# Toxicological profile for

## Aluminum sulfate

***This ingredient has been assessed to determine potential human health effects for the consumer. It was considered not to increase the inherent toxicity of the product and thus is acceptable under conditions of intended use.***

## *1. Name of substance and physico-chemical properties*

### *1.1. IUPAC systematic name*

**16828-11-8:** Dialuminum;trisulfate;hexadecahydrate (PubChem(b)).

**16828-12-9**: Dialuminum;trisulfate;tetradecahydrate (PubChem (c)).

### *1.2. Synonyms*

**16828-11-8:** Aluminium sulfate hexadecahydrate; dialuminum;trisulfate;hexadecahydrate; Sulfuric acid, aluminum salt (3:2), hexadecahydrate; aluminum sulfate.16H2O; Alumiuniumsulfatehexadecahydrate; dialuminum sulfate hexadecahydrate; aluminium sulfate--water (1/16); aluminum(III) sulfate hexadecahydrate (PubChem (b)).

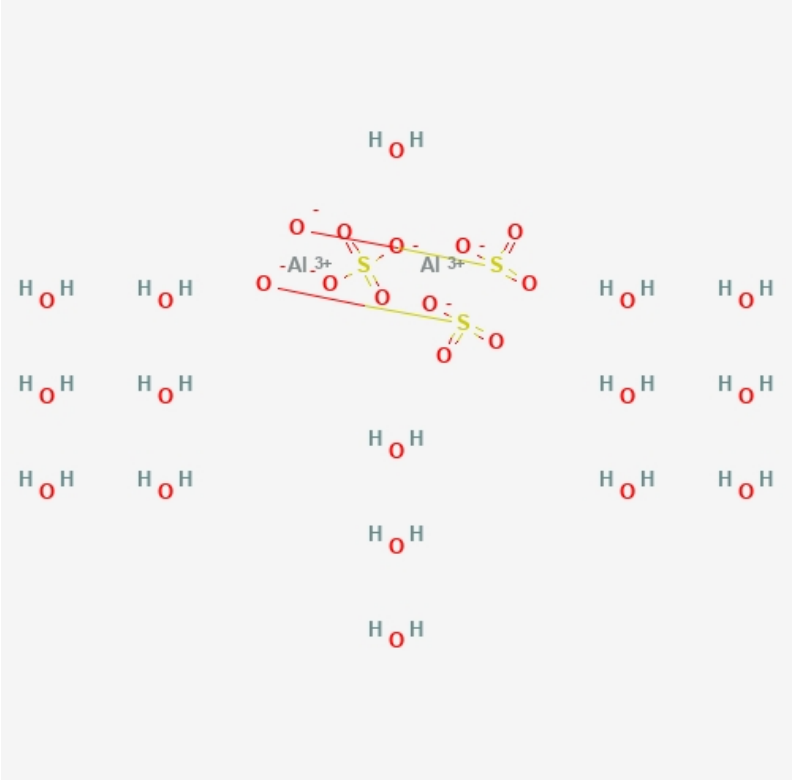
**16828-12-9**: Aluminum sulfate tetradecahydrate; Sulfuric acid, aluminum salt (3:2), tetradecahydrate; Aluminum sulfate ($Al2(SO_4)_3$) hydrate (1:14); dialuminum;trisulfate;tetradecahydrate; UNII-E3UT66504P; ALUMINUM SULPHATE TETRADECAHYDRATE; ALUMINUM SULPHATE (AL2(SO4)3) HYDRATE (1:14); SULFURIC ACID, ALUMINIUM SALT (3:2), TETRADECAHYDRATE (PubChem (c)).

### *1.3. Molecular formula*

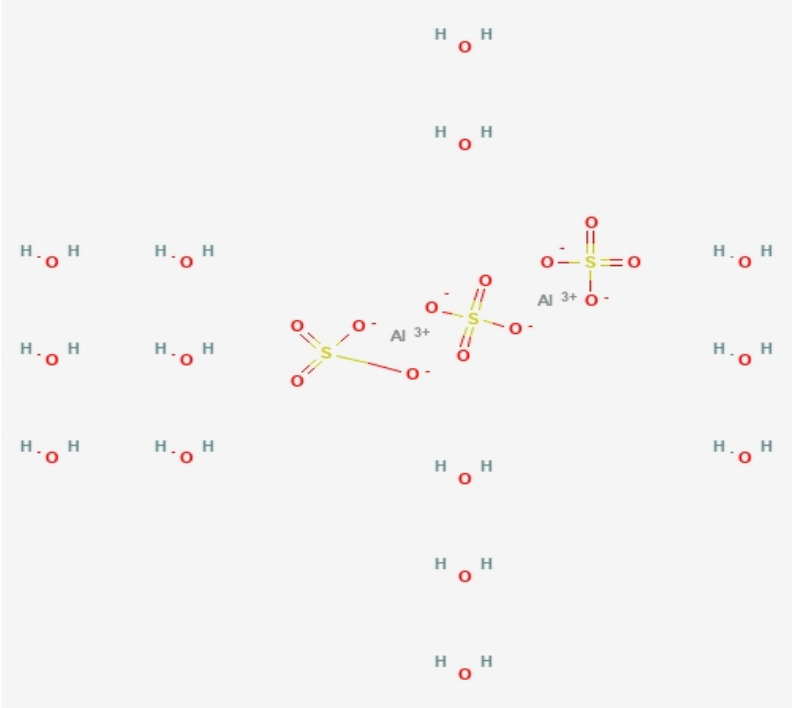
$Al_2H_{22}O_{20}S_3$ (CAS RN 16828-11-8) (PubChem (a)); $Al_2H_{32}O_{28}S_3$ (CAS RN 16828-11-8) (PubChem (b)); $H_{12}AlO_{10}S$ or $H_{32}AlO_{20}S$ (CAS RN 16828-11-8) (ChemSpider); $Al_2H_{28}O_{26}S_3$ (CAS RN 16828-12-9) (PubChem (c))

### *1.4. Structural Formula*

(PubChem(b), (c)).



(CAS RN 16828-11-8)



(CAS RN 16828-12-9)

### *1.5. Molecular weight (g/mol)*

CAS RN 16828-11-8:630.4(computed) (PubChem (b)); 231.135(hexahydrate) or 411.288(hexadecahydrate) (ChemSpider).

CAS RN 16828-12-9: 594,4 (PubChem (c))

### *1.6. CAS registration number*

16828-11-8, 16828-12-9

### *1.7. Properties*

#### *1.7.1. Melting point*

No data available to us at this time.

#### *1.7.2. Boiling point*

No data available to us at this time.

#### *1.7.3. Solubility*

Freely soluble in water. 1000g/l at 20 C (GESTIS)*1*

#### *1.7.4. pKa*

No data available to us at this time.

*1.7.5. Flashpoint*

No data available to us at this time.

*1.7.6. Flammability limits (vol/vol%)*

No data available to us at this time.

*1.7.7. (Auto)ignition temperature*

No data available to us at this time.

*1.7.8. Decomposition temperature*

770°C (GESTIS)

*1.7.9. Stability*

Non-combustible substance. No risk of dust explosion. The substance can react dangerously with: strong bases,oxidizing agents,ammonia, water, amines (GESTIS)

*1.7.10. Vapor pressure*

No data available to us at this time.

*1.7.11. log Kow*

No data available to us at this time.

## *2. General information*

### *2.1. Exposure*

Aluminium sulphates are permitted in liquid egg white foams at 25 mg/kg and candied cherries and at 200 mg/kg (expressed as aluminium).

As taken from EFSA, 2018.

Used as a flocculating agent in the treatment of water intended for human consumption.

As taken from EFSA, 2008.

In Australia, sulfuric acid, aluminium salt (3:2), hexadecahydrate (CAS RN 16828-11-8) has reported use in engineering and water treatment.

As taken from AICIS, 2014.

### *2.2. Combustion products*

No data available to us at this time.

### *2.3. Ingredient(s) from which it originates*

“Aluminium sulfate hexadecahydrate is a hydrate resulting from the formal combination of anhydrous aluminium sulfate with approximately 16 mol eq. of water. It is one of the commonest hydrates of aluminium sulfate. It is an aluminium sulfate and a hydrate.”

As taken from PubChem (b).

## ***3. Status in legislation and other official guidance***

A provisional tolerable weekly intake (PTWI) of 2 mg/kg bw applies to all aluminium compounds in food, including food additives (JECFA, 2012).

In 2017, the Scientific Committee on Health, Environmental and Emerging Risks (SCHEER)

published an opinion on tolerable intake of aluminium with regard to adapting the migration limits for aluminium in toys. The SCHEER used the same study of Poirier et al. (2011) and established a tolerable daily intake (TDI) of 0.3 mg/kg bw per day (EFSA, 2018).

Both sulfuric acid, aluminum salt (3:2), hexadecahydrate (CAS RN 16828-11-8) and sulfuric acid, aluminum salt (3:2), tetradecahydrate (CAS RN 16828-12-9) are not registered under REACH (ECHA).

Aluminium sulphate (CAS RN 16828-11-8), aluminum sulfate (Al2(SO4)3) hydrate (1:14) (CAS RN 16828-12-9) and aluminium sulfate hydrated (no CAS RN given) are not classified for packaging and labelling under Regulation (EC) No. 1272/2008 (ECHA, 2025).

Permissible exposure limit (PEL) for aluminium soluble salts: 2 mg/$m^3$.

As taken from Cal/OSHA

Aluminum sulfate tetradecahydrate (CAS RN 16828-12-9) is classified as an NHP [Natural Health Product] under Schedule 1, item 7 (a mineral) of the Natural Health Products Regulations (Health Canada, 2021).

Aluminum sulfate (CAS RN 16828-12-9) is listed in the US EPA InertFinder Database as approved for food and non-food use pesticide products.

## ***4. Metabolism/Pharmacokinetics***

### *4.1. Metabolism/metabolites*

No data available to us at this time.

### *4.2. Absorption, distribution and excretion*

“Assessment of the bioavailability of aluminium compounds is confounded by limitations in the analytical methodology, particularly for older studies, by concurrent exposure to modifying factors and by 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 work (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 FranzTM 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/cm2 versus 1.81 mg/cm2 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). alum; aluminium ammonium sulfate; and aluminium potassium sulfate. International The Joint Food and Agriculture Organization (FAO) of the United Nations/World Health Organization (WHO) Expert Committee on Food Additives (JECFA) established a provisional tolerable weekly intake (PTWI) of 2 mg Al/ kg bw/day, which is adopted in Australia by FSANZ. The European Food Safety Agency set a Tolerable Weekly Intake of 1 mg Al/kg bw/day for all aluminium compounds (IPCS, 2012). 16/04/2020 IMAP Group Assessment Report https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment_id=993 4/16

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

Aluminum 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)."

As taken from AICIS (2014)

### *4.3. Interactions*

Aluminum (Al) is commonly used in industrial processes and drugs and is thought to induce erythrocytes damage via activation of oxidative stress. Recently, bismuth (Bi)-containing drugs are used in the treatment of various diseases. However, uncertain effects of Bi in blood tissue may participate in the therapeutic efficacy of Bi compounds as related to metals. Hence, this study aimed to determine the roles on human blood cells of the various concentrations of aluminum sulphate (Al(2)(SO(4))(3)) and bismuth subnitrate (BSN), separate and together. With this aim, oxidative status was assessed on erythrocytes by measuring following oxidative stress markers: reduced glutathione (GSH), superoxide dismutase (SOD), glucose-6-phosphate dehydrogenase (G-6-PDH) and catalase (CAT). Two chemicals were tested for their ability to induce cytogenetic change in human lymphocytes using assays for chromosome aberrations (CAs) and sister chromatid exchanges (SCEs). Our results showed that high dose of Al(2)(SO(4))(3) (20 µg/mL) caused oxidative stress and increased CA and SCE frequencies. Whereas, BSN doses did not change CA and SCE rates. Moreover, it led to changes of antioxidant capacity at different concentrations. After concomitant treatment with Al(2)(SO(4))(3) and BSN, the effects of BSN doses were different on enzyme activities and decreased the genotoxic damage. However, the high dose of BSN and Al(2)(SO(4))(3) was shown to enhance the frequencies of CAs and SCEs in a synergistic manner. In conclusion, BSN could be effective in the protection against the blood toxicity of Al(2)(SO(4))(3). As taken from Turkez H et al. 2011. Toxicol Ind Health 27(2):133-42. Pubmed, 2016

## ***5. Toxicity***

### *5.1. Single dose toxicity*

No data available to us at this time.

### *5.2. Repeated dose toxicity*

The lowest published toxic dose (TDLo) by oral administration to chickens is reported to be 190 g/kg bw/14D (intermittent) (Anon, 1995). [This would be equivalent to a daily dose of around 13.6 g/kg bw/day over 14 days.]

### *5.3. Reproduction toxicity*

"Neurodevelopmental effects have been observed in rats and mice at doses of 103–330 mg Al/kg bw/day (ATSDR, 2008). This is equivalent to 652– 2090 mg sulfuric acid, aluminium salt (3:2)."

As taken from AICIS (2014).

### *5.4. Mutagenicity*

No data available to us at this time.

### *5.5. Cytotoxicity*

Aluminium has toxic effects on many organ systems of the human body. Aluminium toxicity also is a factor in many neurodegenerative diseases. We investigated changes in numbers of hippocampal neurons in rats exposed to aluminium using an optical fractionator and we investigated aluminium-induced apoptosis using the transferase mediated dUTP nick end labeling (TUNEL) assay. Twenty-four female rats were divided equally into control, sham and aluminium-exposed groups. The control group received no treatment. The two treatment groups were injected intraperitoneally with 1 ml 0.9% saline without (sham) and with 3 mg/ml aluminium sulfate every day for two weeks. Following the treatments, the brains were removed, the left hemisphere was used for hippocampal neuron counting using an optical fractionator and the right hemisphere was investigated using hippocampal TUNEL assay to determine the apoptotic index. The number of neurons in the stratum pyramidale of the hippocampus was significantly less in the aluminium group than in the control and sham groups; there was no significant difference between the control and sham groups. The apoptotic index also was significantly higher in the aluminium group than in the other two groups. We quantified the toxic effects of aluminium on the rat hippocampus and determined that apoptosis was the mechanism of aluminium-induced neuron death in the hippocampus. As taken from Cabus N et al. 2015. Biotech Histochem 90(2):132-9. Pubmed, 2016

Aluminum (Al) is commonly used in industrial processes and drugs and is thought to induce erythrocytes damage via activation of oxidative stress. Recently, bismuth (Bi)-containing drugs are used in the treatment of various diseases. However, uncertain effects of Bi in blood tissue may participate in the therapeutic efficacy of Bi compounds as related to metals. Hence, this study aimed to determine the roles on human blood cells of the various concentrations of aluminum sulphate (Al(2)(SO(4))(3)) and bismuth subnitrate (BSN), separate and together. With this aim, oxidative status was assessed on erythrocytes by measuring following oxidative stress markers: reduced glutathione (GSH), superoxide dismutase (SOD), glucose-6-phosphate dehydrogenase (G-6-PDH) and catalase (CAT). Two chemicals were tested for their ability to induce cytogenetic change in human lymphocytes using assays for chromosome aberrations (CAs) and sister chromatid exchanges (SCEs). Our results showed that high dose of Al(2)(SO(4))(3) (20 µg/mL) caused oxidative stress and increased CA and SCE frequencies. Whereas, BSN doses did not change CA and SCE rates. Moreover, it led to changes of antioxidant capacity at different concentrations. After concomitant treatment with Al(2)(SO(4))(3) and BSN, the effects of BSN doses were different on enzyme activities and decreased the genotoxic damage. However, the high dose of BSN and Al(2)(SO(4))(3) was shown to enhance the frequencies of CAs and SCEs in a synergistic manner. In conclusion, BSN could be effective in the protection against the blood toxicity of Al(2)(SO(4))(3). As taken from Turkez H et al. 2011. Toxicol Ind Health 27(2):133-42. Pubmed, 2016

### *5.6. Carcinogenicity*

No data available to us at this time.

### *5.7. Irritation/immunotoxicity*

“When treated with sulfuric acid, aluminium salt (3:2), three studies conducted in accordance with OECD TG 405 reported eye irritation. Two of the studies found conjunctival redness and swelling which was not reversible during the test periods (three and seven days). The third test reported conjunctivitis and purulent ophthalmitis which were reversible during the 21-day study.”

As taken from AICIS (2014).

### *5.8. All other relevant types of toxicity*

No data available to us at this time.

## *6. Functional effects on*

### *6.1. Broncho/pulmonary system*

No data available to us at this time.

### *6.2. Cardiovascular system*

Aluminum (Al) is commonly used in industrial processes and drugs and is thought to induce erythrocytes damage via activation of oxidative stress. Recently, bismuth (Bi)-containing drugs are used in the treatment of various diseases. However, uncertain effects of Bi in blood tissue may participate in the therapeutic efficacy of Bi compounds as related to metals. Hence, this study aimed to determine the roles on human blood cells of the various concentrations of aluminum sulphate (Al(2)(SO(4))(3)) and bismuth subnitrate (BSN), separate and together. With this aim, oxidative status was assessed on erythrocytes by measuring following oxidative stress markers: reduced glutathione (GSH), superoxide dismutase (SOD), glucose-6-phosphate dehydrogenase (G-6-PDH) and catalase (CAT). Two chemicals were tested for their ability to induce cytogenetic change in human lymphocytes using assays for chromosome aberrations (CAs) and sister chromatid exchanges (SCEs). Our results showed that high dose of Al(2)(SO(4))(3) (20 μg/mL) caused oxidative stress and increased CA and SCE frequencies. Whereas, BSN doses did not change CA and SCE rates. Moreover, it led to changes of antioxidant capacity at different concentrations. After concomitant treatment with Al(2)(SO(4))(3) and BSN, the effects of BSN doses were different on enzyme activities and decreased the genotoxic damage. However, the high dose of BSN and Al(2)(SO(4))(3) was shown to enhance the frequencies of CAs and SCEs in a synergistic manner. In conclusion, BSN could be effective in the protection against the blood toxicity of Al(2)(SO(4))(3). As taken from Turkez H et al. 2011. Toxicol Ind Health 27(2):133-42. Pubmed, 2016

### *6.3. Nervous system*

Aluminium has toxic effects on many organ systems of the human body. Aluminium toxicity also is a factor in many neurodegenerative diseases. We investigated changes in numbers of hippocampal neurons in rats exposed to aluminium using an optical fractionator and we investigated aluminium-induced apoptosis using the transferase mediated dUTP nick end labeling (TUNEL) assay. Twenty-four female rats were divided equally into control, sham and aluminium-exposed groups. The control group received no treatment. The two treatment groups were injected intraperitoneally with 1 ml 0.9% saline without (sham) and with 3 mg/ml aluminium sulfate every day for two weeks. Following the treatments, the brains were removed, the left hemisphere was used for hippocampal neuron counting using an optical fractionator and the right hemisphere was investigated using hippocampal TUNEL assay to determine the apoptotic index. The number of neurons in the stratum pyramidale of the hippocampus was significantly less in the aluminium group than in the control and sham groups; there was no significant difference between the control and sham groups. The apoptotic index also was significantly higher in the aluminium group than in the other two groups. We quantified the toxic effects of aluminium on the rat hippocampus and determined that apoptosis was the mechanism of aluminium-induced neuron death in the hippocampus. As taken from Cabus N et al. 2015. Biotech Histochem 90(2):132-9. Pubmed, 2016

“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 (less than or equal to 330 mg Al/kg bw/day for bioavailable aluminium salts); rather, exposure to these doses is associated with subtle neurological effects detected with 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 and 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 0.4 mg, 0.5 mg and 1.7 mg Al/kg bw/day in their food (0.4 mg Al/kg bw/day for all groups) and water (as 0, 2 and 20 ppm Al, equivalent to 0, 0.9 and 11.6 mg AlCl3.6H2O/kg bw/day). 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 (0/10) in the low Al dose, two of the rats (2/10) receiving the intermediate Al dose and seven rats (7/10) that consumed Al at the high end of the human range for total dietary Al exhibited significantly lower mean scores on their T-maze task in old age than in middle age, as well as showing dementialike 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 Al/kg bw/day and LOAEL of 0.5 mg Al/kg bw/day in rats (equivalent to 0 and 0.9 mg AlCl3.6H2O) 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 has 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).”
As taken from AICIS (2014).

### *6.4. Other organ systems, dependent on the properties of the substance*

No data available to us at this time.

## *7. Addiction*

JTI is not aware of any information that demonstrates that this ingredient has any addictive effect.

## *8. Burnt ingredient toxicity*

No data available to us at this time.

## *9. Heated/vapor emissions toxicity*

No data available to us at this time.

## *10. Ecotoxicity*

### *10.1. Environmental fate*

No data available to us at this time.

### *10.2. Aquatic toxicity*

“Early-life-stage golden trout (Oncorhynchus aguabonita aguabonita) were exposed to acid and Al to examine the response and determine the sensitivity of a western, alpine salmonid to conditions simulating an episodic pH depression. Freshly fertilized eggs, alevins, and swim-up larvae were exposed for 7 d to one of 12 combinations of pH and Al, and surviving fish were held to 40 d post-hatch to determine the effect of exposure on subsequent survival and recovery. Golden trout are sensitive to conditions simulating episodic acidification events typically observed in the field. Significant mortality occurred when the pH of test waters was below 5.0 in the absence of Al or when pH was 5.5 in the presence of 100 ug/L total Al. Behavioral impairments were sensitive indicators of low pH and Al stress. Impaired locomotory and feeding behavior occurred at pH 5.5 without Al and at Al concentrations greater than or equal to 50 ug/L. In contrast, growth, RNA-to-DNA ratio, and whole-body ion concentration were relatively less sensitive indicators of sublethal acid and Al stress”. As taken from DeLonay, A J, Little, E E, Woodward, F, Brumbaugh, W G, Farag, A M, and Rabeni, C F. Sensitivity of early-life-stage golden trout to low pH and elevated aluminum. United States: N. p., 1993. Web. doi:10.1002/etc.5620120711.

Toxic to aquatic organisms. It hydrolyses in water forming sulphuric acid (INCHEM, 2010).

### *10.3. Sediment toxicity*

No data available to us at this time.

### *10.4. Terrestrial toxicity*

No data available to us at this time.

### *10.5. All other relevant types of ecotoxicity*

No data available to us at this time.

## ***11. References***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## *12. Other information*


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## *13. Last audited*

February 2025