Aquatrade Water Treatment Chemicals (Pty) Ltd

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1 Identification

GHS Product Identifier

NITRIC ACID

Other means of identification

CAS:	7697-37-2
EC:	231-714-2
RTECS:	QU5775000
ICSC:	0183
NSC:	177677
UN:	2031
Chemical Family:	Mineral Acids
Synonyms:	NITRIC ACID Azotic acid Hydrogen nitrate Salpetersaeure Aqua fortis Acidum nitricum Nital Acide nitrique Nitrous fumes Nitryl hydroxide Engraver's acid
Proper Shipping Name:	NITRIC ACID, other than red fuming, with <70% nitric acid
Chemical Formula:	HNO_3

Recommended use of the chemical and restriction on use

Manufacture of inorganic and organic nitrates and nitro compounds for fertilizers, dye intermediates, explosives. Pharmaceutic aid (acidifier). Metallurgy, photo-engraving, etching steel, ore flotation, urethanes, rubber chemicals, reprocessing spent nuclear fuel. Nitric acid is a very common mineral acid. It is used to dissolve noble metals, for etching and cleaning metals, and to make nitrates and nitrocompounds, including organic derivatives found in commercial or military explosives. Medication.

Industry Uses

1. Agricultural chemicals (non-pesticidal)
2. Agrium use in ammmonia nitrate production, final AN use for preparing mining explosives
3. Explosive Materials
4. Industrial/mining explosives.
5. Ion exchange agents
6. Laboratory chemicals
7. Nitric acid sold to distributers for formulation, re-packaging and sale of water treatment chemicals.
8. Nitric acid used in the production of catalysts for distribution to industry for miscellaneous industrial process use.
9. Oxidizing/reducing agents
10. Plasticizers
11. Plating agents and surface treating agents
12. Repackaging/chemical distribution
13. Solvents (for cleaning and degreasing)

14. Used for chemical etching of inorganic films in integrated circuit manufacturing.

Supplier's details

AQUATRADE WATER TREATMENT CHEMICALS (PTY) LTD

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Emergency phone number

E le Sar: +27 82 921 0643 (Available Mon - Fri, GMT 5:00 to 20:00)
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2 Hazard(s) identification

Classification of the substance or mixture

Classification according to Regulation (EC) No 1272/2008

Corrosive to Metals (Category 1), H290
Acute Toxicity - Inhalation (Category 3), H331
Skin Corrosion/Irritation (Category 1A), H314

For the full text of the H-Statements mentioned in this Section, see Section 16.

GHS label elements

Danger


May be corrosive to metals

Causes severe skin burns and eye damage

Toxic if inhaled

Keep only in original container.

Do not breathe dust/fume/gas/mist/vapours/spray.

Wash thoroughly after handling.

Use only outdoors or in a well-ventilated area.

Wear protective gloves/protective clothing/eye protection/face protection.

IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.

IF ON SKIN (or hair): Remove/Take off Immediately all contaminated clothing. Rinse SKIN with water/shower.

IF INHALED: Remove victim to fresh air and Keep at rest in a position comfortable for breathing.

IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.

Immediately call a POISON CENTER or doctor/physician.

Specific treatment (see P330+P351+P353 on this label).

Wash contaminated clothing before reuse.

Absorb spillage to prevent material damage.

Store in a well-ventilated place. Keep container tightly closed.

Store locked up.

Store in corrosive resistant container with a resistant inner liner.

Dispose of contents and container in accordance with local, regional, national, international regulations.

3 Composition/information on ingredients

Description	CAS Number	EINECS Number	%	Note
Nitric acid	7697-37-2	231-714-2	0 - 60	

4 First-aid measures

Description of necessary first-aid measures

Call 112 or 10177 or your local emergency help number immediately, for emergency assistance. Call the Poison Control Center at +27 21 931 6129 – Tygerberg or +27 21 658 5308 – Red Cross, Email: poisonsinformation@uct.ac.za, Website: https://www.afritox.co.za for further instructions. Provide them with information such as the compound taken, quantity and time of ingestion, age, weight and general health status of affected individual. Carefully remove the individual from the exposure area.

General
Speed is essential.
Give first aid and obtain medical attention immediately.
First aiders should be protected adequately (see section "Handling and storage").
Remove affected person from further exposure.
Ensure that eyewash facility and safety showers are provided close to the work place.

Inhalation
Move the injured person to fresh air at once.
Keep the patient warm and at rest in a half upright position.
Apply artificial respiration, if breathing has stopped or shows sign of failing. Mouth to mouth resuscitation may be dangerous.
Administer oxygen if competent person is available.

Ingestion
DO NOT induce vomiting.
If the person is conscious, wash out mouth with water and give water to drink.
Skin contact : Drench with water, remove contaminated clothing and wash or shower the affected skin with plenty of water for at least 15 minutes.

Eye contact
Inmediately irrigate the eyes with eyewash solution or clean water for at least 15 minutes.
Hold eyelids open during flushing. Do not allow victim to rub eyes.

Most important symptoms/effects, acute and delayed

Effects of Short Term Exposure
The substance is corrosive to the eyes, skin and respiratory tract. Corrosive on ingestion. Inhalation may cause asthma-like reactions (RADS). Exposure could cause asphyxiation due to swelling in the throat. Inhalation of high concentrations may cause pneumonitis and lung oedema.

Effects of Long Term Exposure
Repeated or prolonged inhalation may cause effects on the teeth. This may result in tooth erosion. The substance may have effects on the upper respiratory tract and lungs. This may result in chronic inflammation of the respiratory tract and reduced lung function . Mists of this strong inorganic acid are carcinogenic to humans.

Indication of immediate medical attention and special treatment needed, if necessary

Immediate first aid:
Ensure that adequate decontamination has been carried out. If patient is not breathing, start artificial respiration, preferably with a demand-valve resuscitator, bag-valve-mask device, or pocket mask, as trained. Perform CPR as necessary. Immediately flush contaminated eyes with gently flowing water. Do not induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain an open airway and prevent aspiration. Keep patient quiet and maintain normal body temperature. Obtain medical attention.

Basic treatment:
Establish a patent airway (oropharyngeal or nasopharyngeal airway, if needed). Suction if necessary. Watch for signs of

respiratory insufficiency and assist respirations if needed. Administer oxygen by nonrebreather mask at 10 to 15 L/min. Monitor for pulmonary edema and treat if necessary. Monitor for shock and treat if necessary. For eye contamination, flush eyes immediately with water. Irrigate each eye continuously with 0.9% saline (NS) during transport. Do not use emetics. Activated charcoal is not effective. For ingestion, rinse mouth and administer 5 ml/kg up to 200 ml of water for dilution if the patient can swallow, has a strong gag reflex, and does not drool. Do not attempt to neutralize because of exothermic reaction. Cover skin burns with dry, sterile dressings after decontamination.

Advanced treatment:
Consider orotracheal or nasotracheal intubation for airway control in the patient who is unconscious, has severe pulmonary edema, or is in severe respiratory distress. Early intubation, at the first sign of upper airway obstruction, may be necessary. Positive-pressure ventilation techniques with a bag valve mask device may be beneficial. Consider drug therapy for pulmonary edema. Consider administering a beta agonist such as albuterol for severe bronchospasm. Monitor cardiac rhythm and treat arrhythmias as necessary. Start IV administration of D5W /SRP: "To keep open", minimal flow rate/. Use 0.9% saline (NS) or lactated Ringer's(LR) if signs of hypovolemia are present. For hypotension with signs of hypovolemia, administer fluid cautiously. Consider vasopressors if patient is hypotensive with a normal fluid volume. Watch for signs of fluid overload. Use proparacaine hydrochloride to assist eye irrigation.

5 Fire-fighting measures

Suitable extinguishing media

Excerpt from ERG Guide 157 [Substances - TOXIC and/or CORROSIVE (Non-Combustible / Water-Sensitive)]
EVACUATION
Spill

- See Table 1 - Initial Isolation and Protective Action Distances for highlighted materials. For non-highlighted materials, increase, in the downwind direction, as necessary, the isolation distance shown under "PUBLIC SAFETY".

Fire

- If tank, rail car or tank truck is involved in a fire, ISOLATE for 800 meters (1/2 mile) in all directions; also, consider initial evacuation for 800 meters (1/2 mile) in all directions.
- [FLAG] In Canada, an Emergency Response Assistance Plan (ERAP) may be required for this product. Please consult the shipping document and/or the ERAP Program Section (page 391).

FIRE

- Note: Some foams will react with the material and release corrosive/toxic gases.

Small Fire

- CO2 (except for Cyanides), dry chemical, dry sand, alcohol-resistant foam.

Large Fire

- Water spray, fog or alcohol-resistant foam.
- Move containers from fire area if you can do it without risk.
- Use water spray or fog; do not use straight streams.
- Dike fire-control water for later disposal; **DO NOT** scatter the material.

Fire involving Tanks or Car/Trailer Loads

- Fight fire from maximum distance or use unmanned hose holders or monitor nozzles.
- **DO NOT** get water inside containers.
- Cool containers with flooding quantities of water until well after fire is out.
- Withdraw immediately in case of rising sound from venting safety devices or discoloration of tank.
- ALWAYS stay away from tanks engulfed in fire.

Suitable extinguishing media
Nitric acid is not combustible but if involved in a fire use the best means available to extinguish the fire (e.g water, or CO2).

Extinguishing media not to be used
DO NOT use chemical extinguishers or foams or attempt to smother the fire with steam or sand.

Specific hazards arising from the chemical

Specific hazards
Nitric acid is not combustible, but has oxidizing properties and therefore may react with many combustible materials causing fires and releasing toxic fumes (nitrogen oxides). May explode on contact with a powerful reducing agent. Reacts with most common metals to liberate hydrogen which can form explosive mixtures with air.

Hazardous thermal decomposition and combustion products
Nitrogen oxides.

Special fire fighting procedures
Use water sprays to cool fire-exposed containers and structures, to disperse vapours and to protect personnel. **Avoid** disposal of contaminated fire fighting water to the environment.

Special protective actions for fire-fighters

Wear self-contained breathing apparatus and full acid- resistant protective clothing.

6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

Excerpt from ERG Guide 157 [Substances - TOXIC and/or CORROSIVE (Non-Combustible / Water-Sensitive)]
EVACUATION
Spill

- See Table 1 - Initial Isolation and Protective Action Distances for highlighted materials. For non-highlighted materials, increase, in the downwind direction, as necessary, the isolation distance shown under "PUBLIC SAFETY".

Fire

- If tank, rail car or tank truck is involved in a fire, ISOLATE for 800 meters (1/2 mile) in all directions; also, consider initial evacuation for 800 meters (1/2 mile) in all directions.
- [FLAG] In Canada, an Emergency Response Assistance Plan (ERAP) may be required for this product. Please consult the shipping document and/or the ERAP Program Section (page 391).

SPILL OR LEAK

- ELIMINATE all ignition sources (no smoking, flares, sparks or flames in immediate area).
- All equipment used when handling the product must be grounded.
- **DO NOT** touch damaged containers or spilled material unless wearing appropriate protective clothing.
- Stop leak if you can do it without risk.
- A vapor-suppressing foam may be used to reduce vapors.
- **DO NOT** GET WATER INSIDE CONTAINERS.
- Use water spray to reduce vapors or divert vapor cloud drift. **Avoid** allowing water runoff to contact spilled material.
- Prevent entry into waterways, sewers, basements or confined areas.

Small Spill

- Cover with DRY earth, DRY sand or other non-combustible material followed with plastic sheet to minimize spreading or contact with rain.
- Use clean, non-sparking tools to collect material and place it into loosely covered plastic containers for later disposal.

Personal precautions
Those dealing with major releases should wear full protective clothing including respiratory protection. **Avoid** skin and eye contact and inhalation vapours. Evacuate unnecessary personnel.

Environmental precautions

General
Take care to avoid the contamination of watercourses. Inform appropriate authority in case of accidental contamination of watercourses or drains. Dilute with water and neutralise the acid with, for example soda or sodium carbonate, before discharging contaminated material into treatment plants or water courses.

Air spill
Apply water spray or mist to knock down vapors. Vapor knockdown water is corrosive or toxic and should be diked for containment.

Land spill
Dig a pit, pond, lagoon, or holding area to contain liquid or solid material. If time permits, pits, ponds, lagoons, soak holes, or holding areas should be sealed with an impermeable flexible membrane liner. Dike surface flow using soil, sand bags, foamed polyurethane, or foamed concrete. Absorb bulk liquid with fly ash or cement powder. Neutralize with agricultural lime (CaO), crushed limestone, or sodium bicarbonate.

Water spill
Neutralize with agricultural lime (slaked lime), crushed limestone, or sodium bicarbonate.

Methods and materials for containment and cleaning up

For small spillage dilute with water and neutralise cautiously with soda ash and/or lime and recover for disposal. Contain/absorb large spillage with sand or earth as necessary. **DO NOT** use organic compounds, sawdust etc. Use a tool to scoop up solid or absorbed material and place into an appropriately labelled wate container. Pump large amounts of the spilled liquid into containers suitably labelled for disposal.

Remarks
Isolate source of leak as quickly as possible. Ventilate area of spill or leak to disperse vapors if necessary.

7 Handling and storage

Precautions for safe handling

Avoid skin and eye contact and inhalation of vapours. Provide adequate ventilation. Wear eye and hand protection when handling small quantities. Wear full protective equipment where there is a risk of leaks or splashes. When diluting, add acid to water and not water to acid.

Conditions for safe storage, including any incompatibilities

DO NOT permit smoking in the storage area. Keep away from incompatible substances. (See Section Stability and reactivity). Protect containers from corrosion and physical damage. Follow appropriate Industry or National codes for bulk and container storage.

Packaging materials
Containers should of stainless steel and preferably of low carbon, content such as 304L or plastic (e.g. PVC).

Safe Storage
Separated from combustible substances, reducing agents, bases, organic chemicals and food and feedstuffs. Cool. Dry. Store only in original container. Store in cool, well ventilated area; away from heat, ignition source and direct sunlight.

Storage Conditions
As a rule, nitric acid is stored in stainless steel tanks and transported in stainless steel containers.

Store in a cool, dry, well-ventilated location. Separate from alkalies, metals, organics, and other oxidizing materials.

Storage areas should be separated from other premises, well-ventilated, sheltered from sunlight and sources of heat, should have a cement floor, contain no substances with which acid might react. Large stocks must be surrounded by curbs or sills. In the event of leakage & provisions for neutralization should be made. A fire hydrant should be outside storage premises. Electrical equipment should be of the water-proof type and resistant to acid attack. Safety lighting is desirable.

SANS 10263-0 Warehousing
8.4.3.2 Where flammable or **corrosive** substances are stored, the floor shall slope away from the storage area (primary collection area) to a secondary catch basin or sump of capacity at least 10 % of the total available storage volume of the fire section concerned. The secondary catch basin shall be within the fire section, and shall be such that it can be well ventilated. Care shall be taken in the design of such areas to prevent contamination of the soil or ground water.

9.7.2 Every type of storage area inside a warehouse shall be clearly demarcated, for example separate storage areas for poisons, flammables and **corrosives** shall display the relevant hazard class diamond (see table 1). The dimensions of the hazard class diamonds shall be at least 250 mm x 250 mm.

12.8.5 Storage of flammable liquids of class 3, toxic substances of division 6.1 and **corrosives** of class 8

Nitro-methane class 3, UN No. 1261, shall be separated from substances of class 6.1, and cyanides of division 6.1 shall be separated from acids of class 8. Concentrated acids and bases shall be segregated by at least 1 m. Packaged flammable liquids of class 3, toxic substances of division 6.1 and **corrosives** of class 8 that are of category 3 can be stored in the same area, provided that

a) they are kept above floor level, and

b) liquid dangerous goods of one class are not stored above dangerous goods of another class.

12.8.8.4 Corrosives (see class 8 in SANS 10228) that leak or spill from their packaging can cause serious damage to other packages, with potentially hazardous consequences.

Corrosives shall be segregated from toxic substances, infectious substances, aerosols, flammables, oxidizing substances and organic peroxides.

The provisions of above apply to the storage of the following quantities of dangerous goods.

Corrosives (acids and bases)Class 8	
Category 1	> 50 kg
Category 2	> 200 kg
Category 3	> 1 000 kg

8 Exposure controls/personal protection

Control parameters

Exposure limit values
The following values apply to nitric acid (HNO3) vapours
EU OEL 2000/39/EC
Recommended values;
STEL 2.6 mg/m3 (1ppm) (2006/15/EC)
TWA 2 ppm (5 mg/m3) ST 4 ppm (10 mg/m3)
PEL TWA 2 ppm (5 mg/m3) See Appendix G
PEL-TWA 2 ppm (5 mg/m³)
REL-TWA 2 ppm (5 mg/m³)
REL-STEL 4 ppm (10 mg/m³)

Immediately Dangerous to Life or Health
IDLH 25 ppm (NIOSH, 2016)

Threshold Limit Values
8 hr Time Weighted Avg (TWA): 2 ppm
15 min Short Term Exposure Limit (STEL): 4 ppm.

Emergency Response Planning Guidlines (ERPGs)	
ERPG-1	1 ppm (Odor should be detectable near ERPG-1)
ERPG-1	6 ppm
ERPG-1	78 ppm

- The ERPG-1: The maximum airborne concentration below which it is believed nearly all individuals could be exposed for up to 1 hour without experiencing more than mild, transient adverse health effects or without perceiving a clearly defined objectionable odor.
- The ERPG-2: The maximum airborne concentration below which it is believed nearly all individuals could be exposed for up to 1 hour without experiencing or developing irreversible or other serious health effects or symptoms that could impair an individual's ability to take protective action.
- The ERPG-3: The maximum airborne concentration below which it is believed nearly all individuals could be exposed for up to 1 hour without experiencing or developing life-threatening health effects.

Appropriate engineering controls

Engineering measures
Local exhaust ventilation where appropriate. Provide safety showers and eye washing facility at any location where skin or eye contact can occur.

Hygienic measures
When handling the product do not eat, drink or smoke. Wash hands after handling and before eating, smoking and using the lavatory and at the end of the working period. Personal protection : there 2 cases depending nitric acid concentration

Individual protection measures

The selection of PPE is dependent on a detailed risk assessment. The risk assessment should consider the work situation, the physical form of the chemical, the handling methods, and environmental factors. Recommendations below is advisory only and must be evaluated by an industrial hygienist and safety officer familiar with the specific situation of anticipated use by our customers. It should not be construed as offering an approval for any specific use scenario.

Eye/face protection:

Use chemical safety goggles e.g. EN 166 or full face mask EN 402. Contact lenses should not be worn as they may contribute to severe eye injury.

Skin protection:

Handle with gloves. Gloves must be inspected prior to use. Use proper glove removal technique (without touching glove's outer surface) to avoid skin contact with this product. Dispose of contaminated gloves after use in accordance with applicable laws and good laboratory practices. Wash and dry hands. The selected protective gloves have to satisfy the specifications of EU Directive 89/686/EEC and the standard EN 374 derived from it.

Breakthrough time. > 8 hrs
Material: butyl rubber, PVC, PTFE fluoro elastomer.

If used in solution, or mixed with other substances, and under conditions which differ from EN 374, contact the supplier of the CE approved gloves.

Body Protection:

Wear chemical resistant, protective suit (EN 14605) and boots.

Respiratory protection:

Wear suitable breathing apparatus if exposure levels exceed or may exceed the recommended exposure limits e.g.masks equiped with filter type E (EN 14387) and B, self contained breathing apparatus.

9 Physical and chemical properties

Physical and chemical properties

Appearance (physical state, colour etc) @ 20°C and 1013 hPa:	pale yellow to reddish brown liquid
Odour:	Acrid, suffocating odor
Odour threshold:	Odor low: 0.75 mg/m^3 Odor high: 2.50 mg/m^3 Irritating concn: 155.0 mg/m^3
pH:	1
Melting/Freezing Point @ 101 325 Pa:	232 K
Initial boiling point and boiling range @ 101 325 Pa:	356 K
Flash point:	No data
Evaporation rate:	No data
Flammability (solid, gas):	Non flammable

Upper/lower flammability or explosive limits:	Non explosive
Vapour pressure @ 293 K:	6 200 Pa
Vapour density:	2-3 (est) (Air = 1)
Relative density @ 20 °C:	1.513
Solubility(ies) @ 20 °C:	500 000 mg/L
Partition coefficient: n-octanol/water:	-0.21
Auto-ignition temperature:	No data
Decomposition temperature:	No data
Viscosity @ 25 °C:	0.75 mPa
Particle size distribution (Granulometry):	D50
Oxidising properties:	yes @ >= 65% Category 3 Oxidizing Liquid
Dissociation constant:	pKa = -1
Ionization Potential:	11.95 eV
Refractive Index:	1.393 at 125 deg C/D

Corrosivity

- In presence of traces of oxides it attacks all base metals except aluminum & special chromium steels.
- Nitric acid will attack some forms of plastics, rubber, and coatings.

NOTE: The physical data presented above are typical values and should not be construed as a specification.

10 Stability and reactivity

Reactivity

Air and Water Reactions

Fumes in air. Soluble in all proportions with water. Dissolution in water produces heat, fumes, and spattering.

Reactive Group

Acids, Strong Oxidizing.

Reactivity Alerts

Strong Oxidizing Agent. Known Catalytic Activity. Water-Reactive.

Reactivity Profile

- NITRIC ACID, RED FUMING is a powerful oxidizing agent and nitrating agent. Accelerates the burning of combustible material and may cause charring and then ignition of combustible materials. May ignite alcohols, amines, ammonia, beryllium alkyls, boranes, dicyanogen, hydrazines, hydrocarbons, hydrogen, nitroalkanes, powdered metals, silanes, or thiols on contact [Bretherick 1979. p.174].
- Can react violently with finely divided antimony [Pascal 10:504. 1931-34].
- Reacts violently with bromine pentafluoride [Mellor 2, Supp. 1:172. 1956].
- Reacts with hydrogen selenide and hydrogen sulfide with incandescence [Berichte 3:658].
- Mixtures with finely divided magnesium are explosive [Pieters 1957 p. 28].
- Oxidizes magnesium phosphide with incandescence [Mellor 8:842. 1946-47].
- Mixtures with acetic anhydride containing over 50% nitric acid by mass may act as detonating explosives [BCISC 42:2. 1971].
- An etching agent prepared with equal portions of acetone, nitric acid, and 75% aqueous acetic acid exploded four hours after it was prepared and placed in a closed bottle. The explosive material may have been tetranitromethane [Chem. Eng. News 38: 56. 1960].
- Reacts violently with phosphine [Edin. Roy. Soc. 13:88. 1835].
- Explodes in contact with phosphorus trichloride [Comp. Rend. 28:86].
- Reacts exothermically with phthalic acid or phthalic anhydride in the presence of sulfuric acid to give potentially explosive phthaloyl nitrates or nitrites or nitro derivatives of these compounds [Chem. & Ind. 20:790. 1972].
- Reacts energetically with sodium azide [Mellor 8, Supp 2:315. 1967].
- Reacts with uranium with explosive violence [Katz and Rabinowitch 1951].

Chemical stability

Thermally stable in reaction terms at normal storage conditions.

Possibility of hazardous reactions

Reaction with most common metals liberates hydrogen. Exothermic reaction with water.

Conditions to avoid

Direct heat, high temperature to avoid release of nitric acid fumes and damage of container.

Incompatible materials

Combustible materials, organic matter, reducing agents, alkalies, metallic powders, hydrogen sulphide, alcohols, chlorates and carbides, carbon steel, monel, copper, several other metals and alloys, flammable liquids and chromic acid. Can react violently with reducing agents, strong bases, organic materials, chlorides and finally divided metals. Is corrosive to concrete.

Hazardous decomposition products

When heated, nitric acid and NOx vapours may be evolved.

For fire situations see section “Fire-fighting measures”.

11 Toxicological information

Toxicological (health) effects

Toxicokinetics, metabolism and distribution

A qualitative judgement on the toxicokinetic behaviour was performed based on physico-chemical characteristics. Nitric acid is an inorganic substance and thus some physico-chemical characteristics (like the octanol/water partition coefficient) are not defined, limiting the possibilities of a qualitative assessment. Absorption factors of 100% are proposed for oral, inhalation and dermal absorption. For route-to-route extrapolation starting from oral data, an absorption factor of 50% is proposed as a worst-case assumption. Nitrate can be reduced to nitrite by both enteric bacteria and mammalian nitrate reductase activity. Nitric acid is not considered to have bioaccumulative potential as nitrate is highly soluble in water and rapidly excreted via the urine.

Acute Toxicity Summary

Based on the available studies, the LC50 of pure nitric acid (which is a present as a vapour) is found to be >2.65 mg/L, therefore it is classified cat. 3 for acute inhalation toxicity and should be labelled with H331: Toxic if inhaled according to CLP Regulation EC (No.) 1272/2008. As data are available that indicate that the mechanism of toxicity was corrosivity, in addition to classification for inhalation toxicity, the substance should also be labelled as ‘corrosive to the respiratory tract’ (EUH071).
In absence of data, nitric acid is not classified for acute toxicity via oral and dermal route.

Irritation/Corrosion

Nitric acid is classified as a skin corrosive substance category 1A (concentration > or = 20%) and category 1B (5%< or = concentration) according to CLP Regulation Annex VI, table 3.1 (EC Regulation 1272/2008 on classification, labeling and packaging of substances and mixtures).

Sensitisation

The substance is classified as being corrosive to the skin. A further assessment of the skin sensitisation potential is thus not required.

Repeated Dose Toxicity

No repeated dose inhalation studies conducted with standard methodology were available for nitric acid. One study of sufficient quality identified a LOAEC of ≤50 ug/m3 for nitric acid in rabbits based on a significant reduction of superoxide levels; however at this concentration no changes were observed in the total number of cells recovered by lavage, differential count, cell viability, LDH or total soluble protein in lavage fluid, and no indication of airway hyporesponsivity compared to control. In a 90-day inhalation study with NO2 a NOAEC of ≥ 2.15 ppm (4.11 mg/m3) in rats was identified based on no clinical signs and findings different from normal during the whole study period. Clinical pathology examinations revealed no treatment-related changes in the hematology, clinical chemistry and bronchoalveolar lavage parameters. Nitric acid has been identified as corrosive, which could lead to possible future systemic effects. However no evidence of this was identified in the repeated dose studies. Therefore, no classification can be made due to inconclusive data.

Genetic Toxicity

From the results obtained on nitric acid, sodium and potassium nitrates and due to their structural similarities with nitric acid, it is possible to conclude that nitric acid is not expected to cause genetic toxicity and thus should not be classified according to the CLP Regulation.

Carcinogenicity

Based on the above information nitric acid is not expected to have full carcinogenic properties. Nitric acid does not have to be classified according to Regulation 1272/2008 and amendments.

Toxicity to Reproduction

The results of the OECD screening study (a very high NOAEL) on the read-across substance potassium nitrate do indicate that no classification is required for nitric acid according to the CLP Regulation.

Updatedate	Route / Organism	Dose	Effect
Reproductive Effects			
Sep-13	oral/rat	21150 mg/kg (1-21D pregnant)	Reproductive: Effects on embryo or fetus: Fetotoxicity (except death, e.g., stunted fetus)
Sep-13	oral/rat	2345 mg/kg (18D pregnant)	Reproductive: Effects on newborn: Biochemical and metabolic
Acute Toxicity Data			
Sep-13	In Vitro/Human, liver tumor	Inhibitor Concentration (50 percent kill): 40 mmol/L/24H	In Vitro Toxicity Studies: Cell protein synthesis
Sep-13	inhalation/cat	lowest published toxic concentration: 300 mg/m3/2H	Lung, Thorax, or Respiration: Acute pulmonary edema
Sep-13	inhalation/cat	Lowest published lethal concentration: 500 mg/m3	
Sep-13	inhalation/rat	lethal concentration (50 percent kill): 260 mg/m3/30M	
Sep-13	inhalation/rat	lethal concentration (50 percent kill): 130 mg/m3/4H	
Sep-13	inhalation/rat	lowest published toxic concentration: 460 ppm/1H	Nutritional and Gross Metabolic: Weight loss or decreased weight gain
Sep-13	inhalation/rat	Lowest published lethal concentration: 919 ppm/1H	
Sep-13	oral/human	lowest published lethal dose: 430 mg/kg	
Sep-13	skin/rat	lowest published toxic dose: 150 mL/kg	Blood: Methemoglobinemia-Carboxhemoglobinemia
Sep-13	unreported route/man	lowest published lethal dose: 110 mg/kg	
Other Multiple Dose Data			
		lowest published toxic concentration: 1071	

Sep-13	inhalation/rat	µg/m3/24H/84D-continuous	Behavioral: Muscle contraction or spasticity
Sep-13	inhalation/rat	lowest published toxic concentration: 50 µg/m3/4H/3D-intermittent	Lung, Thorax, or Respiration: Respiratory depression

Information on the likely routes of exposure

Workers - Hazard via inhalation route
Systemic effects
Long term exposure
Hazard assessment conclusion: low hazard (no threshold derived)
Acute/short term exposure
Hazard assessment conclusion: low hazard (no threshold derived)
DNEL related information
Local effects
Long term exposure
Hazard assessment conclusion: other toxicological threshold
Value: 2.6 mg/m³
Most sensitive endpoint: repeated dose toxicity
DNEL related information
DNEL derivation method: ECHA REACH Guidance
Acute/short term exposure
Hazard assessment conclusion: other toxicological threshold
Value: 2.6 mg/m³

Workers - Hazard via dermal route
Systemic effects
Long term exposure
Hazard assessment conclusion: low hazard (no threshold derived)
Acute/short term exposure
Hazard assessment conclusion: hazard unknown (no further information necessary)
DNEL related information
Local effects
Long term exposure
Hazard assessment conclusion: high hazard (no threshold derived)
Acute/short term exposure
Hazard assessment conclusion: high hazard (no threshold derived)

Workers - Hazard for the eyes
Local effects
Hazard assessment conclusion: high hazard (no threshold derived)

General Population - Hazard via inhalation route
Systemic effects
Long term exposure
Hazard assessment conclusion: low hazard (no threshold derived)
Acute/short term exposure
Hazard assessment conclusion: low hazard (no threshold derived)
DNEL related information
Local effects
Long term exposure
Hazard assessment conclusion: other toxicological threshold
Value: 1.3 mg/m³
Acute/short term exposure
Hazard assessment conclusion: other toxicological threshold
Value: 1.3 mg/m³

General Population - Hazard via dermal route
Systemic effects
Long term exposure
Hazard assessment conclusion: low hazard (no threshold derived)
Acute/short term exposure
Hazard assessment conclusion: low hazard (no threshold derived)
DNEL related information
Local effects
Long term exposure
Hazard assessment conclusion: high hazard (no threshold derived)
Acute/short term exposure
Hazard assessment conclusion: high hazard (no threshold derived)

General Population - Hazard via oral route
Systemic effects
Long term exposure
Hazard assessment conclusion: low hazard (no threshold derived)
Acute/short term exposure
Hazard assessment conclusion: hazard unknown (no further information necessary)

General Population - Hazard for the eyes
Local effects
Hazard assessment conclusion: high hazard (no threshold derived)

Additional information - General Population
Nitric acid is not expected to be found in consumer products, or if found it will be found only at trace levels (some ppm in solution).

Symptoms related to the physical, chemical and toxicological characteristics

Symptoms
In contact with the eyes, nitric acid produced severe burns. Depending on the concentration and duration of contact with the eye, these burns may result in eyeball shrinkage, symblepharon (adhesions between tarsal and bulbar conjunctivae), permanent corneal opacification, and visual impairment leading to blindness. On the skin, nitric acid as the liquid or concentrated vapor produced immediate, severe, and penetrating burns. Dermal contact with concentrated solutions of nitric acid have caused deep ulcers and stained the skin yellow or yellowish-brown color (xanthoproteic acid formation). Dilute solutions of nitric acid produced mild epidermal irritation and can harden the epithelium without producing the overt corrosion seen after contact with more concentrated solutions.

Symptoms from breathing in (inhaling) nitric acid may include: bluish colored lips and fingernails, chest tightness, choking, coughing, coughing up blood, dizziness, low blood pressure, rapid pulse, shortness of breath, and weakness. Symptoms from swallowing nitric acid may include: severe abdominal pain, burns to skin or mouth, fever, severe mouth pain, rapid drop in blood pressure, throat swelling (which leads to breathing difficulty), severe throat pain, and bloody vomiting.[A.D.A.M. Medical Encyclopedia [Internet]. Atlanta (GA): A.D.A.M., Inc.; 2005. Nitric acid poisoning;

SYMPTOMATOLOGY (after ingestion or skin contact)

- Corrosion of mucous membranes of mouth, throat, and esophagus, with immediate pain and dysphagia. The necrotic areas are at first grayish white but soon acquire a blackish discoloration and sometimes a shrunken or wrinkled texture; The process is described as a "coagulation necrosis."
- Epigastric pain, which may be associated with nausea and the vomiting of mucoid and "coffee-ground" material. At times, gastric hemorrhage may be intense, and the vomitus then contains fresh blood. Profound thirst. 3) Ulceration of all membranes and tissues with which the acid comes in contact.

Nitric acid levels in the range of 250-500 ug/cu m (97-194 ppb) may cause some pulmonary function responses in adolescent asthmatics, but not in healthy adults.

Case reports
Ingestion of some 30 to 40 mL produced acute corrosion and coagulative necrosis of the mouth, esophagus, and stomach, with deaths delayed 12 hours to 14 days or several months, the later attributed to a chemical lobar pneumonitis secondary to aspiration. Among those who survived, strictures of the gastric mucosa and subsequent pernicious anemia were

described.

A case of acute inhalation injury of nitric acid in a 56-year old white male /was reported/. The patient presented conscious and dyspnoic at the emergency department after cleaning a copper chandelier with nitric acid. He had to be intubated 2 hr after admission and mechanically ventilated because of fulminant respiratory insufficiency. As all sources of mechanical ventilation failed, extracorporeal membrane oxygenation had to be established 7 hr after admission. With the additional use of surfactant and low dose inhalation therapy with nitric oxide (NO), the patient could be stabilised for 3 days and lung function improved temporarily. Despite all efforts the patient died at the fourth day from refactory respiratory failure. Pathologic examination revealed massive pulmonary edema without signs of inflammation. Thus, nitric acid inhalation induced pulmonary edema appears to be a most severe situation in which even most modern therapeutic interventions fail.

Nitric acid in contact with eyes causes immediate opacification of corneal and conjunctival epithelium, imparting yellow color when acid is concentrated. In accidental application to eyes of newborn children several eyes have been lost as result of corneal opacification, symblepharon, and shrinkage of globe.

A 25 year-old truck driver developed, and 4 days after succumbed to, acute dyspnea some 3 weeks after inhaling a considerable amount of fumes while cleaning up spilled 60% nitric acid. All stages of extensive bronchiolitis and alveolitis obliterans were found.

A 42 year old male worker was accidentally sprayed in the face with concentrated nitric acid. Immediately after the accident, his face was bathed with water, and he was rushed to /a hospital/ within 30 min. On admission, he was diagnosed as having a burn injury of the face and right cornea. Prolonged wound irrigation using normal saline was started immediately and continued for 24 hr. The burned skin and the corneal ulcer healed without any serious complications, except for some spotted, full thickness skin loss on the left auricle, since moist gauze that had been placed on the auricle slipped off before completion of the hydrotherapy. Yet, even these ulcers epithelialized within 10 days after this injury.

The report presents/ a series of three cases of survival following inhalation of nitric acid fumes, which resulted in acute respiratory distress. Inhalation of nitric acid fumes and its decomposition gases such as nitrogen dioxide results in delayed onset of acute respiratory distress syndrome. Intensive respiratory management, ventilatory support, and steroids can help in survival.

Nitric acid (HNO_3) is a solution of nitrogen dioxide (NO_2) in water commonly used as an industrial chemical and cleaner. Oxides of nitrogen liberated as nitric acid interact with the environment to cause inhalation injuries. The coexistence of HNO_3 with varying oxides of nitrogen likely results in the large continuum of symptoms related to HNO_3 exposure and varying times of onset-acute, subacute, and delayed. Furthermore, dyspnea and evidence of acute lung injury may not occur for several hours after exposure and can lead to rapidly progressive acute respiratory distress syndrome (ARDS). This case illustrates to physicians and occupational health personnel that HNO_3 inhalation may initially appear benign and that onset of severe effects may be delayed. In this case a 66-year-old man developed delayed-onset pulmonary edema, ARDS, and fatal circulatory collapse 53 hr after occupational exposure to HNO_3. This case serves to increase awareness among emergency physicians, as well as occupational health personnel, that patients exposed to HNO_3 may initially be asymptomatic. Patients should be evaluated and observed regardless of the severity or benign nature of symptoms, which occur immediately after exposure, as the most severe symptoms are often delayed in onset and rapidly progressive.

Two patients presented with potentially fatal pulmonary edema after accidental exposure to nitric and hydrofluoric acid fumes during electroplating. Despite aggressive respiratory support, one succumbed to respiratory failure 3.5 hr after inhalation. The other patient also rapidly progressed to respiratory failure. Extracorporeal life support (ECLS) was started 5 hr after exposure at the ED. During ECLS, hypoxia improved, but pulmonary edema shown by chest radiography became aggravated. N-Acetyl cysteine and calcium gluconate were given iv on the first day of admission and nebulised for 48 hr after exposure. Pulmonary secretions were significantly reduced 24 hr after the nebulising therapy began. Ultimately, the patient was discharged without serious pulmonary or neurological complications after 28 days of hospitalisation. In this case, early ECLS, nebulised antioxidant and antidote were available to treat potentially fatal pulmonary edema after exposure to nitric and hydrofluoric acid fumes.

The report describes/ two cases of acute lung injury after the inhalation of nitric acid fumes in an industrial accident. The first patient, who was not using a respirator and standing in close proximity to the site of spillage of concentrated nitric acid, presented within 12 hr with worsening dyspnea and required noninvasive ventilation for type 1 respiratory failure. The second case presented 1 day later with similar symptoms, but only required supportive treatment with high-flow oxygen. Both patients' chest radiographs showed widespread bilateral airspace shadows consistent with acute lung injury.

Both received treatment with systemic steroids. They were discharged from hospital 5 days postexposure. Initial lung function test showed a restrictive pattern that normalized by 3 weeks postexposure. This case series describes the natural history after acute inhalation of nitric acid fumes, and demonstrates that the severity of lung injury is directly dependent on the exposure level. It also highlights the use of noninvasive ventilatory support in the management of such patients.

There is no doubt that very high exposure concentrations of HNO3 are lethal to humans. ''Rapidly progressive pulmonary edema of delayed onset" was observed after an exposure (10-15 min) of three young healthy men to fumes from an explosion of a tank that contained approximately 1,736 liters (L) of 66% HNO3. The onset of "respiratory difficulties" occurred approximately 4-6 hr after the accident; all died within 24 hr of the exposure.

Human exposure studies
The use of a 0.2-ppm NOEL for healthy humans exposed to HNO3 based on a previous study appears appropriate. However, subsequent studies indicate that individuals with asthma might be more sensitive than those without. In these studies, adolescent asthmatic subjects (six males and nine females) with exercise-induced bronchospasm were exposed via a rubber mouthpiece with nose clips to HNO3 aerosol at a concentration of 0.05 ppm (130 ug/cu m). Respiratory function was measured at the end of the exposure. The exposure at 0.05 ppm for 40 min (30 min at rest followed by a 10-min moderate exercise period) produced a decrease in forced expiratory volume (FEV) of 4% and an increase in respiratory resistance of 23%. The authors concluded that individuals with asthma represent a population group that might be exquisitely more sensitive to the effects of HNO3 than healthy individuals.

Investigators showed that a 200-ug/cu m (0.08 ppm) exposure for 2 hr produced no adverse respiratory effects in nine human subjects. That concentration was chosen to represent a concentration of HNO3 that was higher than those observed in ambient air (EPA 1993); ambient concentrations ranged from 0.1 to 20 ppb. Later, the same investigators used the same exposure concentration and duration, but did not observe any adverse effects as judged by bronchoalveolar lavage and pulmonary-function tests. None of the biochemical measures, such as protein levels, lactate dehydrogenase (LDH), and fibronectin, changed as a result of the exposure. The investigators observed a surprising increase in the phagocytic activity of alveolar macrophages from exposed individuals, but that was not believed to be an adverse effect. Therefore, no toxic effects occurred in humans exposed to HNO3 for 2 hr at 0.08 ppm.

Investigators conducted a study to determine the effects of HNO3 gas, not aerosol, in healthy humans. They designed the study to reflect the conditions of exposure that might occur in dry weather. The test subjects (eight males and two females) were exposed to HNO3 at 500 ug/cu m (0.2 ppm) in a 2.5 x 2.5 x 2.4 meter chamber for 4 hr during moderate exercise. Eighteen hours later the subjects underwent bronchoscopy, which included bronchial lavage and bronchial biopsy to evaluate biochemical and morphological changes. The study was done carefully and a number of end points were examined, including pulmonary function. None of the assessments of respiratory toxicity showed any effects from HNO3 gas in the 10 subjects. Therefore, a human no-observed-effect level (NOEL) for HNO3 can be established as 0.2 ppm for 4 hr.

In a study with humans, exposure of 12 nonsmoking subjects with mild asthma for 3 min to an "acid fog" that was 30 milliosmolar (mOsm) at pH 2 significantly increased specific airway resistance. The approximate concentration of HNO3 in those studies was very high, 40 mg/cu m (15 ppm). Subjects inhaled the aerosols through a mouthpiece from an ultrasonic nebulizer. Bronchoconstriction was correlated with acidity of the fog, not the nature of the acid, since fogs made up of either HNO3 or H2SO4 or both showed equally potent effects. These studies showed that very short exposures to high concentrations of HNO3 can cause moderate-to-severe effects in sensitive humans.

The vapor and mists of nitric acid may erode teeth, particularly affecting the canines and incisors. Investigators examined 32 workers who were exposed only to nitric acid; 9% showed evidence of active dental erosion. No occupational hygiene measurements were reported. Nitric acid was suggested to be less potent than sulfuric or hydrochloric acid in eroding teeth.

Other toxicity information
The toxicity of HNO3 is predominately associated with the extremely corrosive nature of this strong acid. In addition, it is an excellent oxidizing agent and reacts immediately with any tissue to cause such effects as skin burns, eye irritation, coughing, dyspnea, and pulmonary edema. Delayed toxicity, possibly as a result of the decomposition of HNO3 to other nitrogen oxides, could produce methemoglobinemia, but no documentation exists to support that hypothesis.

Delayed and immediate effects and also chronic effects from short and long term exposure

Non-Human Toxicity Excerpts
Laboratory animals: acute exposure

A single exposure to nitric acid mist in a concentration of 63 mg/cu m had no apparent adverse effect on rats.

Based on acute parallel exposures of rats to white (97.5%) nitric acid vapor, nitrogen dioxide, or nitrogen dioxide (8%-17% dissolved) in red fuming nitric acid. Investigators concluded nitrogen dioxide was the primary toxic constituent of fuming nitric acid and that nitric acid may potentiate the toxicity of inhaled nitrogen dioxide. Nitric acid vapor alone was less toxic to rats on inhalation than was an equivalent exposure to nitrogen dioxide or the red fuming preparation of nitric acid.

Following a transorotracheal instillation of 0.5% nitric acid (HNO3) in saline, Syrian golden hamsters showed during a 60 day study period a spectrum of airway changes including acute bronchitis, acute bronchiolitis, obliterative bronchiolitis, bronchioletasia, and bronchiectasis. Morphometric changes in the HNO3 treated hamsters included decreased lung volumes and decreased internal surface areas. Biochemical changes showed increases in lung weight and in total collagen and elastin.

Hamsters were exposed to an intratracheal installation of 0.5 mL of 0.08N nitric acid (HNO3) to determine their airway epithelial response. Three weeks after exposure, the left intrapulmonary bronchi in Alcian blue/PAS stained paraffin sections were evaluated for the amount of secretory product in the airway epithelium as a measure of secretory cell metaplasia. Compared to saline treated control animals, acid caused statistically significant secretory cell metaplasia. In addition to the bronchial lesion, acid caused similar interstitial fibrosis, bronchiolectasis, and bronchiolization of alveoli that varied in individual animals from mild to severe. In a separated experiment to study the persistence of the secretory cell metaplasia, hamsters treated with a single instillation of 0.1N HNO3 showed significant secretory cell metaplasia 3, 7, and 17 weeks after exposure. There was a high correlation (r= 0.96) between a subjective assessment of secretory cell metaplasia and objective assessment using a digital image analysis system. Apparently, protons induce secretory cell metaplasia independently of the associated anion; the secretory cell metaplasia persists at least 17 weeks. HNO3 may contribute to the development or maintenance of the secretory cell metaplasia seen in the conducting airways of humans with chronic obstructive pulmonary disease. PubMed Abstract

Respiratory disorders, including pulmonary edema, can occur several hours after an acute exposure and are probably related to inflammation resulting from cellular necrosis in lung tissues. Alveolar type II cells and cell hyperplasia of alveoli are primary cellular responses in the deep lung in animals treated with HNO3 by instillation (1% solution). Bronchiolitis and alveolitis are associated with instillation of 1% HNO3 in rats.

Laboratory animals: Subchronic or Prechronic Exposure

The relationship between the pulmonary histologic changes and the physiologic changes were examined in models of chronic bronchial injury. A model of airway obstruction that avoids long term toxic exposure was created. Seven mongrel dogs weighing 15 to 19 kg were studied. Dogs were exposed to 1% nitric acid on alternate days for four wk. Lung mechanics were measured prior to exposure and after 2 and 4 wk. Both total lung capacity and vital capacity decr and the functional residual capacity/total lung capacity ratio incr. Expiratory flows decr while breathing air and an 80% helium, 20% oxygen mixture; the volume of isoflow incr. Pulmonary resistance incr and dynamic compliance decr. On single breath nitrogen washout, there were incr in the slope of phase III and in closing capacity. Histologically, there was widespread chronic airway inflammation, slight epithelial changes, slight peribronchiolar fibrosis, and an incr in smooth muscle. Pathologic scores were significantly higher in the nitric acid exposed group than in controls. Scores for peripheral and central airway pathology were correlated with results of tests of airway obstruction.

A canine model of nitric acid (HNO3) induced chronic bronchial injury was examined to determine the duration and correlations of pathophysiological changes following HNO3 exposure. Bronchial injury was induced in seven dogs by exposure to nebulized 1% HNO3 3 days per wk for 4 wk. Lung mechanics were assessed prior to exposure, after 4 wk of exposure, and at 1, 3, and 5 mo post exposure. Measurements included arterial blood gas determinations, pulmonary resistance, dynamic compliance, static compliance, flow/volume, the single breath oxygen test, and histamine challenge. The five dogs remaining alive at the end of the 5 mo test period were sacrificed and their lungs and trachea fixed for histological examination. Significant and long term airway obstruction was observed in HNO3 exposed animals, with reductions in maximal expiratory flows. Major pathologic changes were confined to the small, noncartilaginous airways, with widespread chronic airway inflammation evident. Acute changes such as edema and pneumonitis had subsided within 2 to 3 mo of exposure. Hyporesponsiveness to histamine was observed post exposure, with dogs requiring 2.7 times the control dose to achieve a doubling of pulmonary resistance; by 3 mo normal histamine responsiveness had returned in three of the five dogs and by 5 mo all were hyperresponsive, requiring one half the initial dose to double resistance. Significant correlations were found between small airways pathology scores and flows at 50 and 25% of the vital capacity, as well as changes in pulmonary resistance.

Laboratory animals: Chronic Exposure or Carcinogenicity

In rats exposed to 0.013-0.049 mg/L nitric acid aerosol for 375-650 days, mortality was 9-25%. Benign lesions in bone (osteoarthritis) were seen in controls and acid-exposed animals.

Genotoxicity
In an Ames test Salmonella typhimurium TA1535, TA100, TA1537, TA98 were treated with nitric acid at concentrations of 20 - 5,000 ug/plate with and without metabolic activation. The result was negative.

Other toxicity information
Symptoms: Ingestion of any caustic agent leads to corrosion of the mucous membranes of the upper part of the digestive tract. Vomition, colic, and purgation may follow, with prostration and death from acute shock.

Numerical measures of toxicity (such as acute toxicity estimates)

	LC_{50} Inhalation
Sheep	0.004 mg/L 4 hr
Rat	7 mg/L 1hr
Mouse	67 ppm/4hr Nitric acid, red fuming
Rat	334 ppm/30 min
Rat	244 ppm/30 min
Rat	138 ppm/30 min Red fuming nitric acid, 8 to 17% nitrogen dioxide
Rat	65 ppm/4 hr Red fuming nitric acid
Mouse	244 ppm/30 min

Interactive effects

Respiratory tract injury resulting from inhalation of mixtures of ozone and nitrogen dioxide and of ozone and formaldehyde was studied in Sprague-Dawley rats under exposure conditions of rest and exercise. Mixtures of ozone (0.35 or 0.6 ppm) with nitrogen dioxide (respectively 0.6 or 2.5 ppm) doubled the level of lung injury produced by ozone alone in resting exposures to the higher concn and in exercising exposures to the lower concn. Mixtures of ozone and nitrogen dioxide at high and low concn formed respectively 0.73 and 0.02 ppm nitric acid (HNO3) vapor. Chemical interactions among the oxidants, HNO3, and other reaction products (nitrogen pentoxide and nitrate radical) and lung tissue may be the basis for the ozone-nitrogen dioxide synergism. Increased dose and dose rate associated with exercise exposure may explain the presence of synergistic interaction at lower concn than observed in resting exposure. No oxidation products were detected in ozone-formaldehyde mixtures, and the antagonistic interaction observed in lung tissue during resting exposure may result from irritant breathing pattern interactions.

Other information

Medical Surveillance
A complete history and physical exam:
The purpose is to detect existing conditions that might place the exposed employee at incr risk, and to establish a baseline for future health monitoring. Examination of the eyes, respiratory tract, skin, and teeth should be stressed. The skin should be examined for evidence of chronic disorders.

NIOSH recommends that workers subject to nitric acid exposure have comprehensive preplacement and annual medical examinations including a 14"X17" posterior-anterior chest x-ray, pulmonary function tests, and a visual examination of the teeth for evidence of dental erosion.

Respiratory Symptom Questionnaires:
Questionnaires have been published by the American Thoracic Society and the British Medical Research Council. These questionnaires have been found to be useful in identification of people with chronic bronchitis, however certain pulmonary function tests such as FEV 1 have been found to be better predictors of chronic airflow obstruction.

Chest Radiography:
This test is widely used for assessing pulmonary disease. Chest radiographs have been found to be useful for detection of early lung cancer in asymptomatic people, especially for detection of peripheral tumors such as adenocarcinomas. However, even though OSHA mandates this test for exposure to some toxicants such as asbestos, there are conflicting

views on its efficacy in detection of pulmonary disease.

Pulmonary Function Tests:
The tests that have been found to be practical for population monitoring include: Spirometry and expiratory flow-volume curves; Determination of lung volumes; Diffusing capacity for carbon monoxide; Single-breath nitrogen washout; Inhalation challenge tests; Serial measurements of peak expiratory flow; Exercise testing. Spirometry, for the measurement of FVC (forced vital capacity) and FEV1 (forced expiratory volume in 1 second), has been found to be the most reproducible and least variable test of pulmonary function.

Sputum Cytology:
Sputum cytology along with chest radiographs have been the standard procedures for detecting early lung cancer in asymptomatic patients. Sputum cytology has been found to be useful for detection of central tumors, especially squamous carcinomas. For this test to be effective, exfoliated respiratory mucosal cells must be present in the expectorated specimen. Pooling of sputum collected over 2-3 days may enhance the sensitivity of this test by increasing the yield of exfoliated cells in the specimen.

12 Ecological information

Toxicity

Hazard for aquatic organisms
Freshwater
Hazard assessment conclusion: no hazard identified
Marine water
Hazard assessment conclusion: no hazard identified
STP
Hazard assessment conclusion: no hazard identified
Sediment (freshwater)
Hazard assessment conclusion: no hazard identified
Sediment (marine water)
Hazard assessment conclusion: no hazard identified

Hazard for air
Air
Hazard assessment conclusion: no hazard identified

Hazard for terrestrial organisms
Soil
Hazard assessment conclusion: no hazard identified

Hazard for predators
Secondary poisoning
Hazard assessment conclusion: no potential for bioaccumulation

Additional information
Nitrogen is an important nutrient in ecosystems: it is essential for microorganisms, plants and animals.

Nitric acid almost completely (93% at 0,1 M) ionizes into the nitrate ion NO_3^- and the hydronium H_3O^+ under environmental conditions. Once released in the environment, it may be deposited in water, soil and vegetation. When spilled onto soil, nitric acid will infiltrate according to the soil viscosity. During transport through the soil, nitric acid will dissolve some of the soil material, in particular, carbonate based materials (OECD SIDS Nitric acid Assessment Report, 2008; HSDB, 1999). The nitrate released from nitric acid is taken up by plants or denitrified by microorganisms to nitrogen or nitrous oxide.

Because of the quick dissolution nitric acid undergoes once in contact with water, when nitric acid is applied as fertilizer, very low pH values can be attained. The acidity will then gradually diffuse into the soil surrounding the band.

Nitric acid exposed to air will result in nitrogen oxides release (mainly NO_2 and NO, OECD SIDS Nitric acid Assessment Report, 2008).

Conclusion on classification
The main characteristic of the nitric acid that drives its toxicity is embedded in the fact it is a (strong) acid that dissociates in water into its respective ions H+ and NO3- and will affect the environment and its organisms by decreasing the pH. The increasing nitrate concentration is judged to have a minor effect on aquatic organisms compared to the effect of pH. The EU limit value of 50 mg NO3-/L (0.8 mmol NO3-/L) for water corresponds to a pH of 3.1 when all nitrate comes from nitric acid. Therefore, the effects assessment of nitric acid is focused on the pH effect and to a certain extent, this is also a read across exercise that identifies a category of substances which toxicological behavior can be characterized based on the acid properties of the substance.

The substance will therefore not be classified for environmental hazards, based on the available information.

Short-term toxicity to fish
Median lethal pH (96h) 3-3.5 for Lepomis macrochirus (no guideline followed).
Median lethal pH (96h) ca. 3.7 for Oncorhynchus mykiss (no guideline followed).

The available studies show that the pH rather than the anion (nitrate) is causing the toxic effects in fish. This is confirmed by various studies with sodium nitrate: LC50 (96h) > 1000 mg/L to fish (no guideline followed). The read-across rationale can be found in the category approach document attached in Section 13 of IUCLID and is fully incorporated in the CSR.

Additional information
Both K2 studies focus on the pH effects caused by nitric acid. Different pH levels have been tested in bluegill sunfish and rainbow trout.

The pH induced by nitric acid which caused 50% mortality was ca. 3.7 in rainbow trout and between 3.0 and 3.25 in bluegill sunfish.
No mortality was observed at pH 3.5 or above in bluegil sunfish after 96h exposure.

No mortality was observed at pH 4.0 in rainbow trout after 96h exposure, although 50% had died after 7 days of exposure.

The studies show that a pH caused by adding nitric acid roughly between pH 3 (or lower) and 4 is critical for fish. Furthermore, the Swift study shows that the nitrate ion alone (used as control) is not causing any mortality in fish. Also the studies from Westin (1974), Adelman et al. (2009), Trama (1954) and Wallen et al. (1957) conducted with sodium nitrate in marine and freshwater fish showed no effects (LC50 ≥ 1000 mg nitrate/L). It can thus be concluded that it is the low pH which is causing the toxic effects.

As regulatory ecotoxicity tests need to be conducted at pH 6-9, nitric acid will not cause adverse effects to fish when in this pH range.

Long-term toxicity to fish
The endpoint 'long-term toxicity to fish' can be covered by a waiver which is based on two arguments:

- according to REACH Annex IX column 2, the study does not need to be performed when the chemical safety assessment indicates no need to further investigate the effects on aquatic organisms. The CSA indicated no risks for the aquatic environment, so the aforementioned waiving argument is valid.
- according to REACH Annex XI section 1.5, the study can be waived when it can be covered by a structural analogue. Nitric acid will dissociate in the environment into its ions: H+ ions and nitrate ions. The H+ ions will cause a pH decrease, but in the aquatic environment the net pH decrease will be limited due to buffering. Regulatory aquatic ecotoxicity studies should be conducted at relevant environmental pH (i.e. pH 6 -9). At this pH, exposure to nitric acid comes down to exposure to nitrate ions. Studies done on sodium nitrate, which also dissociates into nitrate ions, can be used to support the low long-term aquatic toxicity of nitrate: There are reliable 30d growth rate and 32d embryo-larval tests available for sodium nitrate from Adelman (2009). In the 30d growth rate test the NOEC for juvenile Topeka shiner was 268 mg/L (growth rate) and the NOEC for Fathead minnow was 58 mg/L (mortality).

In the 32d embryo-larval test, the NOEC to Fathead minnow was 157 mg/L based on growth rate (no effect on embryo survival).
These tests confirm the low long-term toxicity of sodium nitrate.

Short-term toxicity to aquatic invertebrates
Median lethal pH (48h) 4.4-4.7 for Ceriodaphnia dubia (US EPA guideline). This study shows that the pH rather than the anion (nitrate) is causing the toxic effects in daphnids. This is confirmed by two additional studies with sodium nitrate (24h

EC50 8609 mg/L for Daphnia magna; similar to OECD TG 202) and potassium nitrate (48h EC50 490 mg/L for Daphnia magna; no guideline followed). The read-across rationale can be found in the category approach document attached in Section 13 of IUCLID and is fully incorporated in the CSR.

Additional information
In the Belanger and Cherry study (1990), the water flea Ceriodaphnia dubia was exposed to nitric acid pH-adjusted test solutions for 48 hours with test solution renewal at 24 hours. The 48-hour LC50 was determined after testing two pH ranges, 3.4-8.1 and 3.2-8.0. The grand probit 48-hour LC50 (combined analyses) was 4.6 pH standard units.

Similar to the other aquatic toxicity endpoints, it is shown that adverse effects due to nitric acid exposure are caused by the decreased pH and not by the nitrate anion. This finding is strengthened by the BASF (1983) and Dowden and Bennett (1965) studies which show high LC50 values in daphnids exposed to sodium nitrate (24h-LC50 = 8609 mg/L) or potassium nitrate (96h-EC50 = 900 mg/L).

As regulatory ecotoxicity tests need to be conducted at pH 6-9, nitric acid will not cause adverse effects to daphnids when in this pH range.

Long-term toxicity to aquatic invertebrates
According to REACH Annex IX no testing is required if the results of the chemical safety assessment indicates no need to investigate further the effects of the substance on aquatic organisms. The CSA indicated no concern for daphnia.Furthermore, long-term effects to daphnia at environmentally relevant pH values are not expected to occur (Belanger and Cherry, 1990).

Toxicity to aquatic algae and cyanobacteria
A waiver is proposed to cover this endpoint, and consequently a study on potassium nitrate (Admiraal, 1977) is provided as supporting evidence. The read-across rationale can be found in the category approach document attached in Section 13 of IUCLID and is fully incorporated in the CSR.

Additional information
According to REACH Annex XI section 1.5, the study can be waived when it can be covered by a structural analogue. Nitric acid will dissociate in the environment into its ions: H+ ions and nitrate ions. The H+ ions will cause a pH decrease, but in the aquatic environment the net pH decrease will be limited due to buffering. Regulatory aquatic ecotoxicity studies should be conducted at relevant environmental pH (i.e. pH 6 -9). At this pH, exposure to nitric acid comes down to exposure to nitrate ions. Therefore this endpoint can be covered by a study conducted with potassium nitrate (Admiraal, 1970), which also dissociates in the environment into its ions. In this study, several marine benthic diatoms were exposed to potassium nitrate. The derived NOEC = 6.75 mmol/L (i.e. 419 mg nitrate/L).

Toxicity to microorganisms
A waiver is proposed to cover this endpoint, and supporting evidence with a study on sodium nitrate (Schwarz, 2008) is provided. The read-across rationale can be found in the category approach document attached in Section 13 of IUCLID and is fully incorporated in the CSR.

Additional information
Nitric acid in water will dissociate in nitrate anions and H+ ions, the latter causing a decrease of pH. However, pH levels in wastewater are typically adjusted in wastewater treatment plants to ensure a neutral discharge to the receiving water (e.g., pH between 6-9). Therefore, the microorganisms are essentially exposed to nitrate and not to nitric acid or to low pH values. The study therefore does not need to be performed, according to REACH Annex VIII column 2.

In addition, nitrogen is an essential nutrient for activated sludge systems, as it comprises part of the biomass in the activated sludge. Typical nitrate levels in domestic sewage are near zero; bacterial oxidation of the ammonia present in sewage produces nitrates. Where excessive ammonia levels are of concern, treatment plants are designed to incorporate a two-step nitrification-denitrification process: (1) ammonia is converted to nitrite and then nitrate, and (2) nitrate is reduced anoxically to NO, N2O, and N2 gases.

Furthermore, in accordance with REACH Annex XI, section 1.5 (Grouping of substances and read-across approach) the toxicity test on microorganisms does not need to be conducted as it can be covered with a study done on sodium nitrate, which also dissociates into nitrate ions. This study (Schwarz, 2008) showed a 3h-EC50 of >1000 mg/L.

Persistence and degradability

The environmental fate endpoints are all waived. Due to its inorganic nature, the adsorption/desorption screening test cannot be conducted, while QSARs are not applicable for these kind of substances. Also biodegradation tests are not applicable to inorganics.

Due to its high solubility in water, nitric acid will be dissociated into its ions (H+ and NO3 -) and in water the H+ ions will form H3O+ ions. The hydrolysis endpoint can therefore be waived. Bioaccumulation is not relevant for such highly soluble and dissociating substances.

Bioaccumulative potential

Refer above "Persistence and degradability".

Mobility in soil

Refer above "Persistence and degradability".

Other adverse effects

No additional data.

13 Disposal considerations

Disposal methods

Waste disposal recommendations:
At the time of review, criteria for land treatment or burial (sanitary landfill) disposal practices are subject to significant revision. Prior to implementing land disposal of waste residue (including waste sludge), consult with environmental regulatory agencies for guidance on acceptable disposal practices.

Dispose of waste and container in accordance with local and/or national regulations. Hazardous waste shall not be mixed together with other waste. Different types of hazardous waste shall not be mixed together if this may entail a risk of pollution or create problems for the further management of the waste. Hazardous waste shall be managed responsibly. All entities that store, transport or handle hazardous waste shall take the necessary measures to prevent risks of pollution or damage to people or animals. Recycle/reuse. Remove for physico-chemical/biological treatment. **DO NOT** discharge into drains or the environment.

Neutralize carefully with lime or carbonates.

Ecology - waste materials:
Avoid release to the environment.

Empty Container:
Consider refilling. Container can be re-used. Rinse/Decontaminate thoroughly before re-use, discarding in trash or return to supplier.

14 Transport information

UN Number

TRANSPORTATION CLASSIFICATION	ADR/RID	ADN(R)	IMDG	ICAO/IATA
Identification Number	2031	2031	2031	2031
Proper Shipping Name	NITRIC ACID, other than red fuming, with <70% nitric acid	NITRIC ACID, other than red fuming, with <70% nitric acid	NITRIC ACID, other than red fuming, with <70% nitric acid	NITRIC ACID, other than red fuming, with <70% nitric acid
Transport Hazard Class(es)	8 	8 	8 	8 
Packing Group	II	II	II	II
Exempt Quantity / Quantity Limits	50Kg F: 20	-	-	**Passenger Aircraft** Forbidden

				Cargo Aircraft 30L
Classification Code	C1	C1	-	-
Environmental Hazards	No	No	No	No
Emergency Response	ERG: 157	-	EMS: F-A, S-B	-
Additional Information	-	-	-	-

UN Proper Shipping Name

Refer section 14.1 above.

Transport hazard class(es)

Refer section 14.1 above.

Packing group, if applicable

Refer section 14.1 above.

Special precautions for user

DO NOT load with Class 1 and 2.3.
Keep aluminium gas cylinders apart from caustic bases.
May be loaded with Class 2.1, 2.2, 5.2, 6.1, 6.2 and 8A if kept at least 1 metre apart.
Can be loaded with all other classes.
Goods of different classes **must** be segregated by an air space of at least 100mm or by an approved segregation device or non-dangerous goods.

P, B, L and O provisions as per SANS 10231:2006
None

GUIDE 157: SUBSTANCES - TOXIC AND/OR CORROSIVE (NON-COMBUSTIBLE/WATER -SENSITIVE)

Health

TOXIC; inhalation, ingestion or contact (skin, eyes) with vapors, dusts or substance may cause severe injury, burns, or death. Reaction with water or moist air will release toxic, corrosive or flammable gases. Reaction with water may generate much heat which will increase the concentration of fumes in the air. Fire will produce irritating, corrosive and/or toxic gases. Runoff from fire control or dilution water may be corrosive and/or toxic and cause pollution.

Fire or Explosion

Non-combustible, substance itself does not burn but may decompose upon heating to produce corrosive and/or toxic fumes. Vapors may accumulate in confined areas (basement, tanks, hopper/tank cars etc.). Substance will react with water (some violently), releasing corrosive and/or toxic gases. Contact with metals may evolve flammable hydrogen gas. Containers may explode when heated or contaminated with water.

Public Safety

CALL Emergency Response Telephone Number. As an immediate precautionary measure, isolate spill or leak area in all directions for at least 50 meters (150 feet) for liquids and at least 25 meters (75 feet) for solids. Keep unauthorized personnel away. Stay upwind. Keep out of low areas. Ventilate enclosed areas.

Protective Clothing

Wear positive pressure self-contained breathing apparatus (SCBA). Wear chemical protective clothing that is specifically recommended by the manufacturer. It may provide little or no thermal protection. Structural firefighters' protective clothing provides limited protection in fire situations ONLY; it is not effective in spill situations where direct contact with the substance is possible.

Evacuation

Fire: If tank, rail car or tank truck is involved in a fire, ISOLATE for 800 meters (1/2 mile) in all directions; also, consider initial evacuation for 800 meters (1/2 mile) in all directions.

Fire

Note: Most foams will react with the material and release corrosive/toxic gases. Small fires: CO2, dry chemical, dry sand,

alcohol-resistant foam. Large fires: Water spray, fog or alcohol-resistant foam. Move containers from fire area if you can do it without risk. Use water spray or fog; do not use straight streams. Dike fire control water for later disposal; do not scatter the material. Fire involving tanks or car/trailer loads: Fight fire from maximum distance or use unmanned hose holders or monitor nozzles. Do not get water inside containers. Cool containers with flooding quantities of water until well after fire is out. Withdraw immediately in case of rising sound from venting safety devices or discoloration of tank. ALWAYS stay away from tanks engulfed in fire.

Spill or Leak

ELIMINATE all ignition sources (no smoking, flares, sparks or flames in immediate area). All equipment used when handling the product must be grounded. Do not touch damaged containers or spilled material unless wearing appropriate protective clothing. Stop leak if you can do it without risk. A vapor suppressing foam may be used to reduce vapors. DO NOT GET WATER INSIDE CONTAINERS. Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact spilled material. Prevent entry into waterways, sewers, basements or confined areas. Small spills: Cover with DRY earth, DRY sand, or other non-combustible material followed with plastic sheet to minimize spreading or contact with rain. Use clean non-sparking tools to collect material and place it into loosely covered plastic containers for later disposal.

First Aid

Move victim to fresh air. Call 911 or emergency medical service. Give artificial respiration if victim is not breathing. Do not use mouth-to-mouth method if victim ingested or inhaled the substance; give artificial respiration with the aid of a pocket mask equipped with a one-way valve or other proper respiratory medical device. Administer oxygen if breathing is difficult. Remove and isolate contaminated clothing and shoes. In case of contact with substance, immediately flush skin or eyes with running water for at least 20 minutes. For minor skin contact, avoid spreading material on unaffected skin. Keep victim warm and quiet. Effects of exposure (inhalation, ingestion or skin contact) to substance may be delayed. Ensure that medical personnel are aware of the material(s) involved and take precautions to protect themselves.

Shipment Methods and Regulations

No person may transport, offer or accept a hazardous material for transportation in commerce unless that person is registered in conformance and the hazardous material is properly classed, described, packaged, marked, labeled, and in condition for shipment as required or authorized by the hazardous materials regulations (49 CFR 171-177).

The International Air Transport Association (IATA) Dangerous Goods Regulations are published by the IATA Dangerous Goods Board pursuant to IATA Resolutions 618 and 619 and constitute a manual of industry carrier regulations to be followed by all IATA Member airlines when transporting hazardous materials.

The International Maritime Dangerous Goods Code lays down basic principles for transporting hazardous chemicals. Detailed recommendations for individual substances and a number of recommendations for good practice are included in the classes dealing with such substances. A general index of technical names has also been compiled. This index should always be consulted when attempting to locate the appropriate procedures to be used when shipping any substance or article.

DOT Emergency Guidelines

If THERE IS NO FIRE, go directly to the Table of Initial Isolation and Protective Action Distances (see table below) to obtain initial isolation and protective action distances. IF THERE IS A FIRE, or IF A FIRE IS INVOLVED, go directly to the appropriate guide (see guide(s) above) and use the evacuation information shown under PUBLIC SAFETY.

Small Spills (from a small package or small leak from a large package	
First ISOLATE in all Directions	30 meters (100 feet)
Then PROTECT persons Downwind during DAY	0.1 kilometers (0.1 miles)
Then PROTECT persons Downwind during NIGHT	0.3 kilometers (0.2 miles)
Large Spills (from a large package or from many small packages)	
First ISOLATE in all Directions	150 meters (500 feet)
Then PROTECT persons Downwind during DAY	0.6 kilometers (0.4 miles
Then PROTECT persons Downwind during NIGHT	1.1 kilometers (0.7 miles

Transport in bulk according to Annex II of MARPOL 73/78 and the IBC Code

No additional data.

15 Regulatory information

Safety, health and environmental regulations specific for the product in question

SA NATIONAL LEGISLATION

Hazardous Substances Act 15 of 1973 and Regulations.
Occupational Health and Safety Act 85 of 1993 and Regulations.

SA NATIONAL STANDARDS

SANS 10228 : 2006 : Identification and Classification of Dangerous Goods for Transport by Road and Rail.
SANS 10231 : 2018 : Transport of dangerous goods - Operational requirements for road vehicles.
SANS 10234 : 2008 : Globally Harmonized System of classification and labelling of chemicals (GHS).
SANS 11014 : 2010 : Safety Data Sheets for chemical Products.

REACH Regulation (EC) No 1907/2006

This product contains only components that have been either pre-registered, registered, are exempt from registration, are regarded as registered or are not subject to registration according to Regulation (EC) No. 1907/2006 (REACH)., The aforementioned indications of the REACH registration status are provided in good faith and believed to be accurate as of the effective date shown above. However, no warranty, express or implied, is given. It is the buyer´s/user´s responsibility to ensure that his/her understanding of the regulatory status of this product is correct.

Seveso III: Directive 2012/18/EU

Listed in Regulation: Not applicable

Clean Water Act Requirements

Nitric acid is designated as a hazardous substance under section 311(b)(2)(A) of the Federal Water Pollution Control Act and further regulated by the Clean Water Act Amendments of 1977 and 1978. These regulations apply to discharges of this substance. This designation includes any isomers and hydrates, as well as any solutions and mixtures containing this substance.

CERCLA Reportable Quantities

Persons in charge of vessels or facilities are required to notify the National Response Center (NRC) immediately, when there is a release of this designated hazardous substance, in an amount equal to or greater than its reportable quantity of 1000 lb or 45.4 kg. The toll free number of the NRC is (800) 424-8802. The rule for determining when notification is required is stated in 40 CFR 302.4 (section IV. D.3.b).

Releases of CERCLA hazardous substances are subject to the release reporting requirement of CERCLA section 103, codified at 40 CFR part 302, in addition to the requirements of 40 CFR part 355. Nitric acid is an extremely hazardous substance (EHS) subject to reporting requirements when stored in amounts in excess of its threshold planning quantity (TPQ) of 1,000 lbs.

FDA Requirements

Nitric acid is an indirect food additive for use only as a component of adhesives.

Chemical safety assessment

Performed for this substance: YES

16 Other information

Other information

Full text of H & P - Statements referred to under section 2

Hazard statements

H290	May be corrosive to metals.
H314	Causes severe skin burns and eye damage.
H331	Toxic if inhaled.

Precautionary statements

P234	Keep only in original container.
P260	Do not breathe dust/fume/gas/mist/vapours/spray.
P264	Wash thoroughly after handling.

P271 Use only outdoors or in a well-ventilated area.
P280 Wear protective gloves/protective clothing/eye protection/face protection.
P301+P330+P331 IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353 IF ON SKIN (or hair): Remove/Take off Immediately all contaminated clothing. Rinse SKIN with water/shower.
P304+P340 IF INHALED: Remove victim to fresh air and Keep at rest in a position comfortable for breathing.
P305+P351+P338 IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310 Immediately call a POISON CENTER or doctor/physician.
P321 Specific treatment (see P330+P351+P353 on this label).
P363 Wash contaminated clothing before reuse.
P390 Absorb spillage to prevent material damage.
P403+P233 Store in a well-ventilated place. Keep container tightly closed.
P405 Store locked up.
P406 Store in corrosive resistant container with a resistant inner liner.
P501 Dispose of contents and container in accordance with local, regional, national, international regulations.

Labelling REGULATION (EC) No 1272/2008
Signal Word
Danger
Pictograms Hazard to Human
GHS05 Corrosive hazard
GHS06 Toxicity hazard
Pictogram Hazard during Transport
Class 8 Corrosive substance

Training advice
Provide adequate information, instruction and training for operators.

Information sources
1. ECHA - European Chemicals Agency https://echa.europa.eu/de/registration-dossier/-/registered-dossier/15881/1
2. National Center for Biotechnology Information. PubChem Database. Nitric acid, CID=944, https://pubchem.ncbi.nlm.nih.gov/compound/944 (accessed on May 6, 2019)

Compiled by Aquatrade Water Treatment Chemicals (Pty) Ltd, R. van Rooyen, SHEQ Co-ordinator and E. Le Sar, Director.

MANUFACTURER/SUPPLIER DISCLAIMER:

IMPORTANT: This information is given without a warranty or guarantee. No suggestions for use are intended or shall be construed as a recommendation to infringe any existing patents or violate any national or local laws. Safe handling and use is the responsibility of the customer. Read the label before using this product. This information is true and accurate to the best of our knowledge.

Revision History

Revision	Date	Change
1.0	2019/05/07	Preparation of the safety data sheet according to SANS 11014:2010