# Aluminium zirconium chloride hydroxides: Human health tier II assessment

**18 September 2014**



## Chemicals in this assessment

| Chemical Name in the Inventory | CAS Number |
|---|---|
| **Aluminium zirconium chloride hydroxide** | 57158-29-9 |
| **Aluminium zirconium chloride hydroxide (Al4ZrCl4(OH)12)** | 98106-52-6 |
| **Aluminium zirconium chloride hydroxide (Al8ZrCl5(OH)23)** | 98106-54-8 |

## 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

This group consists of aluminium zirconium chloride hydroxide salts which can be considered as a source of zirconium and aluminium ions. Data available for aluminium zirconium chloride hydroxide, zirconium acetate, zirconium sulfate, zirconium dioxide and zirconium showed no evidence of systemic toxicity (REACH). Therefore, aluminium ions are considered to be the moiety responsible for systemic toxicity. Considering that the metal ions in this group are expected to have similar bioaccessibility and bioavailability in biological fluids, data available for aluminium zirconium chloride hydroxide and, where necessary, other soluble aluminium compounds, can be read across when data are lacking for the chemicals in this group (OECD, 2007).

# Import, Manufacture and Use

## Australian

No specific Australian use, import, or manufacturing information has been identified for the chemicals in this group.

## International

The following international uses have been identified through European Union Registration, Evaluation, Authorisation and Restriction of Chemicals (EU REACH) dossiers; Galleria Chemica; the European Commission Cosmetic Ingredients and

Substances (CosIng) database; and the United States (US) Personal Care Product Council International Nomenclature of Cosmetic Ingredients (INCI) dictionary.

The chemicals have reported cosmetic use as:

- antiperspirants and deodorants.

# Restrictions

## Australian

No known restrictions have been identified.

## International

Aluminium zirconium chloride hydroxide ($Al_4ZrCl_4(OH)_{12}$) and aluminium zirconium chloride hydroxide ($Al_8ZrCl_5(OH)_{23}$) are listed on the following:

- EU Cosmetics Regulation 1223/2009 Annex III—List of substances which cosmetic products must not contain except subject to the restrictions laid down; and
- Health Canada List of prohibited and restricted cosmetic ingredients (The Cosmetic Ingredient "Hotlist").

# 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

The chemicals in this group fall under the category 'Aluminium, soluble salts (as Al)' in HSIS, and have an exposure standard of 2 $mg/m^3$ time weighted average (TWA) respectively (HSIS).

### International

There are no specific exposure standards for these chemicals. Many countries have exposure standards for aluminium and/or zirconium compounds (Galleria Chemica):

- Canada, China, Mexico, USA, United Kingdom and India have an exposure standard of 5 $mg/m^3$ time-weighted average (TWA) for the category 'Zirconium compounds, (as Zr)'; and
- most countries and regions (including various states of the USA, various provinces of Canada, Greece and the United Kingdom) have a TWA of 2 $mg/m^3$ for the category 'Aluminium, soluble salts (as Al)'.

# Health Hazard Information

Limited data are available to assess the hazards associated with exposure to zirconium salts. Available information on aluminium zirconium chloride hydroxide and simple zirconium salts and compounds (zirconium acetate, zirconium sulfate, zirconium, zirconium dioxide) indicates that effects associated with zirconium ions are local, mild and reversible. Therefore, assessment of this group is based largely on data relating to chronic exposure to soluble aluminium ions which are referred to as "aluminium" for the rest of the report.

## Toxicokinetics

There are no data available for the toxicokinetics of these chemicals. Information regarding the toxicokinetics of aluminium compounds is provided below.

Assessment of the bioavailability of aluminium compounds is confounded by: limitations in the analytical methodology, particularly for older studies; concurrent exposure to modifying factors; and dose-dependency (bioavailability varies according to exposure levels). Speciation appears to be an important factor in absorption and it is widely assumed that soluble aluminium compounds, such as the chloride and lactate salts, are more bioavailable than insoluble compounds, such as aluminium hydroxide or silicates. Studies in laboratory animals and in human volunteers generally show that absorption of aluminium is less than 1 % by any route. Concurrent intake of organic anions (particularly citrate) increases the absorption of aluminium, while other anions, such as silicates and phosphate, may reduce the absorption of aluminium (WHO, 2007).

*Oral exposure*

Aluminium is poorly absorbed following oral exposure (ATSDR, 2008; Environment Canada and Health Canada, 2010). Approximately 0.1–0.6 % of ingested aluminium is usually absorbed, depending on the dose. The observed relationship between dose and bioavailability is inconsistent; increased doses of aluminium decreased its bioavailability in some experimental studies while opposite results were observed in other studies (Environment Canada and Health Canada, 2010). There are indications that the toxicokinetics of aluminium are dose-dependent and since high doses have been administered in many studies, the results of these studies, with respect to their relevance to humans, should be interpreted with caution (WHO, 2007).

Other factors influencing oral bioavailability include solubility of aluminium compounds, gastric pH, nutritional and medical status (for example, people with Down syndrome absorb aluminium at levels five times higher than people without the condition (EHC, 1997; Krewski 2007)).

*Dermal exposure*

There is some evidence from human case studies that small amounts of aluminium do reach the systemic circulation following dermal application. However, to date, no data for dermal bioavailability are available from controlled studies of more than one or two individuals (Environment Canada and Health Canada, 2010). A recent in vitro study of percutaneous absorption of aluminium from antiperspirants through human skin in the Franz™ diffusion cell found insignificant transdermal absorption of aluminium and particularly, low cutaneous absorption which varied according to the formulations tested (aerosol base, stick and roll on). On stripped skin (mimicking damaged or freshly shaven skin), the measured uptake of aluminium was significantly higher (11.50 mg/cm$^2$ versus 1.81 mg/cm$^2$ for normal skin) using the stick formulation (Pineau, 2012).

*Inhalation exposure*

An investigation in New Zealand White rabbits exposed via the nasal-olfactory pathway (sponge soaked in aluminium solutions inserted into nasal recess for four weeks) provided evidence that inhaled aluminium in the olfactory tract can cross the nasal epithelium to reach the brain directly through axonal transport (Environment Canada and Health Canada, 2010).

*Excretion*

Following oral exposure, unabsorbed aluminium is excreted in the faeces. Absorbed aluminium is excreted principally in the urine and, to a lesser extent, in the bile. It was reported that the higher urinary excretion of aluminium in exposed workers, compared to the general population, demonstrates that some inhaled aluminium can reach the systemic circulation (Environment Canada and Health Canada, 2010).

*Distribution*

Aluminium binds to various ligands in the blood and distributes to every organ, with highest concentrations found in bone and lung tissues (ATSDR, 2008). It crosses the brain and placental barriers in very small amounts. Human and animal studies demonstrate accumulation of aluminium in the brain and, in animal studies, in foetuses (ATSDR, 2008; Environment Canada and Health Canada, 2010).

Aluminium is efficiently transferred from blood to milk in lactating animals. Very small concentrations of aluminium are found in the milk of lactating humans. Two studies were undertaken in Canada to measure levels of aluminium in breast milk. They indicated that mean concentrations of aluminium in breast milk were of the same order of magnitude as elsewhere in the world with an average of approximately 0.11 mg/kg (Environment Canada and Health Canada, 2010).

There is evidence that with increasing age of humans, aluminium concentrations increase in the brain tissue, blood and bone. A number of studies indicate that removal of aluminium from the brain is low (Krewski, 2007). In humans, the aluminium levels are higher in the cerebral cortex and hippocampus than in other brain structures (Environment Canada and Health Canada, 2010; Walton, 2009).

## Acute Toxicity

### Oral

The chemicals in this group have low toxicity based on available data read across information. LD50 values for aluminium zirconium chloride hydroxide were greater than 2000 mg/kg bw in Sprague-Dawley (SD) rat studies conducted in accordance with OECD Test Guideline (TG) 401 (REACH).

### Dermal

The chemicals in this group have low toxicity based on available data and read across information. LD50 values for aluminium zirconium chloride hydroxide was greater than 2000 mg/kg bw in SD rat studies conducted in accordance with OECD TG 402 (REACH).

### Inhalation

No data are available.

## Corrosion / Irritation

### Skin Irritation

The chemicals in this group are not irritating to skin based on available data and read across information. Aluminium zirconium chloride hydroxide produced no skin irritation in studies performed in accordance with OECD TG 404 (REACH).

### Eye Irritation

The chemicals in this group are not irritating to eyes based on available data and read across information. Aluminium zirconium chloride hydroxide produced mild reversible eye irritation (mild chemosis and conjunctivitis) in four studies performed in accordance with OECD TG 405 (REACH). These results do not warrant classification.

## Sensitisation

### Skin Sensitisation

The chemicals in this group are not considered to be skin sensitisers based on available data and read across information. Aluminium zirconium chloride hydroxide was not found to induce dermal sensitisation when tested according to OECD TG 406 (REACH). Other soluble aluminium compounds have not been found to be sensitisers (NICNASa and NICNASb).

## Repeated Dose Toxicity

### Oral

No data for these chemicals are available. However, a study of zirconium acetate conducted in accordance with OECD TG 422 found that, based on the observations in the study, the no observed adverse effects level (NOAEL) for zirconium acetate was > 1,000 mg/kg bw/day.

The lowest observed adverse effect level (LOAEL) for $AlCl_3.6H_2O$ was 0.9 mg/kg bw/day based on results from a chronic rat study (greater than 28 months). Neurotoxic effects and neuropathology observed at and above this concentration are described in the neurotoxicity section.

Apart from neurotoxicity, effects associated with oral exposure to aluminium have only been observed at much higher doses.

### Dermal

No data are available.

### Inhalation

Results from human and animal studies investigating the toxicity of soluble and insoluble forms of aluminium suggest that the respiratory tract, particularly the lung, is a sensitive target of airborne aluminium toxicity. The lung effects observed in humans and animals are suggestive of dust overload (ATSDR, 2008).

### Observation in humans

Aluminium has been shown to have neurotoxic effects in addition to bone and blood toxicity in humans during medical treatment in which the gastrointestinal barrier is bypassed (e.g. aluminium-induced encephalopathy through dialysis treatment in patients with renal failure) (ATSDR, 2008).

Interpretation of the human inhalation data is complicated by the lack of exposure assessment and the potential for exposure to both soluble and insoluble aluminium compounds, and concomitant exposure to other toxic compounds. Numerous studies have found impaired lung function in a variety of aluminium workers. Other effects that have been observed include occupational asthma and pulmonary fibrosis (ASTDR, 2008).

Neurotoxicity data are discussed in the neurotoxicity section.

## Genotoxicity

There is no evidence to support classification of these chemicals for genotoxicity based on data available for aluminium zirconium chloride hydroxide and other soluble aluminium compounds.

Negative results were consistently observed in all short-term in vitro assays with and without metabolic activation for reverse mutations in bacteria, mutations in mammalian cells and mammalian chromosomal aberrations. Negative results were also found for other zirconium compounds tested using these in vitro assays including zirconium sulfate, zirconium dioxide and zirconium acetate (REACH).

The weight of evidence does not support classification of aluminium compounds for genotoxicity (NICNASa and NICNASb).

Negative results were observed in most short-term in vitro mutagenic assays for reverse mutations and forward mutations in bacteria as well as assays for morphological transformation in Syrian hamster cells. Positive results were found in in vivo tests for chromosome aberrations in bone marrow cells in mice and rats (ATSDR, 2008; Environment Canada and Health Canada, 2010).

## Carcinogenicity

There are very few data regarding zirconium compounds. In non-guideline studies that were not conducted in accordance with Good Laboratory Practice (GLP), 5 ppm solutions of zirconium sulfate were administered to mice in two studies and rats in a third study over the lifetime of the animals. No increased incidence of tumours were observed in test animals compared with study controls. These studies did not provide enough information to allow the quality of the studies to be evaluated (REACH).

The available data do not support classification of aluminium compounds as carcinogens.

The aluminium ion has not been shown to be carcinogenic in epidemiological studies in humans, nor animal toxicity studies using inhalation, oral and other exposure routes (ATSDR, 2008). The literature concerning oral exposure bioassays for carcinogenicity is very limited. An increase in gross tumours was reported in male rats and female mice in a one-dose study but few study details were reported. Two other studies reported no increased incidence of tumours in rats and mice exposed orally to aluminium compounds (Environment Canada and Health Canada, 2010).

## Reproductive and Developmental Toxicity

There are very few data available regarding zirconium compounds. Available data suggest that they are not toxic to reproduction and development. A study of zirconium acetate conducted in accordance with GLP and OECD TG 422 using SD rats dosed at 100, 300 and 1000 mg/kg bw/day found a NOAEL for zirconium acetate > 1000 mg/kg bw/day (REACH).

*Animal studies*

There are many animal studies investigating the oral reproductive and developmental toxicity of aluminium salts, in particular, the more bioavailable salts such as aluminium lactate and citrate. As a result of the limitations of the animal study data, both WHO and the Canadian assessments based their evaluations on the combined evidence from many studies, concluding that the lowest observed effect levels (LOELs) for total aluminium (Al) exposures for mice, rats and dogs were in the region of 50–75 mg/kg bw/day. Effects in offspring include histopathological changes in testes, delays in maturation, and neurodevelopmental effects such as decreases in forelimb and hindlimb grip strength (Environment Canada and Health Canada, 2010; IPCS, 2007; ATSDR, 2008).

Significant alterations in motor function, sensory function, and cognitive function have been detected in offspring following gestational and/or lactational exposure of rats and mice to aluminium lactate, aluminium nitrate, and aluminium chloride. Neurodevelopmental effects have been observed in rats and mice at doses of aluminium in the range 103–330 mg/kg bw/day (ATSDR, 2008).

The developmental and chronic neurotoxicity of aluminium citrate was investigated in SD rats in a recent study conducted according to GLP and OECD TG 426. Aluminium citrate was administered in drinking water to groups of pregnant rats, commencing on gestational day 6, at concentrations aiming to deliver aluminium doses of 30, 100 and 300 mg/kg bw/day, based on an expected water intake of 120 mL/kg bw/day. Half of the pups of each group were processed for neurohistopathological examination, and the other half were subjected to a regular necropsy followed by brain weight measurement, clinical chemistry, haematology, and collection of tissues and blood for measurement of aluminium and other metals. Overall, the authors concluded that the study indicated a LOAEL for aluminium of 100 mg/kg bw/day and a no observed adverse effect level (NOAEL) of 30 mg/kg bw/day based on dose-related effects on hindlimb and forelimb grip strength in both male and female

pups (Poierer, 2011; IPCS, 2012). These findings support previous studies in which the most consistently affected performance tests include forelimb and/or hindlimb grip strength (ATSDR, 2008).

*Human studies*

A small study of the offspring of 88 pregnant women who were exposed to elevated levels of aluminium sulfate accidentally added to the local water supply in north Cornwall, England in 1988 found no evidence of major problems apparent from birth (REACH).

There is some epidemiological evidence for long-term cognitive impairment in pre-term infants receiving aluminium-containing nutritional solution intravenously (ATSDR, 2008).

## Other Health Effects

### Neurotoxicity

The very limited data on the toxicity of zirconium suggest that it is not neurotoxic. The neurotoxicity of aluminium compounds has been investigated extensively.

Although the neurotoxicity of aluminium has not been established in humans with normal renal function, the data for dialysis encephalopathy (as well as some occupational studies) establish that the human nervous system is susceptible to aluminium toxicity. In addition, neurotoxicity is a well-documented effect of aluminium in orally exposed mice and rats (ATSDR, 2008).

*Animal studies*

There is an extensive database on the toxicity of aluminium in animals. These studies clearly identify the nervous system as the most sensitive target of aluminium toxicity and most of the animal studies have focused on neurotoxicity and neurodevelopmental toxicity. Neurodevelopmental toxicity is covered in the reproductive and developmental toxicity section of this report.

Overt signs of neurotoxicity are rarely reported at the doses tested in the available animal studies (with aluminium doses less than or equal to 330 mg/kg bw/day for bioavailable aluminium salts); rather, exposure to these doses is associated with subtle neurological effects detected in neurobehavioural performance tests (ATSDR, 2008).

A small number of chronic animal studies of aluminium toxicity have been undertaken, although very little research has been undertaken using aged animals (ATSDR, 2008; WHO, 2007; NHMRC, 2013). Neurodegenerative changes in the brain with cognitive deficits is a characteristic response to aluminium in certain species following non-natural exposure situations, generally involving direct application to brain tissue through injection of aluminium solutions and in vitro incubation in rabbits, cats, ferrets, and nonhuman primates (ATSDR, 2008).

One long-term chronic oral toxicity study investigated neurodegeneration in aged rats at aluminium levels relevant to total human intake. Thirty Wistar rats (10 per dose) were orally exposed to aluminium at doses of 0.4 mg, 0.5 mg and 1.7 mg/kg bw/day in their food (0.4 mg/kg bw/day for all groups) and water (as 0, 2 and 20 ppm Al, equivalent to doses of $AlCl_3.6H_2O$ at 0, 0.9 and 11.6 mg/kg bw/day). These doses of aluminium fall within the range humans are exposed to with the highest dose equivalent to the high end of the human range for total dietary intakes. Dosing for the rats commenced from 12 months old (equivalent to 35 year old humans) until the end of their natural life (28 to 37.5 months, equivalent to 82-109 year old humans) and cognitive function was evaluated using the T-maze task. By age 28 months, none of the rats which were administered the low dose (0/10), two which received the intermediate dose (2/10) and seven of the rats which received the high dose (7/10) exhibited significantly lower mean scores on their T-maze task in old age than in middle age. In addition, the affected rats showed dementia-like behaviours such as confusion, inability to focus attention on the task, perseverative activities and incontinence in the T-maze. This study established a no observed effect level (NOEL) of 0.4 mg/kg bw/day and LOAEL of 0.5 mg/kg bw/day for aluminium in rats (equivalent to 0 and 0.9 mg $AlCl_3.6H_2O$) based on significant cognitive deterioration and neuropathology (brain lesions) (Walton, 2009).

Findings included a significant inverse correlation between memory scores and plasma Al; high relative concentrations of Al and lesions in brain regions associated with memory function (equivalent to where Al and tissue damage have been found in

humans with Alzheimer's disease); elevated markers of oxidative stress and other biochemical changes that precede and lead to hallmarks associated with Alzheimer's disease in humans (plaques, tangles, granulovacuolar degeneration) (Walton, 2014).

*Human studies*

As noted previously, patients with renal failure are at risk of neurotoxicity from aluminium (EHC, 1997). There is also some epidemiological evidence for long-term cognitive impairment in pre-term infants receiving aluminium-containing nutritional solution intravenously, and associated with occupational exposures (ATSDR, 2008). Patients with renal failure and infants receiving parenteral nutrition were exposed to aluminium salts directly through dialysis and intravenous injections, thereby by-passing the gastrointestinal system which effectively excludes most orally ingested aluminium.

With respect to the conditions of exposure in the general population, the most relevant available information is provided by the epidemiological investigations into the association between exposure to aluminium through drinking water and Alzheimer's disease and other forms of dementia (ATSDR, 2008). Most epidemiological studies addressed the potential neurotoxicity of aluminium in drinking water or antacids. The results are mixed. Some of the drinking water studies showed an association of aluminium with dementia or Alzheimer's disease, whereas others reported an absence of neuropsychological effects measured in several ways (IPCS, 2012). Nine out of 13 published epidemiological studies of aluminium in drinking water and Alzheimer's disease have shown statistically significant positive relations (Flatten, 2001).

# Risk Characterisation

## Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects (reproductive and developmental toxicity, neurotoxicity).

## Public Risk Characterisation

Given the health effects identified, in particular neurodevelopmental and neurodegenerative, further investigation of the exposures from antiperspirants and deodorants is recommended.

Although use in cosmetic and domestic products in Australia is not known, this group of chemicals is reported to be used extensively in cosmetic products overseas. Exposure through dermal contact and inhalation with these products is expected to be relatively low for intact skin, although French, German and Norwegian risk assessments based on very conservative assumptions suggest it could be of similar magnitude to exposures through food (BfR 2014a and 2014b; AFSSAPS, 2011; and VKM, 2013). The French agency for the safety of sanitary and health products (AFSSAPS) subsequently recommended that the concentration of aluminium in consumer products should be restricted to 0.6 % and that aluminium-containing cosmetics should not be used on impaired skin (AFSSAPS, 2011).

## Occupational Risk Characterisation

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

Given the critical systemic long-term health effects, these chemicals may pose a risk to workers unless adequate control measures to minimise dermal, ocular and inhalation exposure to the chemical are implemented.

# NICNAS Recommendation

This group of chemicals is recommended for Tier III assessment to better characterise all aluminium exposures from antiperspirants and deodorants and whether these exposures are significant contributors to exceeding tolerable weekly intakes for aluminium. The Tier III assessments would be undertaken in conjunction with those for other soluble aluminium compounds with similar use patterns (NICNASa and NICNASb).

## Regulatory Control

### Public Health

The need for any regulatory controls for public health will be determined as part of the Tier III assessment.

### Work Health and Safety

These chemicals are not recommended for classification and labelling under the current approved criteria and adopted GHS. This assessment does not consider classification of physical hazards and environmental hazards. In the absence of specific data on chemicals in this group, data have been read-across from the data available for members of this group. Should empirical data become available for any member of the group indicating that a lower (or higher) classification is appropriate for the specific chemical, this may be used to amend the default classification for that chemical.

## Advice for consumers

Products containing the chemical should be used according to label instructions.

## Advice for industry

*Control measures*

Control measures to minimise the risk from dermal, ocular and inhalation exposure to these 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 chemical is used. Examples of control measures which may minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- using local exhaust ventilation to prevent the chemical 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 chemical.

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.

The airborne concentrations of these chemicals should be kept as low as practically possible and in accordance with the exposure standards to minimise risk.

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 assist with meeting 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 chemical 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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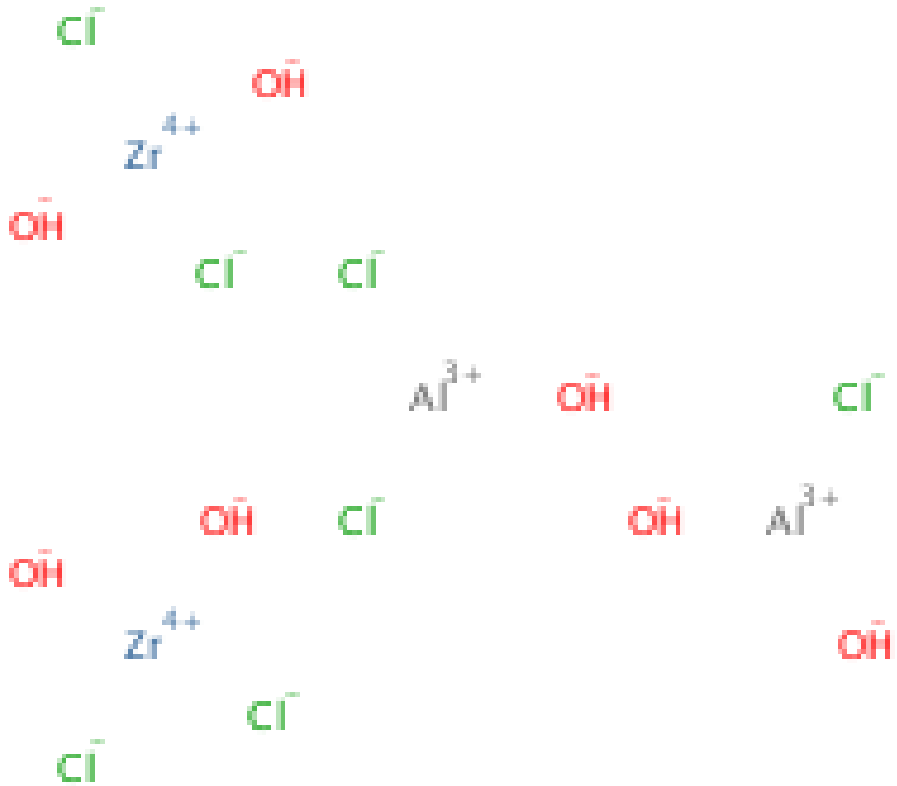
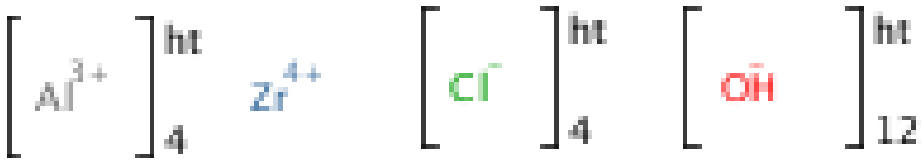
Last Update 18 September 2014

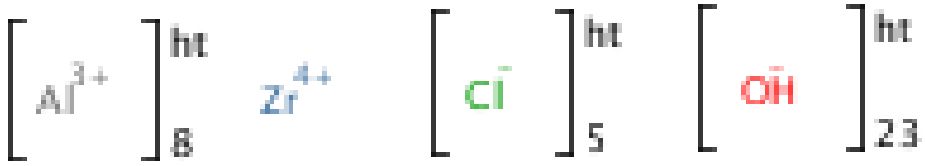
## Chemical Identities

| | |
|---|---|
| Chemical Name in the Inventory and Synonyms | **Aluminium zirconium chloride hydroxide**<br>aluminium zirconium chlorhydrate |
| CAS Number | 57158-29-9 |
| Structural Formula |  |
| Molecular Formula | Unspecified |
| Molecular Weight | 603.63 |

| | |
|---|---|
| Chemical Name in the Inventory and Synonyms | **Aluminium zirconium chloride hydroxide (Al4ZrCl4(OH)12)**<br>aluminium zirconium tetrachlorohydrate<br>tetraaluminium zirconium tetrachloride dodecahydroxide |

| | |
|---|---|
| CAS Number | 98106-52-6 |
| Structural Formula |  |
| Molecular Formula | Al.Cl.HO.Zr |
| Molecular Weight | 545.05 |

| | |
|---|---|
| Chemical Name in the Inventory and Synonyms | **Aluminium zirconium chloride hydroxide (Al8ZrCl5(OH)23)**<br>octaaluminium zirconium pentachloride tricosahydroxide |
| CAS Number | 98106-54-8 |
| Structural Formula | |

| | |
|---|---|
| |  |
| Molecular Formula | Al.Cl.HO.Zr |
| Molecular Weight | 875.50 |

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