydrous (CAS No. 1310-65-2) and lithium hydroxide monohydrate (CAS No. 1310-66-3) with a molecule of water of hydration.

While the hydroxide anion (HO-) of chemicals in this group is responsible for the local effects following application, the lithium (alkali metal cation) ion is the toxicologically relevant moiety responsible for the long-term effects. Therefore, as toxicokinetics and the toxicity of chemicals in this group are expected to be similar and will be driven predominantly by the lithium cation (systemic) and hydroxide anion (OH-) (local), they are grouped together for human health risk assessment (HSDB; REACH a; REACH b).

The chemicals in this group have similar reported uses, and, as hydrates are regarded as a mixture of the anhydrous chemical and water, the hydrates therefore, are not required to be listed on the Australian Inventory of Chemical Substances (AICS). If the anhydrous form is listed, it is valid to use the CAS No. for anhydrous lithium hydroxide (CAS No. 1310-65-2) for the hydrates.

# Import, Manufacture and Use

## Australian

The following Australian industrial uses were reported under previous mandatory and/or voluntary calls for information.

Lithium hydroxide monohydrate (CAS No. 1310-66-3) has reported commercial uses including in lubricants and additives.

Lithium hydroxide monohydrate (CAS No. 1310-66-3) has reported site-limited uses including as a complexing agent.

The total volume introduced into Australia, reported under previous mandatory and/or voluntary calls for information, was between 100 and 1000 tonnes.

### International

The following international uses have been identified through:

- the European Union (EU) Registration, Evaluation and Authorisation of Chemicals (REACH) dossiers;
- Galleria Chemica;
- the Substances and Preparations in the Nordic countries (SPIN) database; and
- the United States (US) National Library of Medicine's Hazardous Substances Data Bank (HSDB).

Lithium hydroxide anhydrous (CAS No. 1310-65-2) has reported cosmetic uses in:

- buffering; and
- hair waving or straightening.

Lithium hydroxide anhydrous (CAS No. 1310-65-2) has reported cosmetic uses in the United States of America (USA), with reported use in 16 products (Personal Care Products Council, 2011).

The chemicals in this group have reported domestic uses in:

- adhesives (binding agents);
- paints, lacquers and varnishes; and
- fillers.

The chemicals in this group have not been reported as present in any domestic products in the USA (Household Products Database, US Department of Health and Human Services).

Lithium hydroxide monohydrate (CAS No. 1310-66-3) has reported commercial uses including:

- in lubricants and additives;
- in production of lithium greases;
- in breathing gas purification systems for spacecraft, submarines etc. to remove carbon dioxide from exhaled gas;
- as a heat transfer medium; and
- as a storage-battery electrolyte.

The chemicals in this group have reported site-limited use including as intermediates.

Lithium hydroxide monohydrate (CAS No. 1310-66-3) has been identified as having a non-industrial use in pharmaceuticals.

## Restrictions

### Australian

No known restrictions have been identified.

### International

Lithium hydroxide monohydrate (CAS No. 1310-66-3) is listed on the following (Galleria Chemica):

- EU Regulation (EC) No 1223/2009 of the European Parliament and of the Council of 30 November 2009 on cosmetic products—Annex III—List of substances which cosmetic products must not contain except subject to the restrictions laid down;
- New Zealand Cosmetic Products Group Standard—Schedule 5—Table 1: Components cosmetic products must not contain except subject to the restrictions and conditions laid down; and
- The Association of Southeast Asian Nations (ASEAN) Cosmetic Directive Annex III Part 1—List of substances which cosmetic products must not contain except subject to restrictions and conditions laid down.

Restrictions for certain types of cosmetic products according to the European Commission Cosmetics Directive Annex III (List of restricted substances) and New Zealand Cosmetic Products Group Standard are:

- the maximum concentration in cosmetic products for hair straightener is 2 % for general use and 4.5 % for professional use;
- pH value not to exceed pH 12.7 for depilatory action; and
- pH value not to exceed pH 11 for other uses as pH adjusters (rinse-off products only).

# Existing Worker Health and Safety Controls

## Hazard Classification

The chemicals are not listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia).

## Exposure Standards

### Australian

No specific exposure standards are available.

### International

The following exposure standards are identified for lithium hydroxide monohydrate (CAS No. 1310-66-3).

An exposure limit of 1 mg/m$^3$ short-term exposure limit (STEL) in Canada, Ireland, South Africa, and the United Kingdom.

# Health Hazard Information

The chemicals in this group are lithium compounds containing the lithium cation and the hydroxide anion ($HO^-$). While the hydroxide anion ($HO^-$) is responsible for the local effects following application, lithium is the toxicologically relevant moiety responsible for the long-term effects.

Limited data are available for specific long-term toxicological endpoints for the chemicals in this group. The long-term toxicity of these chemicals is expected to be similar and will be driven predominantly by lithium; therefore, the data obtained from studies with other lithium compounds have been read across for specific long-term toxicological endpoints for human health risk assessment. Local effects will arise from the hydroxide anion ($HO^-$) functionality and will be specific for the chemicals of this group (see **Grouping rationale**).

## Toxicokinetics

The toxicokinetics of these chemicals mainly focuses on lithium as lithium is the toxicologically relevant moiety with respect to absorption, distribution, metabolism, and excretion. As lithium has been used as a psychiatric drug for almost half a century, a number of publications on lithium pharmacokinetics is available in humans.

The chemicals in this group are strong alkaline substances that dissociate completely in water and form lithium ions and hydroxide ions resulting in high pH solutions. The hydroxide ion will react with free H+ or any acidic species that may be present.

Lithium is absorbed readily and almost completely from the gastrointestinal tract and distributed throughout the body water fraction following absorption. Complete absorption occurs in about eight hours, with the peak plasma concentration occurring 2–4 hours after an oral dose. Although lithium is rapidly taken into the kidneys, transfer to the liver, bone muscle or the brain is slower. Lithium crosses the placenta and is excreted in breast milk. Lithium is not metabolised to any appreciable extent in the human body and excreted almost unchanged completely via the urine. Excretion of lithium is fast with >50 % and >90% excreted within 24 and 48 hours, respectively. As excretion of the lithium is fast, bioaccumulation can be excluded.

Dermal absorption of lithium is considered to be very low and almost negligible in the case of non-corrosive solutions; however, as corrosive substances can cause skin and tissue damage, the lithium ion can be easily absorbed under these situations and become systemically available.

Exposure to lithium via vapours is not expected to be toxicologically relevant due to the negligible low vapour pressure of chemicals in this group. Similarly, resorption and bioavailability of lithium from non-corrosive aerosols is also expected to be low due to the very low log Kow (HSDB; REACH a).

## Acute Toxicity

### Oral

The chemicals in this group have moderate acute toxicity following oral exposure in animal tests. The information is sufficient to support classification (refer to **Recommendation** section).

The reported oral median lethal dose (LD50) was 210–280 mg/kg bw for male/female rats for lithium hydroxide anhydrous (CAS No. 1310-65-2) and 368–491 mg/kg bw for female/male rats for lithium hydroxide monohydrate (CAS No. 1310-66-3). The LD50 for lithium hydroxide monohydrate (CAS No. 1310-66-3) was calculated based on the LD50 of lithium hydroxide anhydrous (CAS No. 1310-65-2). It is also noted that the LD50 values represents a sum of local tissue damage and systemic effects due to the corrosivity of these chemicals (HSDB; REACH b).

### Dermal

As the dermal absorption of lithium is considered to be very low and almost negligible in the case of non-corrosive solutions, this group of chemicals, at non-corrosive concentrations, is likely to have low acute toxicity following dermal exposure. However, as corrosive chemicals can cause skin and tissue damage and can be easily absorbed under these circumstances and become systemically available, these chemicals at corrosive concentrations are likely to be of high acute toxicity following dermal exposure (see **Toxicokinetics** and **Corrosivity**).

Therefore, the dermal LD50 values across this group are likely to be dependent on the concentration at which the chemicals are applied. The observed lethality of corrosive chemicals is not likely as a result of systemic toxicity through percutaneously absorbed material, but consistent with local skin and tissues damage with resulting loss of blood (REACH b).

The dermal LD50 for lithium hydroxide monohydrate (CAS No. 1310-66-3) in rabbits was determined to be between 200 and 3000 mg/kg bw following dermal application for 24 hours. While no mortality occurred at the 200 mg/kg bw, all animals (4/4) died at 3000 mg/kg bw. The test material was extremely corrosive. Skin changes at 24 hours were characterised by total destruction of the skin and partial destruction of the skeletal muscle tissue. Extreme haemorrhaging was also noted at the exposure sites

with large amounts of free blood under the occlusive sleeves. The death of all the rabbits in the 3000 mg/kg bw dose group was considered to be due to the local tissue toxicity of the chemical with the resulting loss of blood. Necrosis was also observed at the exposure sites of the 200 mg/kg animals at seven and 14 days.

Due to the corrosive nature of the chemical and limited dose groups, an appropriate median LD50 for lithium hydroxide monohydrate (CAS No. 1310-66-3) could not be established following dermal exposure. Similar results are also expected for lithium hydroxide (CAS No. 1310-65-2) (REACH b).

### Inhalation

The limited information indicates that the chemicals in this group are likely to be of low acute toxicity in animal tests following inhalation exposure.

In a nose-only acute inhalation exposure study, the four-hour LC50 for lithium hydroxide monohydrate (CAS No. 1310-66-3) was determined to be >6.15 mg/L (>3.4 mg/L for lithium hydroxide—CAS No. 1310-65-2) in rats (five/sex/dose). Respiratory difficulties were noted in two animals with severe necrosis of the snout as a result of exposure. These animals were in moribund condition and were sacrificed on study day five after exposure. Treatment-related clinical signs observed during the study included abdominogenital staining, alopecia on the head and/or neck, ataxia, chromodacryorrhoea, chromorhinorrhoea, decreased faeces, decreased locomotion, diarrhoea, dyspnoea, lacrimation, necrotic snout, oral discharge, rales, squinting eyes, swollen snout, and unthriftiness. Similar results are also expected for lithium hydroxide anhydrous (CAS No. 1310-65-2) (REACH b).

### Observation in humans

Acute intoxication of lithium can occur either in the initial phase in a course of therapy, but also at any point of time during long-term treatment or after an acute overdose. Signs of toxicity vary with the plasma levels of lithium and serious toxic symptoms occur at plasma levels above 2.5 mmol/L lithium. These are fasciculations, muscle contractions, hyperreflexia and hypertonia, drowsiness, confusion, sometimes epileptiform insults, hypotension, coma, and collapse. Changes in the electrocardiogram (ECG) and in the extracorporeal circuit (EEC) have also been noted without being dependent on the plasma levels of lithium (HSDB).

Clinical intoxication by lithium is also common as a result of attempted suicide or erroneous or misunderstood prescriptions. Symptoms and signs of nervous system effects noted with even few milliequivalents (mEq) in the plasma are anorexia, nausea, muscle twitches, apathy, mental confusion, tremor, blurring of vision, coma and death (HSDB).

The major target organ for lithium overdose is the central nervous system, with residual effects in 10 % of cases. A probable oral lethal dose of 0.5 to 5 g/kg of lithium has been reported for a 70 kg human. Various reported cases of poisoning in humans include a 45-year-old man died after an acute ingestion of 90 sustained-release lithium tablets (450 mg each) with a peak level of 6.9 mEq/L despite haemodialysis; a 28-year-old man survived an acute ingestion with a lithium level of 10 mEq/L; and an adult recovered after an acute ingestion of 84 grams (210 tablets of 400 mg) of lithium. Accidental ingestions of an average of two pills typically causes drowsiness, while neurotoxicity has resulted after chronic therapy of 40 mg/kg/day. Mortality due to lithium as a single exposure is rare if recognised quickly and treated aggressively (HSDB).

## Corrosion / Irritation

## Corrosivity

The chemicals in this group were found to be corrosive substances when tested in animal studies. While the appropriate data are limited, the available information was sufficiently strong to support classification (refer to **Recommendation** section).

Although data on ocular exposure are not available due to the corrosive nature of these chemicals, corrosive chemicals are also considered to cause irreversible effects on the eyes.

Lithium hydroxide monohydrate (CAS No. 1310-66-3) was found to be corrosive in an in vitro study (Corrositex TM Assay) for determining the corrosive potential of chemical substances. Lithium hydroxide anhydrous (CAS No. 1310-65-2) was also found to be corrosive in the same assay (REACH b).

## Sensitisation

### Skin Sensitisation

Data are not available on the skin sensitisation potential of the chemicals in this group. However, information available on another lithium compound (lithium carbonate—CAS No. 554-13-2) indicates that these chemicals are not likely to be skin sensitisers.

Lithium carbonate (CAS No. 554-13-2) tested negative in a skin sensitisation test (Buehler test) conducted according to OECD Test Guideline (TG) 406. Dunkin-Hartley guinea-pigs (10/sex) were exposed topically to the undiluted chemical (0.30 g/animal) (six hours/day, once every seven days for three weeks). Animals were challenged topically with the chemical 14 days after the last induction at a new skin site. An additional five male and five female naive animals received 0.30 g of the undiluted test material (challenge control group). No skin reactions were noted on any of the test or challenge control animals at any time during the study (REACH b).

## Repeated Dose Toxicity

### Oral

Although limited data are available on repeated dose toxicity studies in animals for chemicals in this group, the available human data indicate that repeated oral exposure to these chemicals at therapeutic levels is not considered to cause serious damage to human health.

The active moiety for the chemicals in this group with toxicological consequences is the lithium ion. Lithium compounds have been used as a psychiatric drug in humans for long-term bipolar treatment for almost half a century. The recommended dose for this condition in humans is 450 to 900 mg/day lithium carbonate (CAS No. 554-13-2), equivalent to 84 to 169 mg lithium/day. As this dose has not resulted in any concern from its long-term use, it has been used to calculate the no observed adverse effect level (NOAEL) for repeated dose toxicity through oral exposure.

The NOAELs of 4.13 mg/kg bw/day and 7.24 mg/kg bw/day have been calculated for lithium hydroxide anhydrous (CAS No. 1310-65-2) and lithium hydroxide monohydrate (CAS No. 1310-66-3), respectively, for long-term oral toxicity of lithium in humans (REACH a).

In a repeated dose toxicity study, lithium chloride (CAS No. 7447-41-8) was administered to rats (strain unspecified) in drinking water at 20 mmol/L and 50 mmol/ L for two years. The 20 mmol/L concentration had no effects on the behaviour and health of the animals, except for slight and transitory initial disturbances. The plasma levels at this dose were 1.5 to 2 mmol lithium. The 50 mmol/L concentration affected the animals in various ways including decreased food and water intake within a few days of the dosing; progressive drowsiness and asocial behaviour from 3–5 days; difficulty in rousing and, when roused, the gait was staggering and hesitant and the animals rested again as soon as possible. They showed fine muscular tremor and trembling when resting; and became unresponsive and stuporous for a few days later then deterioration progressed to death within two to three weeks. The plasma levels at these doses were 3 mmol lithium (behavioural changes) to 8 mmol lithium (death).

The lithium concentration of 20 mmol/L used in this study is comparable to the highest doses given temporarily to hospitalised patients (plasma lithium level about 2 mmol/L). As a worst case scenario, the daily intake of lithium in rats is 2 mmol/kg bw/day (equivalent to 13.9 lithium mg/kg bw/ day). Therefore, a worst case NOAEL of 13.9 mg lithium/kg bw/ day can be derived in rats. This NOAEL is equivalent to 48 mg/kg bw/day lithium hydroxide anhydrous (CAS No. 1310-65-2) and 84 mg/kg bw/day lithium hydroxide monohydrate (CAS No. 1310-66-3) (REACH a).

### Dermal

No data are available.

However, it is noted that the dermal absorption of lithium is considered to be very low and almost negligible in the case of non-corrosive solutions. As corrosive substances can cause skin and tissue damage, chemicals can be easily absorbed under these situations and become systemically available (see **Toxicokinetics** and **Corrosivity**).

### Inhalation

No data are available.

However, it is noted here that the exposure to lithium via vapours is not expected to be toxicologically relevant due to the negligible low vapour pressure of chemicals in this group. Similarly, resorption and bioavailability of lithium from non-corrosive aerosols is also expected to be low due to the very low log Kow (see **Toxicokinetics).**

### Observation in humans

Reports of toxic responses in workers are rare despite the widespread use of lithium and its compounds. Occupational exposure to lithium compounds can occur through inhalation and dermal contact at workplaces where lithium compounds are produced or used. Some of these workplaces include extracting lithium from its ores, preparing various lithium compounds, welding, brazing, enamelling, and using lithium hydrides.

The general public could be exposed to small amounts of lithium via inhaling ambient air and ingesting food and drinking water, as lithium is found in various environmental media (HSDB).

## Genotoxicity

Limited information indicates that the chemicals in this group are not considered to have mutagenic or genotoxic potential.

Lithium hydroxide anhydrous (CAS No. 1310-65-2) was not mutagenic in the *Salmonella typhimurium* reverse mutation assay (TA 1535, TA 1537, TA 100 and TA 98) and in the *Escherichia coli* reverse mutation assay (WP2uvrA). Lithium hydroxide anhydrous (CAS No. 1310-65-2) was also concluded not to be clastogenic for inducing chromosome aberrations in cultured peripheral human lymphocytes. Lithium hydroxide monohydrate (CAS No. 1310-66-3) also tested negative for mutations and chromosomal aberrations in an in vitro mammalian cell mutagenicity assay performed with mouse lymphoma L5178Y TK ± cells (REACH a).

In vitro and in vivo studies have been used to test various lithium salts. While several studies have reported the genotoxic effects of various lithium compounds at high doses (equivalent to therapeutic doses or higher), many other studies have failed to demonstrate an effect. Considering the chemical properties of the lithium compounds, the Nordic Expert Group stated that it is unlikely that they act as direct mutagens. A secondary effect of increased cell survival caused by inhibition of glycogen synthase kinase 3 (GSK3) by lithium was provided as an explanation of the apparent genotoxicity noted (REACH a).

Patients on lithium therapy did not show an increase in the frequency of sister chromatid exchanges (SCE) in lymphocytes. No morphological evidence of chromosomal damage was noted. No elevation in the frequency of SCEs was found in another study with patients on lithium therapy (HSDB).

## Carcinogenicity

No data are available.

## Reproductive and Developmental Toxicity

Although no data are available for the chemicals in this group in animal studies, information available in humans and also on a similar chemical indicate that the chemicals in this group are not likely to have specific reproductive or developmental toxicity. Any reproductive and developmental effects were only observed secondary to maternal toxicity.

It has been suggested that the developing vascular system could be a target for lithium. Using lithium in psychiatric medicine has been reported to cause congenital defects, especially of the cardiovascular system, such as Ebstein's anomaly (a rare cardiac defect), when given to women during the first trimester of pregnancy. However, more thorough analysis of the records of mothers who received lithium treatment during the first trimester or the entire pregnancy and a multi-centre study of pregnancy outcome after therapeutic lithium exposure during the first trimester did not show any significant teratogenic risk. Similarly, others have also concluded that lithium given in therapeutic doses is not teratogenic (Aral & Vecchio-Sadus, 2008).

In a controlled and prospective study conducted in 138 women, lithium carbonate (CAS No. 554-13-2) was administered at a mean daily dose of 927 mg for treating major affective disorders during the first trimester of pregnancy. Rates of major congenital malformations did not differ between the lithium (2.8 %) and control (2.4 %) groups. This conclusion was further supported in a study where lithium carbonate (CAS No. 554-13-2) was not found to cause any developmental/teratogenic effects in 148 women using lithium during the first trimester of pregnancy. The mean daily dose of lithium was 927 mg/person. Rates of major congenital malformations did not differ between the lithium (2.8 %) and the control (2.4 %) groups. Infants of lithium-treated mothers had a significantly higher birthweight than the controls, despite identical gestational ages (HSDB).

In a reproductive toxicity study, the effect of prolonged subtoxic lithium ingestion (lithium chloride—CAS No. 7447-41-8) on pregnancy was conducted in a group of 52 test and 100 controls rats (albino Wistar). The animals were administered the chemical in a concentration of 20 mmol/L in drinking water, which produced plasma lithium levels of 1.5–2.0 mmol/L. The lithium concentration of 20 mmol/L used in this study is comparable to the highest doses given temporarily to hospitalised patients (plasma lithium level about 2 mmol/L). There were no differences in size and weight, or any other toxic effects, among treated and untreated controls. Lithium treated animals did not show any toxic signs or behaviour changes and malformations or other defects in the lithium-exposed litters. The daily intake of lithium in rats was 2 mmol/kg bw/day (equivalent to 13.9 lithium mg/kg bw/day), and a NOAEL of 13.9 mg lithium/kg bw/day can be derived. This NOAEL is equivalent to 48 and 84 mg/kg bw/day lithium hydroxide anhydrous (CAS No. 1310-65-2) and lithium hydroxide monohydrate (CAS No. 1310-66-3), respectively (REACH a).

In a developmental toxicity study, lithium carbonate (CAS No. 554-13-2) was administered (by gavage) to 25 pregnant Crl:Cd (Sprague Dawley—SD) rats on gestation days (GD) 6–19 at doses of 10, 30, 90 mg/kg bw/day. The NOAEL for maternal toxicity was established as 30 mg/kg bw/day, based on slight but significant reductions in the net weight change, the food intake and piloerection in a few dams. There was no chemical-related increase in the incidence of foetal malformations, external or internal, skeletal or soft tissue variations, or skeletal retardations. The NOAEL for developmental (foetal) toxicity was established as >90 mg/kg bw/day. These NOAELs are equivalent to a maternal NOAEL of 19.5 mg/kg bw/day of lithium hydroxide anhydrous (CAS No. 1310-65-2); foetal NOAEL of 58.3 mg/kg bw/day of lithium hydroxide anhydrous (CAS No. 1310-65-2); maternal NOAEL 34.1 mg/kg bw/day of lithium hydroxide monohydrate (CAS No. 1310-66-3); and foetal NOAEL of 102 mg/kg bw/day of lithium hydroxide monohydrate (CAS No. 1310-66-3) (REACHa).

# Risk Characterisation

## Critical Health Effects

The critical health effects for risk characterisation include systemic acute effects (acute toxicity from oral exposure) and local effects (corrosivity), both depending on the concentration.

## Public Risk Characterisation

Although the use of chemicals in this group in cosmetic products in Australia is not known, chemicals in this group have reported limited cosmetic uses overseas (see **Import, manufacture and use**). The available North American database does not give evidence for widespread use in cosmetic products, with only 16 reported products (Personal Care Products Council, 2011). Considering the limited use and reported cosmetic uses overseas (buffering, hair waving), the concentrations in the cosmetic products and the resultant pH are not considered to be sufficiently high to cause any significant human health concerns.

The use of chemicals in this group in domestic products in Australia is not known. The available North American database also does not give evidence for a widespread use in consumer products (Household Products Database, US Department of Health and Human Services). Considering the limited use of these chemicals in domestic products, these chemicals are not expected to cause any concern for human health.

Therefore, the risk to public health is not considered to be unreasonable and further risk management is not considered necessary for public safety.

### Occupational Risk Characterisation

During product formulation, oral, dermal, and ocular exposure might occur, particularly where manual or open processes are used. These could include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment. Worker exposure to the chemicals at lower concentrations could also occur while using formulated products containing the chemicals. The level and route of exposure will vary depending on the method of application and work practices employed.

Given the critical systemic acute/local health effects, the chemicals could pose an unreasonable risk to workers unless adequate control measures to minimise dermal, ocular and inhalation exposure are implemented. The chemicals should be appropriately classified and labelled to ensure that a person conducting a business or undertaking (PCBU) at a workplace (such as an employer) has adequate information to determine the appropriate controls.

The data available support an amendment to the hazard classification in the HSIS (Safe Work Australia) (refer to **Recommendation** section).

## NICNAS Recommendation

Assessment of these chemicals are considered to be sufficient, provided that the recommended amendment to the classification is adopted, and labelling and all other requirements are met under workplace health and safety and poisons legislation as adopted by the relevant state or territory.

### Regulatory Control

#### Work Health and Safety

The chemicals are recommended for classification and labelling under the current approved criteria and adopted GHS as below. This assessment does not consider classification of physical and environmental hazards.

| Hazard | Approved Criteria (HSIS)[a] | GHS Classification (HCIS)[b] |
|---|---|---|
| Acute Toxicity | Harmful if swallowed (Xn; R22) | Harmful if swallowed - Cat. 4 (H302) |
| Irritation / Corrosivity | Causes burns (C; R34) | Causes severe skin burns and eye damage - Cat. 1B (H314) |

[a] Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)].

[b] Globally Harmonized System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third Edition.

[*] Existing Hazard Classification. No change recommended to this classification

### Advice for consumers

Products containing the chemicals should be used according to the instructions on the label.

## Advice for industry

***Control measures***

Control measures to minimise the risk from dermal, ocular and inhalation exposure to the chemicals should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include substitution, isolation and engineering controls. Measures required to eliminate, or minimise risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemicals are used. Examples of control measures which could minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- using local exhaust ventilation to prevent the chemicals from entering the breathing zone of any worker;
- minimising manual processes and work tasks through automating processes;
- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemicals.

Guidance on managing risks from hazardous chemicals are provided in the *Managing risks of hazardous chemicals in the workplace—Code of practice* available on the Safe Work Australia website.

Personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selecting personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

***Obligations under workplace health and safety legislation***

Information in this report should be taken into account to help meet obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((M)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of the chemicals are prepared; and
- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (M)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of safety data sheets for hazardous chemicals—Code of practice* and *Labelling of workplace hazardous chemicals—Code of practice*, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of these chemicals has not been undertaken as part of this assessment.

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Last Update 13 February 2015

## Chemical Identities

| | |
|---|---|
| Chemical Name in the Inventory and Synonyms | **Lithium hydroxide (Li(OH))**<br>lithium hydroxide anhydrous<br>lithium hydroxide |
| CAS Number | 1310-65-2 |
| Structural Formula | |

| | |
|---|---|
| |  |
| Molecular Formula | HLiO |
| Molecular Weight | 23.95 |

| | |
|---|---|
| Chemical Name in the Inventory and Synonyms | **Lithium hydroxide, monohydrate**<br>lithium hydroxide<br>lithium hydroxide monohydrate |
| CAS Number | 1310-66-3 |
| Structural Formula | |

| | |
|---|---|
| |   |
| Molecular Formula | H2O.HLiO |
| Molecular Weight | 41.96 |

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