

CLH report

Proposal for Harmonised Classification and Labelling

**Based on Regulation (EC) No 1272/2008 (CLP Regulation),
Annex VI, Part 2**

International Chemical Identification: Sodium chlorate

EC Number: 231-887-4
CAS Number: 7775-09-9
Index Number: 017-005-00-9

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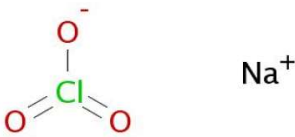
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1 IDENTITY OF THE SUBSTANCE

1.1 Name and other identifiers of the substance

Table 1: Substance identity and information related to molecular and structural formula of the substance

Name(s) in the IUPAC nomenclature or other international chemical name(s)	Sodium chlorate
Other names (usual name, trade name, abbreviation)	/
ISO common name (if available and appropriate)	/
EC number (if available and appropriate)	231-887-4
EC name (if available and appropriate)	Sodium chlorate
CAS number (if available)	7775-09-9
Other identity code (if available)	/
Molecular formula	ClHO ₃ .Na
Structural formula	
SMILES notation (if available)	[Na+].[O-]Cl(=O)=O
Molecular weight or molecular weight range	106.441 g/mol
Information on optical activity and typical ratio of (stereo) isomers (if applicable and appropriate)	Not applicable
Description of the manufacturing process and identity of the source (for UVCB substances only)	Not applicable
Degree of purity (%) (if relevant for the entry in Annex VI)	

1.2 Composition of the substance

Table 2: Constituents (non-confidential information)

Constituent (Name and numerical identifier)	Concentration range (% w/w minimum and maximum in multi-constituent substances)	Current Annex VI (CLP)	CLH in Table 3.1	Current classification and labelling (CLP)	self-and
Sodium chlorate EC no.: 231-887-4	≥98.0%	Oxid. Solid 1; H271 Acute Tox. 4; H302 Aquatic Chronic 2; H411			

Table 3: Impurities (non-confidential information) if relevant for the classification of the substance

Impurity (Name and numerical identifier)	Concentration range (% w/w minimum and maximum)	Current CLH in Annex VI Table 3.1 (CLP)	Current self-classification and labelling (CLP)	The impurity contributes to the classification and labelling

Table 4: Additives (non-confidential information) if relevant for the classification of the substance

Additive (Name and numerical identifier)	Function	Concentration range (% w/w minimum and maximum)	Current CLH in Annex VI Table 3.1 (CLP)	Current self-classification and labelling (CLP)	The additive contributes to the classification and labelling
	No additives				

2 PROPOSED HARMONISED CLASSIFICATION AND LABELLING

2.1 Proposed harmonised classification and labelling according to the CLP criteria

Table 5:

	Index No	International Chemical Identification	EC No	CAS No	Classification		Labelling			Specific Conc. Limits, M-factors	Notes
					Hazard Class and Category Code(s)	Hazard statement Code(s)	Pictogram, Signal Word Code(s)	Hazard statement Code(s)	Suppl. Hazard statement Code(s)		
Current Annex VI entry	017-005-00-9	Sodium chlorate	231-887-4	7775-09-9	Ox. Sol. 1 Acute Tox. 4 * Aquatic Chronic 2	H271 H302 H411	GHS03 GHS07 GHS09 Dgr	H271 H302 H411			
Dossier submitters proposal	017-005-00-9	Sodium chlorate	231-887-4	7775-09-9	Remove Aquatic Chronic 2 Modify Acute Tox. 3	Remove H411 Modify H301	Remove GHS09 Modify GHS06	Remove H411 Modify H301		Add oral; ATE = 100 mg/kg bw	
Resulting Annex VI entry if agreed by RAC and COM	017-005-00-9	Sodium chlorate	231-887-4	7775-09-9	Ox. Sol. 1 Acute Tox. 3	H271 H301	GHS03 GHS06 Dgr	H271 H301		oral; ATE = 100 mg/kg bw	

Table 6: Reason for not proposing harmonised classification and status under public consultation

Hazard class	Reason for no classification	Within the scope of public consultation
Explosives	Hazard class not assessed	No
Flammable gases (including chemically unstable gases)	Hazard class not assessed	No
Oxidising gases	Hazard class not assessed	No
Gases under pressure	Hazard class not assessed	No
Flammable liquids	Hazard class not assessed	No
Flammable solids	Hazard class not assessed	No
Self-reactive substances	Hazard class not assessed	No
Pyrophoric liquids	Hazard class not assessed	No
Pyrophoric solids	Hazard class not assessed	No
Self-heating substances	Hazard class not assessed	No
Substances which in contact with water emit flammable gases	Hazard class not assessed	No
Oxidising liquids	Hazard class not assessed	No
Oxidising solids	Hazard class not assessed	No
Organic peroxides	Hazard class not assessed	No
Corrosive to metals	Hazard class not assessed	No
Acute toxicity via oral route	Harmonised classification proposed	Yes
Acute toxicity via dermal route	Hazard class not assessed	No
Acute toxicity via inhalation route	Hazard class not assessed	No
Skin corrosion/irritation	Hazard class not assessed	No
Serious eye damage/eye irritation	Hazard class not assessed	No
Respiratory sensitisation	Hazard class not assessed	No
Skin sensitisation	Hazard class not assessed	No
Germ cell mutagenicity	Hazard class not assessed	No
Carcinogenicity	Hazard class not assessed	No
Reproductive toxicity	Hazard class not assessed	No
Specific target organ toxicity-single exposure	Hazard class not assessed	No
Specific target organ toxicity-repeated exposure	Hazard class not assessed	No
Aspiration hazard	Hazard class not assessed	No
Hazardous to the aquatic environment	Harmonised classification proposed	Yes
Hazardous to the ozone layer	Hazard class not assessed	No

3 HISTORY OF THE PREVIOUS CLASSIFICATION AND LABELLING

Sodium chlorate was introduced in the Annex I by Commission Directive 93/72/EEC of 1 September 1993 adapting to technical progress for the nineteenth time Council Directive 67/548/EEC.

The environmental classification was included the harmonized classification by Commission Directive 2004/73/EEC of 29 April 2004 adapting to technical progress for the 29th time Council Directive 67/548/EEC.

4 JUSTIFICATION THAT ACTION IS NEEDED AT COMMUNITY LEVEL

Sodium chlorate is classified Aquatic Chronic 2 (H411) in the Annex VI of the CLP regulation. The data that support this classification has been evaluated in this report and considered not valid. The available data that is considered valid for sodium chlorate, as presented in this report, do not support a long-term hazard classification for the environment.

The substance is classified as Acute Tox. 4* for acute oral toxicity. The available data in this report support the classification of Acute Tox. 3 for acute oral toxicity.

[B.] Justification that action is needed at Community level is required.

Reason for a need for action at Community level:

Change in existing entry due to changes in the criteria

Further detail on need of action at Community level

According to the 2nd Adaptation to Technical Progress (ATP) to the CLP Regulation (Commission Regulation (EU) No 286/2011), when adequate chronic toxicity data are available for all three trophic levels, the substance can be classified using the chronic data depending on information on rapid degradation.

Chronic toxicity data of sodium chlorate are available for all three trophic levels (fish, crustacean and algae/aquatic plants). Taking into account that all the chronic toxicity values are above 1 mg/L, no long-term hazard classification is required according to CLP.

As the classification and labelling should properly reflect the hazards of a substance, a change of the classification for the aquatic environment should be considered.

The current Annex VI entry for sodium chlorate includes Aquatic Chronic 2 (H411) in the column "Classification" in Table 3.1. The data used for this classification are not considered valid as evaluated in the REACH registration dossier and explained in section 11 below. The data that is presented in this report and that was the basis for the hazard evaluation in the REACH registration dossier support that the substance does not need to be classified for the aquatic environment.

The current Annex VI entry for sodium chlorate includes Acute Tox. 4* oral as a minimum classification as indicated by the reference * in the column "Classification" in Table 3.1. The data that is presented in this report and that was the basis for the hazard evaluation in the REACH registration dossier supports the classification as Acute Tox. 3 for acute oral toxicity.

This CLH report was initially drafted by Nouryon Pulp and Performance Chemicals AB (former AkzoNobel Pulp and Performance Chemicals AB) and submitted via the Swedish Competent Authority.

5 IDENTIFIED USES

Sodium chlorate is mostly used as an intermediate in the synthesis of chlorates, perchlorates and chlorites and also in pulp and paper bleaching agent (manufacture of chlorine dioxide). The substance can also be used in the metal finishing industry and for pyrotechnics.

6 DATA SOURCES

Information on SODIUM CHLORATE was collected from different external sources:

1) Physico-chemical Literature Search (2009.09.07).

STN Databases:

CAS REGISTRY service : The search was performed by CAS number (7775-09-9).

HCAPLUS : The search was performed by CAS number (7775-09-9), and by specific keywords. The result set has been limited by publication year (>1985).

2) Fate/Ecotoxicology Literature Search (2009.09.07). The search was performed by CAS number (7775-09-9), by synonyms (chemical names) and by specific keywords.

STN Databases : HCAPLUS, AQUIRE, BIOSIS, CSNB, RTECS, TOXCENTER, HSDB

A selection of relevant articles was also made via web search engines or directly on publisher websites.

The last literature search is from 2019-05-23. No new information relevant for hazard assessment was found.

REACH dossier: <https://echa.europa.eu/registration-dossier/-/registered-dossier/14688/1>

European Commission. Draft Assessment Report Chlorate. Prepared by France, January, 2008.

7 PHYSICOCHEMICAL PROPERTIES

Table 75: Summary of physicochemical properties

Property	Value	Reference	Comment (e.g. measured or estimated)
Physical state at 20°C and 101,3 kPa	Crystalline solid, pale yellow	De Ryckel (2006)	
Melting/freezing point	255.0 – 259.4°C at 954 hPa	Paradis (2004)	
Boiling point	Sodium chlorate decomposes when liquefied at 93.6 kPa (>250°C)	Paradis (2003)	
Relative density	2.54 g/cm ³ at 20.2°C	Mourgues (2004)	
Vapour pressure	<3.5E ⁻⁰⁷ hPa at 25°C	Tremains (2004)	
Surface tension	72.9 mN/m at 1g/l and at 20°C	Tremains (2003)	The substance is not considered as surface active
Water solubility	696 – 736 g/l at 20°C for pH 4.49 to 8.70	Groult (2004)	
Partition coefficient n-octanol/water	-2.9 at 20°C	Groult (2004)	Weakly soluble in n-octanol
Flash point	Not relevant		
Flammability	Non flammable	De Ryckel (2006)	
Explosive properties	Non explosive	Mak (2004)	
Self-ignition temperature	No relative self-ignition temperature	Mak (2004)	
Oxidising properties	Oxidizing	De Ryckel (2006)	

Property	Value	Reference	Comment (e.g. measured or estimated)
Granulometry	Determined by sieving: >5000µm: 0.00% 2000-5000µm: 2.00% 1000-2000µm: 25.77% 850-1000µm: 11.06% 600-850µm: 25.71% 500-600µm: 9.11% 400-500µm: 8.93% 200-400µm: 12.20% 150-200µm: 1.5% <150µm: 3.56%	De Ryckel (2006)	
Stability in organic solvents and identity of relevant degradation products	Not applicable		
Dissociation constant	pKa = -1 to -3	Weissenfeld (2004)	
Viscosity	Not applicable		

8 EVALUATION OF PHYSICAL HAZARDS

This part was not evaluated in this dossier and no modification of the classification for physico-chemical properties is proposed.

9 TOXICOKINETICS (ABSORPTION, METABOLISM, DISTRIBUTION AND ELIMINATION)

This part was not evaluated in this dossier.

10 EVALUATION OF HEALTH HAZARDS

Acute oral toxicity

10.1 Acute toxicity - oral route

Table 8: Summary table of animal studies on acute oral toxicity

Method, guideline, deviations if any	Species, strain, sex, no/group	Test substance,	Dose levels, duration of exposure	Value LD ₅₀	Reference
EPA OPP 81-1 (Acute Oral Toxicity)	Rat, Sprague-Dawley 5 per sex/group	Sodium chlorate	- range finding study: 300, 600, 1250, 2500 and 5000 mg/kg bw; (one male and one female per dose) - full acute oral limit test 1: 5000 mg/kg bw	> 5000 mg/kg bw	Study report , 1991 ¹

¹ Key study REACH dossier

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			- full acute oral limit test 2: 2000 mg/kg bw		
OECD Guideline 401 (Acute Oral Toxicity) before 2002	Rat, Charles River CD Range finding: 2 per sex/group, Main study: 8 per sex/group, except in high dose group where 7 females were dosed	Sodium chlorate	- range finding study: 1000, 1500, 5000 mg/kg bw - main study: 1470, 2150, 3160, 4640, 6810 mg/kg bw males and 2150, 3160, 4640, 6810, 10000 mg/kg bw females	Males, 4950 mg/kg in males (95% Confidence limits: 3960 to 6188) Females, 6250 mg/kg in females (confidence limits: 5274 to 7406)	Study report, 1981

Table 9: Summary table of human data on acute oral toxicity

Type of data/report	Test substance,	Lethal dose in mg/kg bw ²	Relevant information about the study (as applicable)	Reference
Review AFSSA (French poison center) ¹	Sodium chlorate	143-286 mg/kg bw (adult)	29 individuals had pathological methemoglobinemia (MetHb \geq 3%). The smallest doses causing a pathological MetHb (\geq 3%) humans in this study were in the order of 10-20 grams of sodium chlorate orally ingested. 13 (45%) of the 29 individuals did not survive.	AFSSA, 2011
Public literature	Sodium chlorate	No data	Deaths from pesticide poisoning in England and Wales: 1945-1989 Sodium chlorate caused 113 deaths, most of these fatalities occurring between 1965 and 1983; only one death has been recorded since 1984.	Casey P, Vale JA, 1994
Public literature	Sodium chlorate	No data	A chemical industry worker died from sodium chlorate intake, amount unknown	Eysseric H et al, 2000
Public literature	Sodium chlorate	214 mg/kg bw (adult)	Outcome in 14 patients poisoned by sodium chlorate. Mortality was high (64%), and death invariably occurred, irrespective of treatment, when the amount of sodium chlorate ingested exceeded 100 g. In this study, the smallest lethal dose published was 15 g (214 mg of chlorate / kg body weight) and concerned a 46-year-old woman who died at medical care in intensive care. In this same series, another death occurred at a woman (unspecified age) following	Helliwell M, Nunn J, 1979

² Estimated intakes per kg bw were calculated with a default body weight assumption of 70 kg for adults, 10 kg for children and 5 kg for infants.

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			the intake of a 30 g dose (429 mg / kg chlorate despite treatment with methylene blue, hemodialysis and exsanguino-transfusion.	
Public literature	Sodium chlorate	No data	Two patients admitted to Halton in 1960 recovered from sodium-chlorate poisoning associated with severe renal failure. Both required hemodialysis (Kolff twin-coil artificial kidney). In one, poisoning occurred accidentally while the patient was using a weed-killer in an atomiser for agricultural purposes; and in the other it was the result of a suicide attempt.	Jackson et al., 1961
Public literature	Sodium chlorate	No death, survived 571 mg/kg bw (adult)	40 g sodium chlorate was taken by error instead of that amount of sodium chloride by a 28-year-old man and he survived.	Klendshoj NC et al., 1962
Public literature	Sodium chlorate	No data	155 cases of which 116 were fatal. Eighteen of these were either suicidal or homicidal.	Witthaus 1911 cited in Klendshoj NC et al., 1962
Public literature	Potassium chlorate	500 mg/kg bw (adult)	Cochran and Smith reported a case of Bright's disease in which potassium chlorate had mistakenly been given instead of potassium chloride. There was evidence that the patient took approximately 35 g over a period of 3 days. Death occurred on the 5th day after the last dose.	Cochran and Smith cited in Klendshoj NC et al., 1962
Public literature	Sodium chlorate	No data	Ansbacher described a rapidly progressing case of a pharmacy student who took the poison in the morning and died the same evening. The symptoms were violent vomiting, deep cyanosis, diarrhea, and the blood was described as dark brown.	Ansbacher cited in Klendshoj NC et al., 1962
Public literature	Sodium chlorate	No data	Gordon and Brown 4 have detailed the case of a woman who had sucked 25 tablets of potassium chlorate daily for from 6 to 10 weeks as a self-prescribed cure for an imaginary malignancy of the tongue. Features of the case were severe hemorrhage, methemoglobinemia, and renal damage, and death was ultimately ascribed to renaltubular damage due to deposition of pigment.	Gordon and Brown cited in Klendshoj NC et al., 1962

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Public literature	Potassium chlorate	No data	Gettler and St. George have cited a fatal case in a 3-year-old boy. The physician prescribed a potassium chlorate gargle over the telephone. The mother misunderstood the directions and gave the child the solution to drink. Death occurred in about 6 hours. The essential findings were 91.9% methemoglobin and acute parenchymatous nephrosis	Gettler and St. George cited in Klendshoj NC et al., 1962
Public literature	Sodium chlorate	No data	The results of the toxicological, macroscopical and microscopical investigations carried out on two cases of suicidal poisoning confirm that death was as a result of chlorate ingestion. No further details are provided.	Oliver JS, Smith H, Watson AA, 1972
Public literature	Sodium chlorate	No data	A homicidal poisoning from sodium chlorate administered intermittently over a period of about 5 weeks.	Jansen H, Zeldenrust J, 1972
Public literature	Potassium chlorate	107 mg/kg bw (adult)	A mentally diseased army officer, aged 33, ate an entire tube of Pebecco Tooth Paste on an empty stomach, corresponding, in the opinion of the author, to 7.5 G of potassium chlorate and died.	Bernstein cited in Klendshoj NC et al., 1962
Public literature	Sodium chlorate	No death, survived 334 mg/kg bw (adult)	A 29 year old man ingested about 20 g of sodium chlorate (230 mg chlorate/kg body weight). He became cyanotic, and his hemoglobin dropped to 11 g/100 mL within 24 hr; methemoglobin and methemoalbumin were detected in his plasma. He was anuric for 14 days, then gradually improved, and he was released from the hospital after 6 wk.	HSDB, 2005, referring to National Research Council, 1987 Bloxham CA et al., 1979
Public literature	Sodium chlorate	2143-2857 mg/kg bw (adult)	A case of severe sodium chlorate poisoning was observed within 5 h after suicidal ingestion of 150–200 g of the herbicide. Methaemoglobinaemia was the early symptom of the intoxication.	Steffen C, Seitz R, 1981
Public literature	Sodium chlorate	71-143 mg/kg bw (adult) 133 mg/kg bw (child)	A dose of 5-10 g can prove fatal in adults, as can a dose of 2 g in small children.	Hartley, D. and H. Kidd, 1987
Public literature	Sodium chlorate	No data	A 26 year old man committed suicide, the cause of death was determined to	Cunningham, 1982

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			<p>be methaemoglobinaemia following ingestion of a poison. The toxicological analysis revealed 700 mg of chlorate per 100 ml urine and the stomach contents gave a positive result for chlorate.</p> <p>A 52 year old female committed suicide, toxicological analysis revealed 152,4 mg of chlorate per 100 ml blood and 362,0 mg of chlorate per 100 ml stomach contents.</p>	
Public literature	Sodium chlorate	800 mg/kg bw (LD50 adult female)	The oral LD ₅₀ in adult women is reported to be 800 mg/kg bw	Lewis, R.J., Sr., 1996
Public literature	Potassium chlorate	No death, survived 600 mg/kg bw (infant)	A 3 month old boy survived ingestion of 3g sodium chlorate.	Vakili M, 1977
Public literature	Potassium chlorate	100 mg/kg bw (adult)	A 76 year old woman died after ingesting a table spoon, about 7 g, of potassium chlorate.	Fukumoto K, Fukumoto H, 1970
Public literature	Potassium chlorate	Unknown	In 1911 reports of 143 cases of poisoning were reported, 116 with a fatal outcome.	Witthaus 1911 cited in Clinical Toxicology, London, 1969
Public literature	Potassium chlorate	Unknown	7 fatal cases were reported between 1911-1940. 6 accidental and 1 suicide.	Cochrane 1940 cited in Clinical Toxicology, London, 1969
Public literature	Sodium chlorate	Unknown	Sodium chlorate poisoning is uncommon. 1 case reported in 1967.	General Register Office, London, 1969
Public literature	Sodium chlorate	No death, survived, 186 mg/kg bw (adult)	55 year old man swallowed 13 g	Davies 1956 cited in Clinical Toxicology, London, 1969
Public literature	Sodium chlorate	No death, survived, 200 mg/kg bw (adult)	<p>Two cases of renal failure due to sodium chlorate poisoning.</p> <p>67 year old female ingested 14 g of sodium chlorate and survived.</p> <p>In total 12 cases were reported (including the 10 reported by Derot 1948) in this publication and 8 were accidental poisonings.</p>	Jackson 1962 cited in Clinical Toxicology, London, 1969
Public literature	Sodium chlorate	200-429 mg/kg bw (adult)	6 out of 10 cases died, the fatal dose was about 30 g, one person died after 14 g.	Derot 1948 cited in Clinical Toxicology, London, 1969
Public literature	Potassium chlorate	Unknown	5 year old girl swallowed a 2% solution of potassium chlorate, exposed for 7 days and died after 10 days.	Ehnbom 1889 cited in Clinical Toxicology, London, 1969
Public literature	Potassium chlorate	1300 mg/kg bw (adult)	35 year old woman died after consuming tablets of potassium chlorate for 5 days, in total 91 g	Pharm. J. 1950 cited in Clinical Toxicology, London, 1969
Public literature	Potassium chlorate	No death, survived 429-500 mg/kg bw (adult)	Patient received 30-35 g for 3 days.	Cochrane 1940 cited in Clinical

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				Toxicology, London, 1969
Public literature	Potassium chlorate	571 mg/kg bw (adult)	48 year old woman drank 150-200 g of water with 40 g	Balsazs, 1934 cited in Clinical Toxicology, London, 1969
Public literature	Potassium chlorate	107 mg/kg bw (adult)	Man swallowed 7.5 g included in tooth paste.	Bernstein, 1930 cited in Clinical Toxicology, London, 1969
Public literature	Potassium chlorate	267-333 mg/kg bw (child)	8 year old boy was poisoned with an unknown amount, estimated 4-5 g	Wagner, 1934 cited in Clinical Toxicology, London, 1969
Public literature	Sodium chlorate	Unknown	78 year old man was poisoned	Clinical Toxicology, London, 1969
Public literature	Sodium chlorate	333-667 mg/kg bw (child)	17 year old boy with down syndrome	Clinical Toxicology, London, 1969
Public literature	Sodium chlorate	No death, survived, 1071 mg/kg bw (adult) 1286 mg/kg bw (adult)	18 year old man 75 g chlorate in water, survived 78 year old man 90 g chlorate in water, died	O'Grady J, Jarecsni E, 1971
Public literature	Sodium chlorate	Unknown	57 year old man, 43 year old man and a 19 year old man committed suicide by eating chlorate	Timperman J, Maes R, 1966
Public literature	Sodium chlorate	No death, survived, 714 mg/kg bw (adult)	23 year old woman consumed about 50 g and survived	Yoshida Y et al., 1977
Public literature	Sodium chlorate	Unknown	13 year old boy tasted sodium chlorate by dipping his finger in the crystals and licking it. He became ill but survived.	Stavrou et al., 1978
Public literature	Sodium chlorate	No death, survived, 571 mg/kg bw (adult)	22 year old male ingested 40 g and survived. 57 year old male ingested an unknown amount and survived.	Granier P et al., 1985

a) unknown means that the original article could not be retrieved

Table 10: Summary table of other studies available for acute oral toxicity

Method, guideline, deviations if any	Species, strain, sex, no/group	Test substance,	Dose levels, duration of exposure	Value LD ₅₀	Reference
Not specified, acute study	Dog, collie and boxer, 5 in total, sex not specified	Sodium chlorate	one dog: 0.5 g/kg bw two dogs: 1 g/kg bw one dog: 2 g/kg bw	One of the the 1 g/kg bw dogs died (boxer) and the 2 g/kg bw dog died (collie)	Sheahan, 1971
Not specified, acute study	Not specified	Sodium chlorate	Not specified	1200 - 7000 mg/kg bw	Ben-Dyke, 1970
Not specified, acute study	Rat , no further details specified	Sodium chlorate	Not specified	7-8 g/kg bw	Frank, 1948 in Smith et al, 2012
Not specified, acute study	Rat , no further details specified	Sodium chlorate	Not specified	1200 mg/kg bw	Edson, 1960 in Smith et al, 2012

10.1.1 Short summary and overall relevance of the provided information on acute oral toxicity

Animal studies

The key animal study (Study report, 1991i) was performed in accordance with EPA Acute Toxicity Guideline, OPP 81-1 (equivalent to OECD Guideline 401, Acute Oral Toxicity) on sodium chlorate in compliance with GLP. The test material, Sodium Chlorate Crystal, was evaluated for its acute oral toxicity potential in 30 Sprague Dawley rats. Ten animals were used in a dose range finding study (dose levels: 5, 2.5, 1.25, 0.6 and 0.3 g/kg bw). Thereafter Sodium Chlorate was administered as gavage doses (5.0 g/kg and second (2.0 g/kg) in a first limit test. No mortality occurred in animals dosed at 2.0 g/kg and 1 animal died at dose level 5.0 g/kg. Clinical signs of toxicity at 5.0 g/kg included hunched posture and reduced feces, which were no longer evident on Day 3. At 2.0 g/kg only hunched posture was observed at 2 -4 hours post dosing in one male. There was no significant effect on body weight gain in animals surviving to termination. Necropsy findings at 5.0 g/kg showed green discoloration of the intestines, a light green fluid in the stomach, pink liquid in the abdominal cavity and dark red lung discoloration. At 2.0 g/kg only slight to moderate redness in the lungs of all animals was observed. Conclusions: The acute oral LD50 of Sodium Chlorate Crystal was determined to be greater than 5000 mg/kg bw.

A valid supporting study (Study report, 1981) was equivalent to OECD Guideline 401 (Acute Oral Toxicity). The study was not designed and performed according to GLP. The test material, Sodium Chlorate, was evaluated for its acute oral toxicity potential in Charles River CD rats. Twelve animals were used in a range finding study (dose levels: 5000, 1500 and 1000 mg/kg bw). During the main study Sodium Chlorate was administered as gavage doses at levels of 10000, 6810, 4640, 3160, 2150 and 1470 mg/kg to 8 males and 8 females per dose group, with the exception of 10000 mg/kg dose in which 7 females were dosed and 1470 mg/kg dose in which 8 males were dosed. Mortality occurred in 10 males dosed at the 4640 mg/kg and 6810 mg/kg level. In total 12 females died at the 6810 mg/kg level and the 10000 mg/kg level. Clinical signs of toxicity included ataxia at dose levels greater than 2150 (males) and 4640 (females) mg/kg bw. At dose levels greater than 3160 (male) and 2150 (female) mg/kg bw signs of decreased motor activity, yellow semi-solid discharge from the anus and yellow wet fur around the inguinal and perianal regions were observed. The animals that died during the study showed discoloration of the thoracic and abdominal organs. Necropsy findings among survivors consisted of one male rat at 4640 mg/kg bw which exhibited a slightly mottled right kidney. There was a small gain in body weight in animals surviving to termination.

Conclusions: The acute oral LD50's of Sodium Chlorate were determined to be ca. 4950 mg/kg in males and ca. 6250 mg/kg in females.

A review from 2012 (Smith et al., 2012) shows that single or short duration (<3 d) exposures to oral chlorate at concentrations < 150 mg/kg bw have not produced acute toxicity or clinical signs (labored breathing, methemoglobinemia) in cattle, chicken, horse, rabbit, sheep, or swine.

The study of Sheahan (1971) is included as supporting evidence but is of limited reliability due to study design and poor reporting. The additional studies in table 10 are of very limited reliability due to lack of information on the studies and are only included for completeness and are thus not considered in the assessment of acute oral toxicity for sodium chlorate.

In conclusion, animal studies (rat and dog) with sodium chlorate show a low acute toxicity after oral (LD50 = 4950-6250 mg/kg bw) exposure (Study report, 1991i, Study report, 1981, Sheahan 1971).

In the records on the decision on classification of sodium chlorate from TC C&L, 1989 a LD50 of 1200 mg/kg bw in rat was identified. However, the study report is not available to the dossier

submitter. The EFSA scientific opinion (Risks for public health related to the presence of chlorate in food, EFSA 2015) also mention that “other publications” reported oral LD50 for sodium chlorate to be 1200 mg/kg b.w. (equivalent to 936 mg chlorate/kg bw) in rats (Lewis, 1996; HSDB, 2003; as cited in EFSA scientific opinion 2015) and the oral LD50 for potassium chlorate to be 1 870 mg/kg bw (equivalent to 1272 mg chlorate/kg bw) in rats (RTECS, 1994; as cited in EFSA scientific opinion 2015). In the Registry of Toxic Effects of Chemical Substances from NIOSH the oral LD50 in rat was 1870 mg/kg. None of these rat studies are available to the dossier submitter to be able to assess quality and reliability. These studies would, if considered as sufficiently robust, justify classification in category 4.

Human data

Numerous human case studies are reported for sodium chlorate. In table 9, several case reports cited originally in the dossier for OECD cooperative Chemicals Assessment Programme, High production volume (HPV) chemicals and included in the REACH dossier are listed. Most are only abstract and a robust summary is therefore not provided in annex I of this report.

The human case studies are described accidental poisoning (Clinical toxicology, 1969; Casey P, Vale JA, 1994; Eysseric H et al, 2000; Fukumoto K, Fukumoto H, 1970; General Register Office, London, 1969; Helliwell M, Nunn J, 1979; Jackson RC et al, 1961; Vakili M, 1977; Stavrou et al. 1978), suicide (Bloxham CA et al., 1979; Granier P et al., 1985; General Register Office, London, 1969; Jackson RC et al, 1961; Klendshoj NC et al., 1962; O’Grady J, Jarecsni E, 1971; Oliver JS, Smith H, Watson AA, 1972; Steffen C, Seitz R., 1981; Timperman J, Maes R, 1966; Yoshida Y et al., 1977) and homicide attempts (Jansen H, Zeldenrust J, 1972).

In summary, the studies report that doses of 5 to 10 grams (71-143 mg/kg bw³) can be fatal in adults, and doses of 2 grams (0.2 g/kg bw³) in children. But also multiple cases are described surviving intakes ranging from 40 g (571 mg/kg bw³) to even 150-200 grams (2.1-2.9 g/kg bw³). This is likely related to the possibility of dialysis treatment in case of renal failure after 1960s.. In many cases, the lethal dose in human are above 20 g (285 mg/kg bw³) (Helliwell and Nunn, 1979). The oral LD50 in adult women is reported to be 800 mg/kg (Lewis, 1996). The SIDS Initial Assessment Report on Sodium chlorate summarises that lethality is reported from after 4 hours up to 34 days, with an average of about 4 days, and that the acute toxicity of chlorate is mediated by methemoglobin. In the NTP technical report on the toxicology and carcinogenesis studies of sodium chlorate from 2005 the acute toxicity in humans has been summarised and it was stated that death has been most frequently associated with doses of 20 g or greater, although recovery has been noted in patients who ingested as much as 200 g. Moreover, the report describes that *chlorate toxicity after ingestion in humans can be characterized primarily by gastrointestinal irritation, massive intravascular hemolysis, disseminated intravascular coagulation, cyanosis, and renal failure. Gastrointestinal irritation appears to be the result of a direct effect of the chlorate ion on the gastrointestinal mucosa. The intravascular hemolysis occurs subsequent to the formation of methemoglobin in exposed erythrocytes, eventually resulting in cyanosis. In addition, chlorate exerts a direct toxic effect on the proximal tubule of the kidney, causing necrosis and preventing the formation of urine and subsequent elimination of chlorate from the blood stream, thus prolonging exposure of the erythrocytes.*

Nephrotoxicity also seems mediated by methaemoglobin catalysis. Methaemoglobin thus autocatalytically increases methaemoglobin formation and destruction of the erythrocyte, which is shown in in vitro experiments (Steffen C, Wetzel, 1993).

In an evaluation of sodium chlorate by the French poison control center (2011) a retrospective study was conducted over a period ranging from 1999 to 2009 to identify human cases of exposure to chlorate preparations reported to poison control and toxicovigilance centers. 29 individuals had

³ Estimated intakes per kg bw were calculated with a default body weight assumption of 70 kg for adults, 10 kg for children and 5 kg for infants.

pathological methemoglobinemia (MetHb \geq 3%). The lowest doses at which methemoglobinemia was observed were in the range of 10-20 grams of sodium chlorate taken by ingestion. 13 (45%) of the 29 individuals did not survive. The cause of death related to the hematological and renal effects of chlorates as described above and / or the complications of reanimation. In this study, the lowest estimated oral lethal doses were 8.8 g (125 mg/kg bw⁴), 17.5 g (250 mg/kg bw⁴), and 28.8 g (410 mg/kg bw⁴), of sodium chlorate which are in same range as previously published lowest lethal doses in humans.

Records from the TC C&L from 1989 on the classification of sodium chlorate indicates lowest human lethal doses of 50 mg/kg and 214 mg/kg in adults and a LD50 for children at 185 mg/kg. We could not track the references but the lowest lethal dose 50 mg/kg may have been cited from Gosselin, Clinical Toxicology of Commercial Products (CTCP) 4th ed, 1976 where oral lethal dose is reported to be 50-500 mg/kg for a 70 kg person. The lowest lethal dose 214 mg/kg may have been cited from Helliwell and Nunn, 1979 where the lowest lethal dose was 15 g = 214 mg/kg bw for a 70 kg person.

In the scientific opinion by EFSA (2015), a report from NRC (1980) is cited where lethal doses in adults were estimated to be 20 to 35 g for sodium chlorate and 5 to 30 g for potassium chlorate. Consequently, the oral lethal dose for these salts were 71 to 500 mg/kg (based on adult bw 70 kg).

Summary

Animal studies with sodium chlorate show a low acute toxicity (LD50 > 5000 mg/kg bw) after oral exposure. In contrast, available human data show lethality in humans at lower concentrations of chlorate and the acute toxicity of chlorate has been associated with methemoglobin. There are marked species differences in susceptibility to form methemoglobin where humans appears as more severely affected than rodent species.

10.1.2 Comparison with the CLP criteria

Based on available data from one study of sodium chlorate in rat, the LD50 was reported to be > 5000 mg/kg bw. This does not meet the criteria of classification in Acute Tox. 4 for oral administration. However, information from human poisoning incident reports demonstrate lethal effects of sodium- and potassium chlorate at concentration ranges that warrant classification.

The information from the publications summarized should be viewed with care for deriving an exact acute toxicity estimate for sodium chlorate since a lethal dose which is fatal to 50% (LD50) of the exposed group cannot be derived. Due to vomiting occurring, sometimes rapidly after ingestion, the absorbed quantity is often uncertain. Therefore, variability occurs in the doses causing lethality. However, the data do show that in mg/kg bw humans are more sensitive to acute sodium- and potassium chlorate toxicity when compared to animals.

According to the Guidance on the Application of the CLP Criteria (v.5, July 2017) *“The minimum dose or concentration or range shown or expected to cause mortality after a single human exposure can be used to derive the human ATE directly, without any adjustments or uncertainty factors”*.

The lowest lethal doses or ranges reported in this CLH-proposal are the following:

- 71-142 mg/kg bw in adults (based on assumption of default body weight 70 kg) and 133 mg/kg bw in children (Hartley and Kidd, 1987)
- 100 mg/kg bw in a 76 year old woman (based on assumption of default body weight as 70 kg) (Fukomoto and Fukomoto, 1970)
- 107 mg/kg bw in an adult man (based on assumption of default body weight as 70 kg) (Bernstein cited in Klendshoj 1962)

⁴ Estimated intakes per kg bw were calculated with a default body weight assumption of 70 kg for adults, 10 kg for children and 5 kg for infants.

- 125 mg/kg bw in an 43 year old male (based on assumption of default body weight as 70 kg) (AFSSA report 2011).
- 214 mg/kg bw in an 46 year old woman (based on assumption of default body weight as 70 kg) (Helliwell and Nunn, 1979)

Thus, based on a number of human case reports indicating lowest lethal doses < 300 mg/kg bw the dossier submitter consider that a category 3 classification rather than the current category 4 (minimum classification) is justified. In line with CLP guidance (to use the minimum dose or range shown or expected to cause mortality) for deriving a human ATE and considering that the available human data is quite incoherent we propose to use the converted Acute Toxicity point Estimate (cATpE), which is 100 mg/kg bw for category 3, to set the ATE.

The alternative view of the Registrant is as follows: Gathered from the available human data that the lowest lethal doses were reported to be 5 to 10 grams but that most frequently the lethal doses were above 20 g (Helliwell and Nunn, 1979) the relevant starting point for deriving the ATE is above 286 mg/kg bw. In the light of the quality of the data and related uncertainties the registrant believes there is no logical choice to use the minimum dose as the basis for the ATE, and suggest maintaining the Acute Tox. 4 classification.

The reasoning behind the TC C&L classification (Xn; R22) is not available to the dossier submitter. Therefore we do not know if the human data or the rat LD0 of 1200 was used to compare against the guidance values under DSD for Xn; R22 (200 – 2000 mg/kg bw) which later has been translated into Acute Tox. 4 (oral) under CLP.

10.1.3 Conclusion on classification and labelling for acute oral toxicity

Currently sodium chlorate has a harmonised classification as Acute Tox. 4* (H302) for the oral route of exposure. Based on the weight of the evidence of available human data and using expert judgement the dossier submitter proposes that classification of sodium chlorate in Acute Tox. 3, H301 is warranted with a cATpE of 100 mg/kg bw. A more stringent classification is thus proposed.

10.2 Acute toxicity - dermal route

Not assessed in this CLH-proposal.

10.3 Acute toxicity - inhalation route

Not assessed in this CLH-proposal.

10.4 Skin corrosion/irritation

Hazard class not assessed in this CLH-proposal.

10.5 Serious eye damage/eye irritation

Hazard class not assessed in this CLH-proposal.

10.6 Respiratory sensitisation

Hazard class not assessed in this CLH-proposal.

10.7 Skin sensitisation

Hazard class not assessed in this CLH-proposal.

10.8 Germ cell mutagenicity

Hazard class not assessed in this CLH-proposal.

10.9 Carcinogenicity

Hazard class not assessed in this CLH-proposal.

10.10 Reproductive toxicity

Hazard class not assessed in this CLH-proposal.

10.11 Specific target organ toxicity-single exposure

Hazard class not assessed in this CLH-proposal.

10.12 Specific target organ toxicity-repeated exposure

Hazard class not assessed in this CLH-proposal.

10.13 Aspiration hazard

Hazard class not assessed in this CLH-proposal.

11 EVALUATION OF ENVIRONMENTAL HAZARDS

In water sodium and potassium are naturally present and the amounts added with the test substance are not considered to have an impact on the total concentration and on the test result. Sodium is the most abundant of the alkali metals, the fifth most abundant metal in the Earth's crust with an average value of 22,700 mg/kg, and the principal cation in sea water, at a typical concentration 10,500 mg/l. Sodium values in stream water range over four orders of magnitude, from 0.23 to 1284 mg/l, with a median value of 6.58 mg/l. Potassium occurs in various minerals, from which it may be dissolved through weathering processes. Seawater contains about 400 mg/l potassium. It tends to settle, and consequently ends up in sediment mostly. Rivers generally contain about 2-3 mg/l potassium.

The counter ion present is therefore not relevant for the test results and will not contribute to the effects caused by the substance.

Many physico-chemical properties are not relevant or cannot be derived for inorganic substances. Water solubility was measured for both substances and as can be seen in the table below, both substances are readily soluble in water. There is also limited aquatic ecotoxicity data available for potassium chlorate. For both sodium- and potassium chlorate studies with marine algae were performed. From those studies it can be concluded that both substances are non-toxic to marine algae with NOEC values greater than 100 mg/l.

Sodium and potassium chlorate are strong acids with pKa values in the range of -1 to -3 (theoretical range⁵), meaning that both sodium and potassium chlorate almost totally dissociated in water, producing sodium/potassium cations and chlorate anions.

The conclusions drawn for sodium chlorate are also valid for potassium chlorate and vice versa.

All results depicted in the tables in the sections below are expressed as test substance (Na- or KClO₃) as well as Chlorate ion. Results have been converted to test substance or Chlorate ion by molecular weight of the different species.

MW (g/mol):

- NaClO₃: 106.44
- KaClO₃: 122.55
- ClO₃⁻: 83.45

Example calculation:

$$\begin{aligned} \text{EC50} &= 1000 \text{ mg NaClO}_3/\text{l} \\ 1000/106.44 \times 83.45 &= 784 \text{ mg ClO}_3^-/\text{l} \end{aligned}$$

11.1 Rapid degradability of organic substances

Not applicable since sodium chlorate is an inorganic compound.

11.2 Environmental transformation of metals or inorganic metals compounds

Not applicable since sodium chlorate is not a metal compound.

11.3 Environmental fate and other relevant information

⁵ Expert statement: An experimental determination of the dissociation constant of sodium chlorate is not possible due to its strong acidity and because the sodium chlorate decomposes on acidification and produces toxic chlorine dioxide and chlorine gas. Based on the available information, the pK_a of sodium chlorate is in the range of -1 to approx. -3 (Weissenfeld, 2004).

11.3.1 Ready biodegradability

Biotic conversions of sodium chlorate, an inorganic substance should not be assessed in standard OECD TG 301 tests for ready biodegradability, and OECD TG 302 tests for inherent biodegradability because these tests only detect biodegradation of organic compounds under aerobic conditions. The attempt of L'Haridon (2003) to detect biodegradation of sodium chlorate in the Sturm test (OECD TG 301 B) using a specific analysis of chlorate was therefore unsuccessful. Degradation of sodium chlorate in the Sturm test was thought to be possible by L'Haridon (2003) because of the existence of anaerobic niches within the sludge particles used as inoculum. These anaerobic niches do occur in properly operated biological wastewater treatment plants (high activated sludge concentrations and low oxygen levels of ~2 mg/L) but not in an OECD TG 301 tests (low level of activated sludge and oxygen levels of >>9 mg/L). Moreover, the amount of biodegradable reducing agents in a standard OECD TG 301 test is limiting also preventing chlorate reduction.

“Ready” biodegradability of sodium chlorate transformation can be shown easily using the methodology of the Closed Bottle test (OECD TG 301 D) with one major modification (van Ginkel et al, 1995). The test was modified by adding excess amounts of reducing agents such as fatty acids, amino acids, carbohydrates. A minor part of the reducing agent was oxidized with the molecular oxygen present in the bottles thereby creating anaerobic conditions. The tests were inoculated with low concentrations of activated sludge, soil, digested sludge or dilutions of river and ditch water in line with the OECD TG 301. Complete removal of chlorate was achieved within 28 days with all inocula tested and most reducing agents.

The ease with which chlorate reduction occurs naturally is also demonstrated by Bryan and Rohlich (1954) who used chlorate reduction as a measure for the Biological Oxygen Demand (BOD) showing that chlorate is rapidly reduced by microorganisms using organic compounds as carbon and energy source present in sewage.

A valid ready biodegradability test result is not available for sodium chlorate because chlorate is an electron acceptor like molecular oxygen. Nevertheless all aspects important for achieving a ready biodegradability test result i.e. ultimate (complete) biodegradation, rate of biodegradation and number and occurrence of competent micro-organisms present in “unacclimated” ecosystems and biological treatment plants have been investigated (see above). Ready biodegradability tests only detect growth-linked biodegradation. Microorganisms are capable of growth on sodium chlorate in the presence of reducing agents under anaerobic conditions. The biodegradation pathway proves that chlorate is reduced completely to chloride. The biodegradation kinetics of chlorate have been determined with mixed and pure cultures. The maximum growth rates of chlorate reducing microorganisms range from 0.04 to 0.56h⁻¹, which is comparable or much higher than growth rates of nitrifying bacteria. Ammonium is oxidized readily in OECD TG 301 tests due to these nitrifying bacteria. Painter and King (1983) used a model based on the Monod equation to interpret the biodegradation curves in ready biodegradability tests. According to this model, growth rates of competent micro-organisms of 0.01 h⁻¹ or higher do result in a ready biodegradation of the test substance. Reduction of chlorate has been detected in terrestrial ecosystems, fresh water, marine environment, compost, and aquifers. These findings demonstrate the wide distribution of chlorate-reducing micro-organisms and that sodium chlorate can be considered as readily biodegradable.

No standard tests are available for sodium chlorate, but from tests only deviating from OECD TG 301 TG methodology with respect to the absence of oxygen (van Ginkel et al., 1995) it can be concluded that sodium chlorate is readily biodegradable.

Hence, in applying a weight of evidence approach to this specific case it can be concluded that the substance should be considered as rapidly degradable for classification purposes.

Up to recently, perchlorate and chlorate were thought to be primarily antropogenic. Recent evidence makes a strong case for more widespread natural occurrence of perchlorate, outside of the long-established occurrence in caliches of the Atacama Desert in Chile. Improved sensitivity of perchlorate detection techniques shows widespread existence of ppb levels of perchlorate. Not all perchlorate

detected could be traced to anthropogenic sources. Natural perchlorate in soils is rare but occurs in other arid environments at levels up to 0.6 weight %. In the southern high plains groundwater, perchlorate is better correlated with iodate, known to be of atmospheric origin, compared to any other species (Dasgupta et al, 2005).

Natural perchlorate may be formed from chloride aerosol by electrical discharge and by exposing aqueous chloride to high concentrations of ozone (Bao and Gu, 2004; Bohlke et al 2005). Information regarding the perchlorate formation process is however, still largely unknown. Perchloric acid is the stable end product of the atmospheric chemistry because of its resistance to photolysis (Simonaitis and Heicklen, 1975) and occurs in aerosols in stratosphere of the earth at 0.5 to 5 parts per trillion (Murphy and Thomson, 2000). Perchlorate was also detected in rain and snow samples. This strongly suggests that some perchlorate is formed in the atmosphere and a natural perchlorate background of atmospheric origin should exist. In soils and surface waters perchlorate is reduced via chlorate. Chlorate is therefore part of natural chloro-oxy acid cycle (Figure 1). The existence of a chloro-oxy acid cycle does explain the enormous potential for chlorate reduction in the environment.

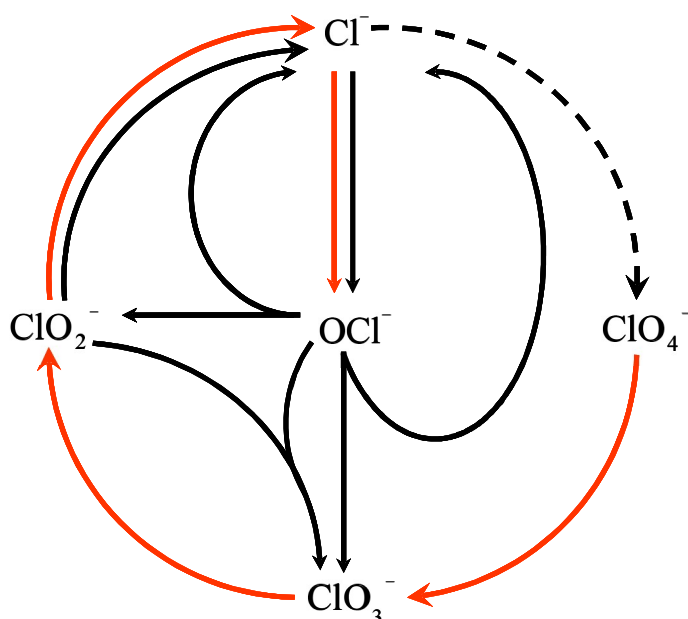


Figure 1: Chloro-oxo acid cycle. The dashed arrow represents the recent findings of perchlorate formation in aerosols. The red arrow are reactions catalysed by enzymes present in (per)chlorate respiring bacteria, nitrate reductases and peroxidases (formation of hypochlorite). The black arrows indicate chemical reactions occurring under ambient environmental temperatures.

11.3.2 Hydrolysis

Sodium chlorate is highly soluble in water (696-736 g/L at 20°C for pH 4.49 to 8.70 at 20°C, see table 7). Sodium chlorate in aqueous solutions is known for its chemical stability under environmental conditions (Urbanski 1998).

Based on the chemical structure it is expected that sodium chlorate is resistant to hydrolysis. Due to its chemical structure, sodium chlorate is considered as stable in sterile water whatever the pH is. There are no relevant metabolites, degradation or reaction products that necessitate studies to answer this point.

Sodium chlorate is rapidly biodegradable. Studies on the hydrolysis as a function of the pH are therefore not required.

11.3.3 Other convincing scientific evidence

11.4 Bioaccumulation

Although no measured data on bioaccumulation were identified, based on the environmental fate and behaviour of the substance (the complete dissociation in water due to low dissociation constant and the high water solubility) no significant bioaccumulation is expected.

11.5 Acute aquatic hazard

For all trophic levels, tests according to international standard guidelines were performed in compliance with GLP.

Table 6: Summary of relevant information on acute aquatic toxicity

IUCLED section	Method	Species	Test material	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Remarks	Reference	Klimish score	Adequacy of study
6.1.1	EPA OPP 72-1	<i>Oncorhynchus mykiss</i>	Sodium chlorate	LC ₅₀ (96h) >1000 mg/l (nominal)	LC ₅₀ (96h) >784 mg/l (nominal)	Fresh-water species	Study report (1991a)	1	Key study
6.1.1	EPA OPP 72-3	<i>Cyprinodon variegatus</i>	Sodium chlorate	LC ₅₀ (96h) >1000 mg/l (nominal)	LC ₅₀ (96h) >784 mg/l (nominal)	Marine species	Study report (1991c)	1	Key study
6.1.3	EPA OPP 72-2	<i>Daphnia magna</i>	Sodium chlorate	EC ₅₀ (48h) >1000 mg/l (nominal)	EC ₅₀ (48h) >784 mg/l (nominal)	Fresh-water species	Study report (1991d)	1	Key study
6.1.3	In-house method	<i>Mysidopsis bahia</i>	Sodium chlorate	LC ₅₀ (96h) >1000 mg/l (nominal)	LC ₅₀ (96h) >784 mg/l (nominal)	Marine species	Study report (1991f)	2	Key study
6.1.5	EPA OPP 122-2	<i>Selenastrum capricornutum</i>	Sodium chlorate	E _b C ₅₀ (72h) = 129 mg/l (nominal) E _b C ₅₀ (96h) = 133 mg/l (nominal)	E _b C ₅₀ (72h) = 101 mg/l (nominal) E _b C ₅₀ (96h) = 104 mg/l (nominal)	Fresh-water species	Study report (1991e)	1	Supporting study
6.1.5	According to own protocol	<i>Nitzschia closterium</i>	Potassium chlorate	15 mg NO ₃ ⁻ /l; E _r C ₅₀ (72h) > 735 mg/l (nominal)	15 mg NO ₃ ⁻ /l; E _r C ₅₀ (72h) > 500 mg/l (nominal)	Marine species	Stauber J.L. (1998)	2	Key study
6.1.6	OECD 221	<i>Lemna minor</i>	Sodium chlorate	Biomass growth: EC ₅₀ (7d) = 73.7 mg/l (nominal) Growth rate: EC ₅₀ (7d) = 134 mg/l (nominal) Biomass dry weight: EC ₅₀ (7d) = 128 mg/l (nominal)	Biomass growth: EC ₅₀ (7d) = 57.5 mg/l (nominal) Growth rate: EC ₅₀ (7d) = 105 mg/l (nominal) Biomass dry weight: EC ₅₀ (7d) = 100 mg/l (nominal)	Fresh-water species	Study report (2003)	1	Key study
6.1.9	U.S. EPA-FIFRA, Guideline 72-3	<i>Crassostrea virginica</i>	Sodium chlorate	EC ₅₀ (96h) > 1000 mg/l LC ₅₀ (96h) > 1000 mg/l (nominal)	EC ₅₀ (96h) > 784 mg/l LC ₅₀ (96h) > 1000 mg/l (nominal)	Marine species	Study report (1991g)	2	Other information
6.1.9	ISO/DC 20666	<i>Brachionus plicatilis</i>	Sodium chlorate	EC ₅₀ (96h) = 596 mg/l	EC ₅₀ (96h) = 467 mg/l	Marine species	Study report (2010b)	1	Supporting study

11.5.1 Acute (short-term) toxicity to fish

Five acute toxicity studies were found for fresh water fish of which four are valid and one is valid with restrictions (Toussaint, et al. (2001) because the test was not performed according to standard test protocol (see table 12). One valid test on marine fish was also found.

Table 7. Acute toxicity to fish

Method	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Test material	Remarks	Reference	Klimish score	Adequacy of study
GLP, Flow through test, 96 hours, according to EPA OPP 72-1 Key study (fresh water)	LC ₅₀ >1000 mg/l	LC ₅₀ >784 mg/l	Sodium chlorate	<i>Oncorhynchus mykiss</i>	Study report (1991a)	1	Key study
GLP, Flow through test, 96 hours, according to EPA OPP 72-1	LC ₅₀ >1000 mg/l	LC ₅₀ >784 mg/l	Sodium chlorate	<i>Lepomis macrochirus</i>	Study report (1991b)	1	Supporting study
GLP, Semi-static test, 96 hours, according to OECD 203	LC ₅₀ >1000 mg/l	LC ₅₀ >784 mg/l	Sodium chlorate	<i>Brachydanio rerio</i>	Study report (1991h)	1	Supporting study
GLP, Semi-static test, 96 hours, according to OECD 203	LC ₅₀ >1000 mg/l	LC ₅₀ >784 mg/l	Sodium chlorate	<i>Pimephales promelas</i>	Study report (1993)	1	Supporting study
96 hours test according to own protocol	LC ₅₀ = 2585 mg/l	LC ₅₀ = 2027 mg/l	Sodium chlorate	<i>Oryzias latipes</i>	Toussaint, et al. (2001)	2	Supporting study
GLP, Flow through test, 96 hours, according to EPA OPP 72-3 Key study (marine)	LC ₅₀ >1000 mg/l	LC ₅₀ >784 mg/l	Sodium chlorate	<i>Cyprinodon variegatus</i>	Study report (1991c)	1	Key study

All studies can be found in IUCLID in section 6.1.1

For all studies LC₅₀s >1000 mg/l were observed. In the first two studies (Study report (1991a) and (1991b)) with *Oncorhynchus mykiss* and *Lepomis macrochirus* chemical analysis of the stock solution was performed. The measured concentrations were 105 and 103% of the nominal concentration and therefore the nominal test concentrations were used to derive the endpoints. Except of the stock solution, no chemical analysis was performed during the test. But it was considered that the concentration of sodium chlorate was stable during the test.

The LC₅₀ for fresh water fish is greater than 1000 mg/l.

Similar to fresh water fish, the LC₅₀ of sodium chlorate for marine fish (Study report, 1991c) was also greater than the highest test concentration of 1000 mg/l. This indicates that there is no influence of the marine conditions on the toxicity of sodium chlorate.

11.5.2 Acute (short-term) toxicity to aquatic invertebrates

Two valid studies with *Daphnia magna* are available (see table 13). Both studies were performed in compliance with GLP and according to standard protocol. In both studies chemical analyses were carried out, but Study report (1991d) only analyses on the stock solution were performed. In study report (1995), the endpoint was expressed in mg chlorate ion per liter, this was transformed to mg sodium chlorate per liter for the purpose of this classification proposal.. The original value was 919.3 mg chlorate/l. The results of the study performed in 1995 were heterogeneous and must therefore be

considered reliable with restrictions, but nevertheless the results confirm the outcome of the study report of 1991d.

One study valid with restrictions is available for the marine crustacean *Mysidopsis bahia* (Study report, 1991f).

Table 8. Acute toxicity to invertebrates

Method	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Test material	Remarks	Reference	Klimish score	Adequacy of study
Flow through test, 48 hours, according to EPA OPP 72-2 Key study (fresh water)	EC ₅₀ >1000 mg/l	EC ₅₀ >784 mg/l	Sodium chlorate	<i>Daphnia magna</i>	Study report (1991d)	1	Key study
Static test, 48 hours, according to EPA OPP 72-2	EC ₅₀ = 1172 mg/l	EC ₅₀ = 919.3 mg/l	Sodium chlorate	<i>Daphnia magna</i>	Study report (1995)	2	Supporting study
In-house method Key study (marine)	LC ₅₀ > 1000 mg/l	LC ₅₀ >784 mg/l	Sodium chlorate	<i>Mysidopsis bahia</i>	Study report (1991f)	2	Key study

All studies can be found in IUCLID in section 6.1.3

The EC50 for fresh water and marine invertebrates is greater than 1000 mg/l, showing that sodium chlorate is not harmful to aquatic organisms

11.5.3 Acute (short-term) toxicity to algae or other aquatic plants

Algae

Two studies on sodium chlorate are available, one on *Selenastrum capricornutum* and one on *Scenedesmus subspicatus* were found (see table 14). One was valid without restrictions (study report, 1991e) and the other one was valid with restrictions because not all details on the results were provided.

The E_bC₅₀-value of the study with *Scenedesmus subspicatus* (Study report, 2004c) was higher than 1592.3 mg/l, because 50% inhibition was not reached an E_rC₅₀ could not be determined. The Fe₂O₃ present in the test substance interfered with the spectrofotometrical measurements and increased the extinction, this is not considered to have a significant impact on the results of this test. In the calculation of the biomass a correction is made for the higher extinctions measured at t=0. The higher values at t=0 do not have an influence on the slope of the growth curve, which is used for the calculation of the E_bC₅₀.

Table 9. Toxicity to fresh water aquatic algae

Method	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Test material	Remarks	Reference	Klimish score	Adequacy of study
96 hours test according to EPA OPP 122-2	Cell growth: E _b C ₅₀ -72h = 129 mg/l E _b C ₅₀ -96h = 133 mg/l	Cell growth: E _b C ₅₀ -72h = 101 mg/l E _b C ₅₀ -96h = 104 mg/l	Sodium chlorate	<i>Selenastrum capricornutum</i>	Study report (1991e)	1	Supporting study
72 hours test according to OECD 201	Biomass: E _b C ₅₀ > 1592.3 mg/l	Biomass: E _b C ₅₀ > 1248.4 mg/l	Sodium chlorate	<i>Scenedesmus subspicatus</i>	Study report (2004c)	2	Supporting study

All studies can be found in IUCLID in section 6.1.5

The lowest acute value for freshwater algae was found for *Selenastrum capricornutum*. The E_bC_{50} was 129 mg/l after 72 hours.

One valid test with marine algae is available (Study report, 2010a) (see table 15).

Table 10. Toxicity of sodium chlorate to marine algae

Method	Results Expressed as Na/KClO ₃	Expressed as ClO ₃ ⁻	Test material	Remarks	Reference	Klimish score	Adequacy of study
72 hours test according to ISO 10253 guideline	EC ₅₀ > 1000 mg/l	EC ₅₀ > 784 mg/l	Sodium chlorate	<i>Skeletonema costatum</i>	Study report (2010a)	1	Supporting study

All studies can be found in IUCLID in section 6.1.5

Study report (2010a) was performed as a standard test with *Skeletonema costatum* according to guideline and GLP. Chemical analyses were performed on the test concentrations. The report found that *S. costatum* was not sensitive to sodium chlorate, with an EC₅₀ greater than 1000 mg/l.

Two tests (valid with restriction) were found for marine algae performed with potassium chlorate (see table 16).

Table 11. Toxicity of potassium chlorate to marine algae

Method	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Test material	Remarks	Reference	Klimish score	Adequacy of study
72 hours test according to own protocol with potassium chlorate	15 mg NO ₃ ⁻ /l: E _r C ₅₀ > 1469 mg/l	15 mg NO ₃ ⁻ /l: E _r C ₅₀ > 1000 mg/l	Potassium chlorate	<i>Dunaliella tertiolecta</i>	Stauber J.L. (1998b)	2	Supporting study
72 hours test according to own protocol with potassium chlorate	15 mg NO ₃ ⁻ /l: E _r C ₅₀ > 735 mg/l	15 mg NO ₃ ⁻ /l: E _r C ₅₀ > 500 mg/l	Potassium chlorate	<i>Nitzschia closterium</i>	Stauber J.L. (1998a)	2	Key study

All studies can be found in IUCLID in section 6.1.5

The tests were performed with potassium chlorate and originally the endpoints were expressed in mg chlorate/l. The original data for the EC₅₀ was for *Dunaliella tertiolecta* > 1000 mg chlorate/l and for *Nitzschia closterium* > 500 mg chlorate/l. The endpoints presented in Table 16 are recalculated to mg potassium chlorate/l for comparison with the studies on sodium chlorate.

The test was carried out at three different nitrate levels namely <0.005, 1 and 15 mg nitrate/l. At the lower nitrate concentrations cell growth in the controls was not according to the standard criteria and these results cannot be used therefore. Only valid results are given in the table above corresponding to a concentration of nitrate used in the standard bioassay (i.e. 15 mg nitrate/l).

The difference in toxicity noted between marine and freshwater algae, appears to be related more to the relative difference in concentration of nitrate in freshwater and marine compartments than to different mechanisms of toxicity between species. The concentration of nitrate in the test water influences the effect concentration of chlorate indicating that competitive inhibition occurs between nitrate and chlorate with excess nitrate inhibiting chlorate toxicity. This is supported by the acute studies on marine species using chlorate at several nitrate concentrations.

Aquatic plants

One valid study was found for freshwater aquatic plants (see table 17).

Table 12. Toxicity to aquatic plants

Method	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Test material	Remarks	Reference	Klimish score	Adequacy of study
7 days test according to OECD 221 Key study (fresh water)	Biomass growth: EC ₅₀ = 73.7 mg/l Growth rate: EC ₅₀ = 134 mg/l Biomass dry weight: EC ₅₀ = 128 mg/l	Biomass growth: EC ₅₀ = 57.5 mg/l Growth rate: EC ₅₀ = 105 mg/l Biomass dry weight: EC ₅₀ = 100 mg/l	Sodium Chlorate	<i>Lemna minor</i>	Study report (2003)	1	Key study

All studies can be found in IUCLID in section 6.1.6

Lemna minor is for sodium chlorate the most sensitive freshwater species tested. The lowest value for acute toxicity to freshwater plants is EC₅₀ of 73.7 mg/l based on biomass growth of *Lemna minor*.

This indicates that sodium chlorate is not harmful to aquatic plants

11.5.4 Acute (short-term) toxicity to other aquatic organisms

One short-term study valid with restrictions was found for molluscs (see table 18). From this study (Study report, 1991g) it can be seen that marine molluscs are not sensitive to sodium chlorate with an EC₅₀ value based on shell growth and an LC₅₀ value both greater than 1000 mg/l.

Another study with the marine rotatoria *Brachionus plicatilis* is available (study report, 2010b). There was a dose dependent reduction in reproduction observed for *B. plicatilis*, when exposed to sodium chlorate. The EC₅₀ was calculated to be 596 mg/l. Mortality of parent rotatoria was not observed at any concentration so EC₅₀ for parent mortality is greater than 1000 mg/l.

Table 13. Toxicity to other aquatic organisms

Method	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Test material	Remarks	Reference	Klimish score	Adequacy of study
96h test according to U.S. EPA-FIFRA, Guideline 72-3	EC ₅₀ > 1000 mg/l LC ₅₀ > 1000 mg/l	EC ₅₀ > 784 mg/l LC ₅₀ > 784 mg/l	Sodium chlorate	<i>Crassostrea virginica</i>	Study report, (1991g)	2	Other information
ISO/DC 20666	EC ₅₀ (96h) = 596 mg/l	EC ₅₀ (96h) = 467 mg/l	Sodium chlorate	<i>Brachionus plicatilis</i>	Study report (2010b)	1	Other information

All studies can be found in IUCLID in section 6.1.9

11.6 Long-term aquatic hazard

Table 14: Summary of relevant information on chronic aquatic toxicity

IUCRID section	Method	Species	Test material	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Remarks	Reference	Klimish score	Adequacy of study
6.1.2	OECD 210	<i>Danio rerio</i>	Sodium chlorate	NOEC (36d) ≥ 500 mg/l (nominal)	NOEC (36d) ≥ 392 mg/l (nominal)	Fresh-water species	Study report (2004a)	1	Key study
6.1.4	OECD 211	<i>Daphnia magna</i>	Sodium chlorate	NOEC (21d) ≥ 500 mg/l (nominal)	NOEC (21d) ≥ 392 mg/l (nominal)	Fresh-water species	Study report (2004b)	1	Key study
6.1.5	EPA OPP 122-2	<i>Selenastrum capricornutum</i>	Sodium chlorate	Cell growth: NOEC (96h) = 62.5 mg/l (nominal)	Cell growth: NOEC (96h) = 49.0 mg/l (nominal)	Fresh-water species	Study report (1991e)	1	Supporting study
6.1.5	72 hours test according to own protocol based in ISO	<i>Phaeodactylum tricornutum</i>	Sodium chlorate	NOE _s C = 64 mg/l NOE _r C = 128 mg/l (nominal)	NOE _s C = 50 mg/l NOE _r C = 100 mg/l (nominal)	Marine species	Study report (1994b)	2	Supporting study
6.1.5	72 hours test according to own protocol with potassium chlorate	<i>Nitzschia closterium</i>	Potassium chlorate	15 mg NO ₃ ⁻ /l: NOE _r C = 147 mg/l (nominal)	15 mg NO ₃ ⁻ /l: NOE _r C = 100 mg/l (nominal)	Marine species	Stauber J.L. (1998a)	2	Key study
6.1.6	OECD 221	<i>Lemna minor</i>	Sodium chlorate	NOEC (7d) = 10 mg/l	NOEC (7d) = 7.8 mg/l	Fresh-water species	Study report (2003)	1	Key study
6.1.9	ISO/DC 20666	<i>Brachionus plicatilis</i>	Sodium chlorate	EC10 (96h) = 21 mg/l (nominal) NOEC (96h) = 46 mg/l (nominal)	EC10 (96h) = 16.5 mg/l (nominal) NOEC (96h) = 36 mg/l (nominal)	Marine species	Study report (2010b)	1	Supporting study

11.6.1 Chronic toxicity to fish

One chronic fish study of sodium chlorate using an early life stage test on *Danio rerio* (see table 20) is available. The test was performed according to the OECD guideline 210 without deviations and in compliance with GLP. Chemical analyses showed that the test substance concentrations were stable during the test and close to nominal concentrations.

All embryos hatched at the highest concentration tested of 500 mg/l as well as in the control and post-hatch mortality was less than that of the control, the NOEC was considered to be at or greater than the highest concentration tested. No teratogenic malformations were noted for any larvae at any concentration.

Based on results from weight and length, the LOEC could not be calculated and the NOEC was determined as greater than or equal to the highest concentration tested, 500 mg/l.

Table 15. Chronic toxicity to fish

Method	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Test material	Remarks	Reference	Klimish score	Adequacy of study
36 days flow-through test according to OECD 210 Key study	NOEC ≥ 500 mg/l	NOEC ≥ 392 mg/l	Sodium chlorate	<i>Danio rerio</i>	Study report (2004a)	1	Key study

All studies can be found in IUCLID in section 6.1.2

The NOEC for fish is equal or greater than 500 mg/l. This result shows that sodium chlorate is also not harmful in the early life stages of fish.

11.6.2 Chronic toxicity to aquatic invertebrates

One chronic study with *Daphnia magna* was found (see table 21). The test was performed according to OECD guideline 211 and in compliance with GLP. Chemical analyses showed that the test substance concentrations were stable and close to nominal concentrations. The test was therefore considered valid without restrictions.

Reproductive output and length of adults at the end of the study were lower in the control than in any other concentration tested. Using Dunnett's and Bonferroni-t tests, the LOEC based on weight was found to be greater than 500 mg/l. Based on these results the NOEC for reproduction, weight and length is 500 mg/l.

Table 16. Chronic toxicity to invertebrates

Method	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Test material	Remarks	Reference	Klimish score	Adequacy of study
GLP, 21 days semi-static test according to OECD 211	NOEC ≥ 500 mg/l	NOEC ≥ 392 mg/l	Sodium chlorate	<i>Daphnia magna</i>	Study report (2004b)	1	Key study

All studies can be found in IUCLID in section 6.1.4

The NOEC for invertebrates is equal or greater than 500 mg/l. This result shows that sodium chlorate has also no effect on growth and reproduction of *Daphnia magna* up to 500 mg/l

11.6.3 Chronic toxicity to algae or other aquatic plants

Algae

Three studies, with fresh water species, one on *Selenastrum capricornutum* and two on *Scenedesmus subspicatus* were found (see table 22). One was valid without restrictions (Study report, 1991e) and two were valid with restrictions because not all details on the results were provided.

The NOEC value of the study with *Scenedesmus subspicatus* (Study report, 2004c) was 396.9 mg/l based on biomass and 1592.3 mg/l based on growth rate. The Fe₂O₃ present in the test substance interfered with the spectrophotometrical measurements and increased the extinction, this is not considered to have a significant impact on the results of this test. In the calculation of the biomass a correction is made for the higher extinctions measured at t=0. The higher values at t=0 do not have an influence on the slope of the growth curve, which is used for the calculation of the NOEC.

The same species was tested in Study report (1994a). The original value for the NOEC was 1569 mg chlorate/l.

Table 17. Toxicity to fresh water aquatic algae

Method	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Test material	Remarks	Reference	Klimish score	Adequacy of study
96 hours test according to EPA OPP 122-2	Cell growth: NOEC = 62.5 mg/l	Cell growth: NOEC = 49 mg/l	Sodium chlorate	<i>Selenastrum capricornutum</i>	Study report (1991e)	1	Supporting study
72 hours test according to OECD 201	Biomass: NOEC = 396.9 mg/l Growth rate: NOEC = 1592.3 mg/l	Biomass: NOEC = 311 mg/l Growth rate: NOEC = 1248.4 mg/l	Sodium chlorate	<i>Scenedesmus subspicatus</i>	Study report (2004c)	2	Supporting study
72 hours test according to OECD 201	NOEC = 2001 mg/l	NOEC = 1569 mg/l	Sodium chlorate	<i>Scenedesmus subspicatus</i>	Study report (1994a)	2	Supporting study

All studies can be found in IUCLID in section 6.1.5

The lowest NOEC for freshwater algae was found for *Selenastrum capricornutum* and was 62.5 mg/l.

For sodium chlorate, two tests with marine algae are available: one valid study (Study report, 2010a) and one valid with restriction (Study report, 1994b) (see table 23). Study report (2010a) is a standard test with *Skeletonema costatum* according to ISO 10253 guideline and in compliance with GLP. Chemical analyses were performed on the test concentrations. *S. costatum* was not sensitive to sodium chlorate, with a NOEC greater than or equal to 1000 mg/l. A test performed with *Phaeodactylum tricornerutum* found a NOEC of 64 mg sodium chlorate/l for biomass and a NOEC of 128 mg sodium chlorate/l for the growth rate.

Furthermore, Rosemarin *et al.*, 1986 and 1994, studied the effects of sodium chlorate in a mesocosm study. The test was considered to be invalid because the study was not performed according to standard methods and not in compliance with GLP. Though certain aspects were described in detail, there are parts which are not clear e.g. on method and materials. It is also not certain that replicates contain the same number and type of species; a) *Fucus vesiculosus* on original stone substrate with associated organisms were put in the pools. It is not known what these associated organisms were, whether a similar number was introduced into each pool and if they had an impact on the test result. b) Raw seawater was let into the pools. There are no details on the substances present in this water and it is not known if this water was treated before it entered the pools.

The EC₅₀ after 6 months of exposure and a NO₃ concentration of < 0.039 mg N/l, for apical growth of *F. vesiculosus* is ca. 80 µg ClO₃⁻/l and clear negative effects were seen at about 15-20 µg ClO₃⁻/l, which could be seen as 6 months-LOEC. As the study was not performed according to standard methods and the duration of the study was much longer than standard duration, the results are difficult to compare to standard test results.

Table 18. Toxicity of sodium chlorate to marine algae

Method	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Remarks	Reference	Klimish score	Adequacy of study
72 hours test according to ISO 10253 guideline	NOEC ≥ 1000 mg/l	NOEC ≥ 784 mg/l	<i>Skeletonema costatum</i>	Study report (2010a)	1	Supporting study
72 hours test according to own protocol based in ISO	NOE ₅ C = 64 mg/l NOE ₇ C = 128 mg/l	NOE ₅ C = 50 mg/l NOE ₇ C = 100 mg/l	<i>Phaeodactylum tricorutum</i>	Study report (1994b)	2	Supporting study
Own method – 6 months, mesocosm	EC50 ca. 102 µg ClO ₃ ⁻ /l	EC50 ca. 80 µg/l	<i>Fucus vesiculosus</i>	Rosemarin, <i>et al.</i> , 1986 and 1994	3	Invalid study

All studies can be found in IUCLID in section 6.1.5

Two tests (valid with restriction) were found for marine algae performed with potassium chlorate (see table 24).

Table 19. Toxicity of potassium chlorate to marine algae

Method	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Remarks	Reference	Klimish score	Adequacy of study
72 hours test according to own protocol with potassium chlorate	15 mg NO ₃ ⁻ /l: NOE ₅ C = 735 mg/l	15 mg NO ₃ ⁻ /l: NOE ₅ C = 500 mg/l	<i>Dunaliella tertiolecta</i>	Stauber J.L. (1998b)	2	Key study
72 hours test according to own protocol with potassium chlorate	15 mg NO ₃ ⁻ /l: NOE ₅ C = 147 mg/l	15 mg NO ₃ ⁻ /l: NOE ₅ C = 100 mg/l	<i>Nitzschia closterium</i>	Stauber J.L. (1998a)	2	Key study

All studies can be found in IUCLID in section 6.1.5

The tests were performed with potassium chlorate and originally the endpoints were expressed in mg chlorate/l. The original data for the NOEC based on growth rate was for *Dunaliella tertiolecta* > 500 mg chlorate/l and for *Nitzschia closterium* > 100 mg chlorate/l. The endpoints presented in Table 24 are recalculated to mg potassium chlorate/l for comparison with the studies on sodium chlorate. The tests were carried out at three different nitrate levels namely <0.005, 1 and 15 mg nitrate/l. At the lower nitrate concentrations cell growth in the controls was not according to the standard criteria and these results cannot be used therefore. Only valid results are given in the table above.

The concentration of nitrate in the test water influences the effect concentration of chlorate indicating that competitive inhibition occurs between nitrate and chlorate with excess nitrate inhibiting chlorate toxicity. This is supported by the acute studies on marine species using chlorate at several nitrate concentrations.

When comparing results from different marine algae toxicity studies, comparison should be made between studies performed under the same standard conditions (i.e. nitrate concentrations) as much as possible. The chlorate anion is not directly toxic; the mechanism of chlorate toxicity in plants and algae is indirect. The toxicity of chlorate is coupled to its reduction to chlorite and this reduction is linked to an active, functioning nitrate reductase system. The activity of this reductase system is related to the concentration of nitrate present, therefore it is important to compare studies with similar levels of nitrate.

All tested marine algae species showed similar sensitivity to chlorate under standard test conditions, with NOEC values for growth rate ≥ 100 mg ClO₃⁻/l (128 mg/l sodium chlorate or 147 mg/l potassium chlorate) in a test using standard nitrate concentrations.

Under standard test conditions with standard nitrate concentrations chlorate is not harmful to marine algae.

Aquatic plants

One valid study on sodium chlorate was found for freshwater aquatic plants (see table 25). The NOEC for fresh water aquatic plants is 10 mg/l.

Table 20. Toxicity to aquatic plants

Method	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Test material	Remarks	Reference	Klimish score	Adequacy of study
7 days test according to OECD 221 Key study (fresh water)	Biomass growth: NOEC = 10 mg/l Growth rate: NOEC = 10 mg/l Biomass dry weight: NOEC = 10 mg/l	Biomass growth: NOEC = 7.8 mg/l Growth rate: NOEC = 7.8 mg/l Biomass dry weight: NOEC = 7.8 mg/l	Sodium chlorate	<i>Lemna minor</i>	Study report (2003)	1	Key study

All studies can be found in IUCLID in section 6.1.6

Lemna minor is the most sensitive freshwater species tested to sodium chlorate. The lowest value for chronic toxicity to freshwater plants is 10 mg sodium chlorate /l based on *Lemna minor*.

11.6.4 Chronic toxicity to other aquatic organisms

One long-term study (valid without restrictions) is available for the rotifer *Brachionus plicatilis* (see table 26). This study was performed according to standard guideline ISO/DC 2066 in compliance with GLP. *B. plicatilis* turned out to be the most sensitive marine species with an EC₁₀ of 21 mg/l.

Table 21. Toxicity to other aquatic organisms

Method	Results Expressed as Na/KClO ₃	Results Expressed as ClO ₃ ⁻	Test material	Remarks	Reference	Klimish score	Adequacy of study
96h test according to ISO/DC 20666 Key study (marine)	EC ₁₀ = 21 mg/l NOEC = 46 mg/l	EC ₁₀ = 16.5 mg/l NOEC = 36.1 mg/l	Sodium chlorate	<i>Brachionus plicatilis</i>	Study report, (2010b)	1	Supporting study

All studies can be found in IUCLID in section 6.1.9

11.7 Comparison with the CLP criteria

11.7.1 Acute aquatic hazard

Acute aquatic toxicity data are available for all three trophic levels (fish, invertebrates, and algae).

The LC50_96h values obtained for fish (freshwater and marine water) for sodium chlorate were all greater than 1000 mg/l. EC50_48h obtained from two studies performed according to EPA OPP 72-2 and in compliance with GLP were greater than 1000 mg/L for *Daphnia magna*. Similarly, a toxicity test to the marine crustacean *Mysidopsis bahia* showed LC50_96h > 1000 mg/L.

Algae species (freshwater and marine) were demonstrated to be more sensitive to sodium chlorate than fish (freshwater and marine water). The lowest acute value for algae was found for *Selenastrum capricornutum* from a test performed according to EPA OPP 122-2. The sodium chlorate E_bC50_72h was 129 mg/L.

Concerning other freshwater aquatic organisms, there is one valid study on aquatic plants performed according to OECD 221 available. The sodium chlorate EC_{50_7d} (biomass growth) of 73.7 mg/L was obtained for *Lemna minor*.

Two marine studies on two different taxonomic groups are also available. In marine molluscs *Crassostrea virginica* the EC_{50_96h} value based on shell growth and LC_{50_96h} value for sodium chlorate were both greater than 1000 mg/L. In the rotifer *Brachionus plicatilis* the EC_{50_96h} for sodium chlorate was 596 mg/L.

These species cover a wide range of trophic levels and taxa and are considered as surrogate for all aquatic organisms. The acute aquatic toxicity based on the lowest of the available toxicity values is between 10 and 100 mg/L. It corresponds to EC_{50_7d} (biomass growth) of 73.7 mg/L obtained with *Lemna minor* (study performed with sodium chlorate). These values are all above 1 mg/L which is the classification cut-off for category Acute 1.

As a conclusion, sodium chlorate is not classified for the acute aquatic hazard.

11.7.2 Long-term aquatic hazard (including bioaccumulation potential and degradation)

Sodium chlorate has a harmonised classification as Aquatic Chronic 2 (H411) and is included in the Annex VI of the CLP regulation.

Sodium chlorate is highly soluble in water (696-736 g/L at 20°C for pH 4.49 to 8.70 at 20°C, see table 7). Sodium chlorate in aqueous solutions is known for its chemical stability under environmental conditions (Urbanski 1998). Based on the chemical structure it is expected that sodium chlorate is resistant to hydrolysis.

Sodium chlorate is considered to be rapidly biodegradable based on non-standard tests where oxygen was absent (van Ginkel et al., 1995). The maximum growth rates of chlorate reducing microorganisms range from 0.04 to 0.56 h⁻¹, growth rates of competent micro-organisms of 0.01 h⁻¹ or higher do result in a ready biodegradation of the test substance.

Although no measured data on bioaccumulation were identified, based on the environmental fate and behaviour of the substance (the complete dissociation in water and the high water solubility) no significant bioaccumulation is expected (BCF < 500).

Chronic toxicity data are available for all three trophic levels (fish, invertebrates, and algae).

In a chronic fish study performed according to OECD guideline 210 the NOEC_{36 d} was equal to or greater than 500 mg/l sodium chlorate. Similarly to the chronic toxicity fish test, no effect was observed at the highest concentration tested i.e. 500 mg/l sodium chlorate in a chronic invertebrate toxicity test according to OECD guideline 211 (*Daphnia magna* Reproduction Test). Therefore, NOEC_{21d} for reproduction, weight and length was equal to or greater than 500 mg/l sodium chlorate.

Algae species (freshwater and marine) were demonstrated to be more sensitive to sodium chlorate than fish and invertebrates. The lowest NOEC was found for *Selenastrum capricornutum* with a NOEC_{96h} of 62.5 mg/l.

Concerning other freshwater aquatic organisms, one valid study according to OECD 221 was available on *Lemna minor*. It was more sensitive compared to algae with a NOEC_{7d} (growth rate) of 10 mg/l.

Two other taxonomic groups of marine organisms were tested as well. Molluscs (*Crassostrea virginica*) were not sensitive to sodium chlorate at 1000 mg/l in a short-term test (96h). The rotifer *Brachionus plicatilis* was the most sensitive marine species with a NOEC_{96h} of 46 mg/l based on reproduction.

These species cover a wide range of trophic levels and taxa and are considered as surrogate for all aquatic organisms. The chronic aquatic toxicity based on the lowest of all the available toxicity values is above 1 mg/l and correspond to NOEC_{7d} (growth rate) of 10 mg/l sodium chlorate obtained with *Lemna minor*.

For non-rapidly degradable substances for which there are adequate chronic toxicity data available, the classification cut-off for category Chronic 2 is 1 mg/l. For rapidly degradable substances for which there are adequate chronic toxicity data available, the classification cut-off for category Chronic 3 is also 1 mg/l.

Adequate chronic toxicity data are available for all three trophic levels and the lowest chronic value is above 1 mg/l. Sodium chlorate is considered rapidly degradable as described in section 11.3.1, but even if sodium chlorate would be considered as non rapidly degradable in the aquatic environment, it does not lead to any classification for the chronic aquatic hazard.

As a conclusion, sodium chlorate does not need to be classified for the chronic aquatic hazard.

11.8 CONCLUSION ON CLASSIFICATION AND LABELLING FOR ENVIRONMENTAL HAZARDS

The observed acute aquatic toxicity for sodium chlorate is above the cut-off criterion of 1 mg/l. **Sodium chlorate does therefore not need to be classified for the acute aquatic hazard.**

Adequate chronic toxicity data are available for all three trophic levels. The chronic aquatic toxicity for sodium chlorate is above the cut-off criterion of 1 mg/l. Even if a worst-case considering that sodium chlorate is not rapidly degradable in the aquatic environment is applied, **sodium chlorate does therefore not need to be classified for the chronic aquatic hazard.**

As a conclusion, no classification for environmental hazards is warranted for sodium chlorate according to the criteria in Annex I of the CLP Regulation (Commission Regulation (EU) No 286/2011).

12 EVALUATION OF ADDITIONAL HAZARDS

This part was not evaluated in this dossier.

13 ADDITIONAL LABELLING

14 REFERENCES

- AFFSA (2011) Expositions au Chlorate de sodium enregistrées dans la BNCI Analyse des données des Centres antipoison et de toxicovigilance (juillet 1999 – novembre 2008)
- Bao, H.M. and Gu, B.H. (2004) Natural perchlorate has a unique oxygen isotope signature *Environ Sci Technol* 38 5073-5077
- Ben-Dyke, R., Sanderson, D. M., Noakes, N. (1970). Acute toxicity data for pesticides. *World rev. pest contr.* 9(3), 119-127.
- Bloxham, C.A., Wright, N., Hoult, J.G., 1979, Self-poisoning by sodium chlorate--some unusual features. *Clin Toxicol.* 15(2):185-188.
- Bohlke, J.K., Sturchio, N.C., Gu, B., Horita, J., Brown, G.M., Jackson, W.A., Batista, J. and Hatzinger, P.B. (2005) Perchlorate isotope forensics et al (2005) *Anal Chem* 77 7838-7842)
- Bryan and G. A. Rohlich (1954). Biological reduction of sodium chlorate as applied to measurement of BOD. *Sewage and Industrial wastes* Vol 26; no 11; 1315 - 1324.
- Casey, P. and Vale, J.A., 1994, Deaths from pesticide poisoning in England and Wales: 1945-1989. *Hum Exp Toxicol.* 13(2):95-101
- Clinical Toxicology (1969) Poisoning by potassium and sodium chlorate, *Clinical toxicology*, 1969, p:101-109
- Cunningham, N.E. (1982). Chlorate poisoning – two cases diagnosed at autopsy. *Med. Sci. Law* **22**, 281-282.
- Dasgupta, P.K., Martinelango, P.K., Jackson, W.A., Anderson, T.A., Tian, K., Tock, R.W. and Rajagopalan, S. (2005) The origin of naturally occurring perchlorate; the role of atmospheric processes *Environ Sci Technol* 39 1569-1575
- De Ryckel, B. (2006). Physical and chemical properties and storage stability tests for Sodium Chlorate. Testing laboratory: CRA-W, Pesticides Research Department, Gembloux, Belgium. Report no.: 20793. Owner company: Cefic Sodium Chlorate Herbice Registration Group. Report date: 2006-08-23.
- EFSA (2015) Risks for public health related to the presence of chlorate in food. *EFSA Journal* 2015;13(6):4135. Link: <https://www.efsa.europa.eu/en/efsajournal/pub/4135>
- Eysseric, H., Vincent, F., Peoc'h, M., Marka, C., Aitken, Y., Barret, L., 2000, A fatal case of chlorate poisoning: confirmation by ion chromatography of body fluids. *J Forensic Sci.* 45(2):474-477.
- Fukumoto K, Fukumoto H, 1970, A fatal case of potassium chlorate poisoning [Japanese], *Nippon Hoigaku Zasshi.* 24(3):242-246
- General Register Office, London, 1969, Poisoning cases in 1967. *The Pharmaceutical Journal*, 74-77
- Granier, P., Danel, V., Barnoud, D., Lavagne, P., Faure, J., 1985, Intoxication par le chlorate de soude. *Presse Med.* 11;14(19):1099

Groult (2004). Water solubility. Testing laboratory: CIT, Evreux Cedex France. Report no.: 28186 PSE. Owner company: CEFIC Sodium Chlorate Herbicide Registration Group. Report date: 2004-10-15.

Hartley, D. and H. Kidd (eds.). The Agrochemicals Handbook. 2nd ed. Lechworth, Herts, England: The Royal Society of Chemistry, 1987., p. A294/Aug 87

Hayes, W. J. and E. R. Laws. 1991. Handbook of Pesticide Toxicology. Volume 2. Classes of Pesticides. Academic Press, Inc. NY.

Helliwell, M. and Nunn, J., 1979, Mortality in sodium chlorate poisoning. Br Med J. 28;1(6171):1119.

HSDB, 2005, Sodium chlorate. Hazardous Substances Data Bank. Accessed online at: <http://toxnet.nlm.nih.gov>

Jackson RC, Elder WJ, McDonnell H, 1961, Sodium-chlorate poisoning complicated by acute renal failure. Lancet. 23; 2:1381-1383.

Jansen, H. and Zeldenrust, J., 1972, Homicidal chronic sodium chlorate poisoning. Forensic Sci. 1(1):103-105.

Klendshoj NC, Burke WJ, Anthone R, Anthone S., 1962, Chlorate poisoning. JAMA.180:1133-1134

Lewis, R.J., Sr. (1996). Sax's Dangerous Properties of Industrial Materials, 9th ed., pp. 2953-2954. Van Nostrand Reinhold, New York.

L'Haridon, J. (2003). Activated sludge, respiration inhibition test. Testing laboratory: CIT, Evreux Cedex France. Report no.: 22932 EAS. Owner company: CEFIC Sodium Chlorate Herbicide Registration Group. Report date: 2003-07-18.

L'Haridon, J. (2003). Ready biodegradability CO₂ evolution test. Testing laboratory: CIT, Evreux Cedex France. Report no.: 22933 ECS. Owner company: Cefic Sodium Chlorate Herbicide Registration Group. Report date: 2003-07-04.

Murphy, D.M. and Thomson, D.S. (2000) Halogen ions and NO + in the mass spectra of aerosols in the upper troposphere and lower stratosphere Geophys Res Lett 27 3217-3220

NTP TR 517, 2005. Sodium Chlorate. [Link](#).

O'Grady J, Jarecsni E, 1971, Sodium chlorate poisoning. Br J Clin Pract.25(1):38-39.

Oliver JS, Smith H, Watson AA, 1972, Sodium chlorate poisoning. J Forensic Sci Soc.12(3):445-448.

Painter, H A, King, E F, "A Mathematical Model of Biodegradability Screening Tests as an Aid to Interpretation of Observed Results," Regulatory Toxicology and Pharmacology, vol. 3, 144-151 (1983).

Proudfoot, A.T., Stewart, M.S., Levitt, T., and Widdop, B. (1979). Acute poisoning with chemicals used in agriculture and horticulture. Prescr. J. 19, 183-189.

Rosemarin, A., Lehtinen, K.J., Notini, M. and Mattsson, J. (1994). Effects of pulp mill chlorate on Baltic sea algae. *Environmental Pollution* 85: 3-13.

Rosemarin, A., M., Mattsson, J., Lehtinen, K.J., Notini and Nylen, E.(1986). Effects of pulp mill chlorate in *Fucus vesiculosus* – A summary of projects. *Ophelia*, Suppl. 4: 219-224.

Sheahan B. J., Pugh, D. M., Winstanley, E. W. (1971). Experimental sodium chlorate poisoning in dogs. *Res Vet Sci.*; 12(4):387-389.

Simonaitis, R. and Heicklen, J. (1975) Perchloric acid; possible sink for stratospheric chlorine. *Planet Space Sci* 23 1567-569

Smith et al (2012) INVITED REVIEW: Efficacy, metabolism, and toxic responses to chlorate salts in food and laboratory animals. *J. Anim. Sci.* 2012.90:4098–4117

Stauber J.L. (1998). Toxicity of chlorate to marine microalgae. *Aquatic toxicology* 41 pp. 213-227.

Stavrou, A., Butcher, R., and Sakula, A. (1978). Accidental self-poisoning by sodium chlorate weed-killer. *Practitioner* 221, 397-399.

Steffen C, Seitz R, 1981, Severe chlorate poisoning: report of a case. *Arch Toxicol.* 48(4):281-288.

Steffen, C., Wetzel, E., 1993, Chlorate poisoning: mechanism of toxicity. *Toxicology*; 84(1-3):217-231

Study report (1991a). Acute flow-through toxicity of sodium chlorate to the rainbow trout, *Oncorhynchus mykiss*. Testing laboratory: EnviroSystems Division of Resource Analysts, Hampton, New Hampshire. Report no.: 90143-AW. Owner company: Sodium Chlorate Reregistration Task Force. Report date: 1991-05-06.

Study report (1981). Acute oral toxicity study in rats-Sodium chlorate. Testing laboratory: Litton Bionetics Inc., Kensington, Maryland, US. Report no.: 22097. Owner company: Kerr-McGee. Report date: 1981-05-01.

Study report (1991c). Acute flow-through toxicity of sodium chlorate to the sheepshead minnow, *Cyprinodon variegatus*. Testing laboratory: EnviroSystems Division of Resource Analysts, Hampton, New Hampshire. Report no.: 90115-DE. Owner company: Sodium Chlorate Reregistration Task Force. Report date: 1991-05-07.

Study report (1991b). Acute flow-through toxicity of sodium chlorate to the bluegill sunfish *Lepomis macrochirus*. Testing laboratory: EnviroSystems Division of Resource Analysts, Hampton, New Hampshire. Report no.: 90142-AW. Owner company: Sodium Chlorate Reregistration Task Force. Report date: 1991-05-06.

Study report (1991d). Acute flow-through toxicity of sodium chlorate to the daphnid, *Daphnia magna*. Testing laboratory: EnviroSystems Division of Resource Analysts, Hampton, New Hampshire. Report no.: 90144-AW. Owner company: Sodium Chlorate Reregistration Task Force. Report date: 1991-05-06.

Study report (1991e). Static acute toxicity of sodium chlorate to the freshwater algae, *Selenastrum capricornutum*. Testing laboratory: EnviroSystems Division of Resource Analysts, Hampton, New

Hampshire. Report no.: 90161-AW. Owner company: Sodium Chlorate Reregistration Task Force. Report date: 1991-05-06.

Study report (1991f). Acute Flow-through Toxicity of Sodium Chlorate To The Mysid, *Mysidopsis bahia*. Testing laboratory: EnviroSystems Division Resource Analysts, Incorporated. Report no.: 90117-DE. Owner company: Sodium Chlorate Reregistration Task Force. Report date: 1991-05-06.

Study report (1991g). Acute Flow-through Mollusc Shell Deposition Test with Sodium Chlorate. Testing laboratory: EnviroSystems Division Resource Analysts, Incorporated. Report no.: 90116-DE. Owner company: Sodium Chlorate Registration Task Force. Report date: 1991-05-06.

Study report (1991h). Acute toxicity of sodium chlorate to *Brachydanio rerio*. Report no.: CRL F91163. Owner company: CEFIC Sodium Chlorate Herbicide Registration Group.

Study report (1991i). EPA acute oral toxicity limit test. Testing laboratory: Product safety labs, east Brunswick, New Jersey, US. Report no.: T-488. Owner company: Sodium Chlorate Reregistration Task Force. Report date: 1991-01-24.

Study report (1993). Acute toxicity of sodium chlorate to *Pimephales promelas*. Testing laboratory: Akzo Nobel Research, Arnhem, The Netherlands. Report no.: CRL F93115. Owner company: CEFIC Sodium Chlorate Herbicide Registration Group. Report date: 1993-06-18.

Study report (1994a). Sodium chlorate: toxicity to the green alga *Scenedesmus subspicatus*. Testing laboratory: Brixham Environmental Laboratory, UK. Report no.: BLS1742/B. Owner company: Eka Chemicals AB. Report date: 1994-11-13.

Study report (1994b). Sodium chlorate: toxicity to the marine alga *Phaedactylum tricornutum*. Testing laboratory: Brixham environmental laboratory, UK. Report no.: BLS1741/B. Owner company: Eka Chemicals AB. Report date: 1994-11-13.

Study report (1995). Acute toxicity of Sodium chlorate to the water flea, *Daphnia magna*, under static test conditions. Testing laboratory: Environmental Science & Engineering, Inc., (ESE), Gainesville, Florida, US. Report no.: 3195436-0100-3100. Owner company: Sodium Chlorate Reregistration Task Force. Report date: 1995-09-14.

Study report (2003). Sodium chlorate aquatic plant toxicity test, *Lemna minor*, static, 7 d. Testing laboratory: Dr. U. Noack-Laboratorien, Germany. Report no.: TLA93762. Owner company: CEFIC Sodium Chlorate Herbicide Registration Group.

Study report (2004a). Chronic toxicity of sodium chlorate to *Danio rerio* in an early-life stage toxicity test under flow-through conditions. Testing laboratory: Akzo Nobel Chemicals Research, Arnhem, The Netherlands. Report no.: CER F04023. Owner company: CEFIC Sodium Chlorate Herbicide Registration Group. Report date: 2004-08-30.

Study report (2004b). Chronic toxicity of sodium chlorate to *Daphnia magna* in a 21 day reproduction test under semi-static conditions. Testing laboratory: Akzo Nobel Chemicals Research, Arnhem, The Netherlands. Report no.: CER F03043 T 02006 ODC. Owner company: CEFIC Sodium Chlorate Herbicide Registration Group. Report date: 2004-01-27.

Study report (2004c). Effects of Sodium chlorate on the growth of the freshwater green alga, *Scenedesmus subspicatus*. Testing laboratory: Akzo Nobel Chemicals Research, Arnhem, The

Netherlands. Report no.: CER F04022 T02006 AL. Owner company: CEFIC Sodium Chlorate Herbicide Registration Group. Report date: 2004-07-05.

Study report (2010a). Sodium Chlorate Growth inhibition of the marine alga *Skeletonema costatum*. Testing laboratory: NIVA Norwegian Institute for Water Research, Oslo, Norway. Report no.: G089/2. Owner company: EKA Chemicals AB, Sweden. Report date: 2010-06-30.

Study report (2010b). Sodium Chlorate Effect on reproduction to the marine rotatoria *Brachionus plicatilis*. Testing laboratory: NIVA Norwegian Institute for for Water Research, Oslo, Norway. Report no.: G089/1. Owner company: EKA Chemicals AB, Sweden. Report date: 2010-06-30.

Timperman J, Maes R, 1966, Suicidal poisoning by sodium chlorate A report of three cases. J Forensic Med. 13(4):123-129

Toussaint, *et al.* (2001). Acute toxicity of four drinking water disinfection by-products to Japanese medaka fish. Bull Environ Contam Toxicol. 66(2):255-262.

Vakili M, 1977, Chlorate poisoning in childhood--a case report. J Trop Pediatr Environ Child Health. 23(3):119.

Van Ginkel et al (1995). Reduction of chlorate with various energy substrates and inocula under anaerobic conditions. Chemosphere 31, 4057 - 4066.

Vos, A. (2009), Determination of water solubility of potassium Chlorate, Akzo Nobel Technology & Engineering; 2.389.042

Weissenfeld, M. (2004). DISSOCIATION CONSTANT OF Sodium chlorate IN WATER. Test Facility: RCC Ltd, Itingen, Switzerland. study no.: 854989. Owner company: Cefic Sodium Chlorate Herbicide Registration Group. Report date: September 17, 2004.

Yoshida, Y., Hirose, Y., Konda, S., Kitada, H., Shinoda, A., 1977, A cytological study of Heinz body-hemolytic anemia. Report of a case of sodium chlorate poisoning complicated by methaemoglobinemia and acute renal failure. Nippon Ketsueki Gakkai Zasshi. 40(1):147-151