API Pond Ammonia Test Solution #1

Mars Fishcare North America, Inc.

Chemwatch Hazard Alert Code: **2**

Chemwatch: **4650-13**
Version No: **7.1.1.1**
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Issue Date: **11/12/2018**
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L.GHS.USA.EN

SECTION 1 IDENTIFICATION

Product Identifier

Product name	API Pond Ammonia Test Solution #1
Synonyms	Solution ID# 3335A
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	Ammonia test solution for product 162 and 164M.

Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	**Mars Fishcare North America, Inc.**
Address	50 E. Hamilton Street, Chalfont PA 18914 United States
Telephone	215 822 8181
Fax	215 997 1290
Website	Not Available
Email	Not Available

Emergency phone number

Association / Organisation	**Mars Fishcare North America, Inc.**
Emergency telephone numbers	ChemTel: 1-800-255-3924
Other emergency telephone numbers	ChemTel: 1-813-248-0585

SECTION 2 HAZARD(S) IDENTIFICATION

Classification of the substance or mixture

NFPA 704 diamond

Note: The hazard category numbers found in GHS classification in section 2 of this SDSs are NOT to be used to fill in the NFPA 704 diamond. Blue = Health Red = Fire Yellow = Reactivity White = Special (Oxidizer or water reactive substances)

Classification	Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 4, Acute Toxicity (Inhalation) Category 4, Eye Irritation Category 2A, Acute Aquatic Hazard Category 3

Label elements

Hazard pictogram(s)	
SIGNAL WORD	**WARNING**

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Hazard statement(s)

H302	Harmful if swallowed.
H312	Harmful in contact with skin.
H332	Harmful if inhaled.
H319	Causes serious eye irritation.
H402	Harmful to aquatic life.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) General

P101	If medical advice is needed, have product container or label at hand.
P102	Keep out of reach of children.
P103	Read label before use.

Precautionary statement(s) Prevention

P271	Use only outdoors or in a well-ventilated area.
P261	Avoid breathing mist/vapours/spray.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P363	Wash contaminated clothing before reuse.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
P330	Rinse mouth.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
25322-68-3	>60	polyethylene glycol
14402-89-2	1-10	SOD NITROPRUSSIDE SOLUTION
54-21-7	1-10	SODIUM SALICYLATE-USP

SECTION 4 FIRST-AID MEASURES

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.

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	‣ Seek medical attention without delay; if pain persists or recurs seek medical attention. ‣ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ‣ Immediately remove all contaminated clothing, including footwear. ‣ Flush skin and hair with running water (and soap if available). ‣ Seek medical attention in event of irritation.
Inhalation	‣ If fumes or combustion products are inhaled remove from contaminated area. ‣ Lay patient down. Keep warm and rested. ‣ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ‣ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ‣ Transport to hospital, or doctor.
Ingestion	‣ **IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.** ‣ For advice, contact a Poisons Information Centre or a doctor. ‣ Urgent hospital treatment is likely to be needed. ‣ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ‣ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ‣ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. **Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:** ‣ **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. **NOTE:** Wear a protective glove when inducing vomiting by mechanical means.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

for salicylate intoxication:

- Pending gastric lavage, use emetics such as syrup of Ipecac or delay gastric emptying and absorption by swallowing a slurry of activated charcoal. Do not give ipecac after charcoal.
- Gastric lavage with water or perhaps sodium bicarbonate solution (3%-5%). Mild alkali delays salicylate absorption from the stomach and perhaps slightly from the duodenum.
- Saline catharsis with sodium or magnesium sulfate (15-30 gm in water).
- Take an immediate blood sample for an appraisal of the patient's acid-base status. A pH determination on an anaerobic sample of arterial blood is best. An analysis of the plasma salicylate concentration should be made at the same time. Laboratory controls are almost essential for the proper management of severe salicylism.
- In the presence of an established acidosis, alkali therapy is essential, but at least in an adult, alkali should be withheld until its need is demonstrated by chemical analysis. The intensity of treatment depends on the intensity of acidosis. In the presence of vomiting, intravenous sodium bicarbonate is the most satisfactory of all alkali therapy.
- Correct dehydration and hypoglycaemia (if present) by the intravenous administration of glucose in water or in isotonic saline. The administration of glucose may also serve to remedy ketosis which is often seen in poisoned children.
- Even in patients without hypoglycaemia, infusions of glucose adequate to produce distinct hyperglycaemia are recommended to prevent glucose depletion in the brain. This recommendation is based on impressive experimental data in animals.
- Renal function should be supported by correcting dehydration and incipient shock. Overhydration is not justified. An alkaline urine should be maintained by the administration of alkali if necessary with care to prevent a severe systemic alkalosis. As long as urine remains alkaline (pH above 7.5), administration of an osmotic diuretic such as mannitol or perhaps THAM is useful, but one must be careful to avoid hypokalaemia. Supplements of potassium chloride should be included in parenteral fluids.
- Small doses of barbiturates, diazepam, paraldehyde, or perhaps other sedatives (but probably not morphine) may be required to suppress extreme restlessness and convulsions.
- For hyperpyrexia, use sponge baths.

The presence of petechiae or other signs of haemorrhagic tendency calls for a large Vitamin K dose and perhaps ascorbic acid. Minor transfusions may be necessary since bleeding in salicylism is not always due to a prothrombin effect.

- Haemodialysis and haemoperfusion have proved useful in salicylate poisoning, as have peritoneal dialysis and exchange transfusions, but alkaline diuretic therapy is probably sufficient except in fulminating cases.

[*GOSSELIN, et.al.: Clinical Toxicology of Commercial Products*]

The mechanism of the toxic effect involves metabolic acidosis, respiratory alkalosis, hypoglycaemia, and potassium depletion. Salicylate poisoning is characterised by extreme acid-base disturbances, electrolyte disturbances and decreased levels of consciousness. There are differences between acute and chronic toxicity and a varying clinical picture which is dependent on the age of the patient and their kidney function. The major feature of poisoning is

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metabolic acidosis due to "uncoupling of oxidative phosphorylation" which produces an increased metabolic rate, increased oxygen consumption, increased formation of carbon dioxide, increased heat production and increased utilisation of glucose. Direct stimulation of the respiratory centre leads to hyperventilation and respiratory alkalosis. This leads to compensatory increased renal excretion of bicarbonate which contributes to the metabolic acidosis which may coexist or develop subsequently. Hypoglycaemia may occur as a result of increased glucose demand, increased rates of tissue glycolysis, and impaired rate of glucose synthesis. **NOTE:** Tissue glucose levels may be lower than plasma levels. Hyperglycaemia may occur due to increased glycogenolysis. Potassium depletion occurs as a result of increased renal excretion as well as intracellular movement of potassium.

Salicylates competitively inhibit vitamin K dependent synthesis of factors II, VII, IX, X and in addition, may produce a mild dose dependent hepatitis. Salicylates are bound to albumin. The extent of protein binding is concentration dependent (and falls with higher blood levels). This, and the effects of acidosis, decreasing ionisation, means that the volume of distribution increases markedly in overdose as does CNS penetration. The extent of protein binding (50-80%) and the rate of metabolism are concentration dependent. Hepatic clearance has zero order kinetics and thus the therapeutic half-life of 2-4.5 hours but the half-life in overdose is 18-36 hours. Renal excretion is the most important route in overdose. Thus when the salicylate concentrations are in the toxic range there is increased tissue distribution and impaired clearance of the drug.

HyperTox 3.0 http://www.ozemail.com.au/-ouad/SALI0001.HTA
Treat symptomatically.
For cyanide intoxication (and for certain nitriles which produce cyanide ion)

- Signs symptoms of acute cyanide poisoning reflect cellular hypoxia and are often non-specific.
- Cyanosis may be a late finding.
- *A bradycardic*, hypertensive and tachypneic patient suggests poisoning especially if CNS and cardiovascular depression subsequently occurs.
- Immediate attention should be directed towards assisted ventilation, administration of 100% oxygen, insertion of intravenous lines and institution of cardiac monitoring.
- Obtain an arterial blood gas immediately and correct any severe metabolic acidosis (pH below 7.15).
- Mildly symptomatic patients generally require supportive care alone. Nitrites should not be given indiscriminately - in all cases of moderate to severe poisoning, they should be given in conjunction with thiosulfate. As a temporizing measure supply amyl nitrite perles (0.2ml inhaled 30 seconds every minute) until intravenous lines for sodium nitrite are established. 10 ml of a 3% solution is administered over 4 minutes to produce 20% methaemoglobin in adults. Follow directly with 50 ml of 25% sodium thiosulfate, at the same rate, IV. If symptoms reappear or persist within 1/2-1 hour, repeat nitrite and thiosulfate at 50% of initial dose. As the mode of action involves the metabolic conversion of the thiosulfate to thiocyanate, renal failure may enhance thiocyanate toxicity.
- Methylene blue is not an antidote. [Ellenhorn and Barceloux: Medical Toxicology]

If amyl nitrite intervention is employed then Medical Treatment Kits should contain the following:

- One box containing one dozen amyl nitrite ampoules
- Two sterile ampoules of sodium nitrite solution (10 mL of a 3% solution in each)
- Two sterile ampoules of sodium thiosulfate solution (50 mL of a 25% solution in each)
- One 10 mL sterile syringe. One 50 mL sterile syringe. Two sterile intravenous needles. One tourniquet.
- One dozen gauze pads.
- Latex gloves
- A "Biohazard" bag for disposal of bloody/contaminated equipment.
- A set of cyanide instructions on first aid and medical treatment.

- Notes on the use of amyl nitrite:-

- AN is highly volatile and flammable - do not smoke or use around a source of ignition.
- If treating patient in a windy or draughty area provide some shelter or protection (shirt, wall, drum, cupped hand etc.) to prevent amyl nitrite vapour from being blown away. Keep ampoule upwind from the nose, the objective is to get amyl nitrite into the patients lungs.
- Rescuers should avoid AN inhalation to avoid becoming dizzy and losing competence.
- Lay the patient down. Since AN dilates blood vessels and lowers blood pressure, lying down will help keep patient conscious.
- **DO NOT** overuse - excessive use might put the patient into shock. Experience at DuPont plants has not shown any serious after-effects from treatment with amyl nitrite.

ADDITIONAL NOTES:

- Major medical treatment procedures may vary e.g. US (FDA method as recommended by DuPont) uses amyl nitrite as a methaemoglobin generator, followed by treatment with sodium nitrite and then sodium thiosulfate.

MODES OF ACTION: Amyl nitrite (AN) reacts with haemoglobin (HB) to form about 5% methaemoglobin (MHB). Sodium nitrite (NaNO2) reacts with haemoglobin to form approximately 20-30% methaemoglobin. Methaemoglobin attracts cyanide ions (CN) from tissue and binds with them to become cyanmethaemoglobin (CNMHB). Sodium thiosulfate (Na2S2O3) converts cyanmethaemoglobin to thiocyanate (HSCN) which is excreted by the kidneys.
i.e. AN + HB = MHB NaNO2 + HB = MHB CN + MHB = CNMHB Na2S2O3 + CNMHB + O2 = HSCN

- The administration of the antidote salts is intravenous in normal saline, Ringers lactate or other available IV fluid.
- European practice may use 4-dimethylaminophenol (DMAP) as a methaemoglobin generator. Also hydroxycobalamin (Vitamin B12a) is used. Hydroxycobalamin works by reacting with cyanide to form cyanocobalamin (Vitamin B12) which is excreted in the urine.
- European and Australian NOHSC (ASCC) propose dicobalt edetate (Kelocyanor) as antidote. This acts by chelating cyanide to form stable cobalticyanide, which is excreted in the urine. In all cases hyperbaric therapy may increase the efficiency of a cyanide antidote kit.

for non-steroidal anti-inflammatories (NSAIDs)

- Symptoms following acute NSAIDs overdoses are usually limited to lethargy, drowsiness, nausea, vomiting, and epigastric pain, which are generally reversible with supportive care. Gastrointestinal bleeding can occur. Hypertension, acute renal failure, respiratory depression, and coma may occur, but are rare. Anaphylactoid reactions have been reported with therapeutic ingestion of NSAIDs, and may occur following an overdose.
- Patients should be managed by symptomatic and supportive care following a NSAIDs overdose.
- There are no specific antidotes.
- Emesis and/or activated charcoal (60 to 100 grams in adults, 1 to 2 g/kg in children), and/or osmotic cathartic may be indicated in patients seen within 4 hours of ingestion with symptoms or following a large overdose (5 to 10 times the usual dose).
- Forced diuresis, alkalinisation of urine, hemodialysis, or haemoperfusion may not be useful due to high protein binding.

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SECTION 5 FIRE-FIGHTING MEASURES

Extinguishing media

- Water spray or fog.
- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	‣ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Special protective equipment and precautions for fire-fighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Use water delivered as a fine spray to control fire and cool adjacent area. ‣ Avoid spraying water onto liquid pools. ‣ **DO NOT** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	‣ Combustible. ‣ Slight fire hazard when exposed to heat or flame. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). ‣ May emit acrid smoke. ‣ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ‣ Remove all ignition sources. ‣ Clean up all spills immediately. ‣ Avoid breathing vapours and contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Contain and absorb spill with sand, earth, inert material or vermiculite. ‣ Wipe up. ‣ Place in a suitable, labelled container for waste disposal.
Major Spills	Environmental hazard - contain spillage. Moderate hazard. ‣ Clear area of personnel and move upwind. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ No smoking, naked lights or ignition sources. ‣ Increase ventilation. ‣ Stop leak if safe to do so. ‣ Contain spill with sand, earth or vermiculite. ‣ Collect recoverable product into labelled containers for recycling. ‣ Absorb remaining product with sand, earth or vermiculite. ‣ Collect solid residues and seal in labelled drums for disposal. ‣ Wash area and prevent runoff into drains. ‣ If contamination of drains or waterways occurs, advise emergency services.

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Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	‣ **DO NOT USE** brass or copper containers / stirrers ‣ **DO NOT** allow clothing wet with material to stay in contact with skin ‣ Avoid all personal contact, including inhalation. ‣ Wear protective clothing when risk of exposure occurs. ‣ Use in a well-ventilated area. ‣ Prevent concentration in hollows and sumps. ‣ **DO NOT** enter confined spaces until atmosphere has been checked. ‣ Avoid smoking, naked lights or ignition sources. ‣ Avoid contact with incompatible materials. ‣ When handling, **DO NOT** eat, drink or smoke. ‣ Keep containers securely sealed when not in use. ‣ Avoid physical damage to containers. ‣ Always wash hands with soap and water after handling. ‣ Work clothes should be laundered separately. ‣ Use good occupational work practice. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS. ‣ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	‣ Store in original containers. ‣ Keep containers securely sealed. ‣ No smoking, naked lights or ignition sources. ‣ Store in a cool, dry, well-ventilated area. ‣ Store away from incompatible materials and foodstuff containers. ‣ Protect containers against physical damage and check regularly for leaks. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Glass container is suitable for laboratory quantities ‣ Metal can or drum ‣ Packaging as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	‣ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates. Several members of the family described as metal cyano complexes are endothermic and tend towards explosive instability; most are capable of violent oxidation under appropriate circumstances. BRETHERICKS HANDBOOK OF REACTIVE CHEMICAL HAZARDS, 4th Edition ferricyanide: ‣ mixtures with water, acids,or alcohols may slowly decompose producing hydrocyanic acid ‣ reacts explosively with strong oxidisers, ammonia chromium trioxide, chromic acid, chromic anhydride, sodium nitrite ‣ reacts violently with copper(II) nitrate, trihydrate. ‣ Avoid reaction with oxidising agents

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X — Must not be stored together
0 — May be stored together with specific preventions
+ — May be stored together

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US ACGIH Threshold Limit Values (TLV)	sodium nitroprusside	Hydrogen cyanide and cyanide salts, as CN - Cyanide salts	Not Available	Not Available	5 mg/m3	TLV® Basis: URT irr; headache; nausea; thyroid eff
US ACGIH Threshold Limit Values (TLV)	sodium nitroprusside	Iron salts, soluble, as Fe	1 mg/m3	Not Available	Not Available	TLV® Basis: URT & skin irr

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US OSHA Permissible Exposure Levels (PELs) - Table Z1	sodium nitroprusside	Cyanides (as CN)	5 mg/m3	Not Available	Not Available	(4) Varies with compound.

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
polyethylene glycol	Polyethylene glycol	30 mg/m3	1,300 mg/m3	7,700 mg/m3
SOD NITROPRUSSIDE SOLUTION	Sodium nitroferricyanide	0.21 mg/m3	2.3 mg/m3	14 mg/m3
SODIUM SALICYLATE-USP	Sodium salicylate; (Salicylic acid, sodium salt)	7.1 mg/m3	78 mg/m3	470 mg/m3

Ingredient	Original IDLH	Revised IDLH
polyethylene glycol	Not Available	Not Available
SOD NITROPRUSSIDE SOLUTION	25 mg/m3	Not Available
SODIUM SALICYLATE-USP	Not Available	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls

Enclosed local exhaust ventilation is required at points of dust, fume or vapour generation.

HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours.

Barrier protection or laminar flow cabinets should be considered for laboratory scale handling.

A fume hood or vented balance enclosure is recommended for weighing/ transferring quantities exceeding 500 mg.

When handling quantities up to 500 gram in either a standard laboratory with general dilution ventilation (e.g. 6-12 air changes per hour) is preferred. Quantities up to 1 kilogram may require a designated laboratory using fume hood, biological safety cabinet, or approved vented enclosures. Quantities exceeding 1 kilogram should be handled in a designated laboratory or containment laboratory using appropriate barrier/ containment technology.

Manufacturing and pilot plant operations require barrier/ containment and direct coupling technologies.

Barrier/ containment technology and direct coupling (totally enclosed processes that create a barrier between the equipment and the room) typically use double or split butterfly valves and hybrid unidirectional airflow/ local exhaust ventilation solutions (e.g. powder containment booths). Glove bags, isolator glove box systems are optional. HEPA filtration of exhaust from dry product handling areas is required.

Fume-hoods and other open-face containment devices are acceptable when face velocities of at least 1 m/s (200 feet/minute) are achieved. Partitions, barriers, and other partial containment technologies are required to prevent migration of the material to uncontrolled areas. For non-routine emergencies maximum local and general exhaust are necessary. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, etc. evaporating from tank (in still air)	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe.

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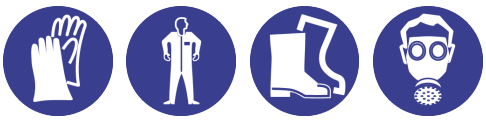
	Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used. The need for respiratory protection should also be assessed where incidental or accidental exposure is anticipated: Dependent on levels of contamination, PAPR, full face air purifying devices with P2 or P3 filters or air supplied respirators should be evaluated. The following protective devices are recommended where exposures exceed the recommended exposure control guidelines by factors of: 10; high efficiency particulate (HEPA) filters or cartridges 10-25; loose-fitting (Tyvek or helmet type) HEPA powered-air purifying respirator. 25-50; a full face-piece negative pressure respirator with HEPA filters 50-100; tight-fitting, full face-piece HEPA PAPR 100-1000; a hood-shroud HEPA PAPR or full face-piece supplied air respirator operated in pressure demand or other positive pressure mode.
Personal protection	
Eye and face protection	▸ Safety glasses. ▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	**NOTE:** ▸ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and.has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades

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	For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower.

Respiratory protection

Type A-P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Reddish-orange liquid with a mild odour; mixes with water.

Physical state	Liquid	**Relative density (Water = 1)**	1.152
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	8.3	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Applicable	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 STABILITY AND REACTIVITY

Continued...

Reactivity	See section 7
Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful. The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation of vapours, fumes or aerosols, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress. The very low volatility of polyethylene glycols (PEGs) make inhalation exposure unlikely other than in the form of mist which may be formed by violent agitation or at high temperatures. No toxic effects have been reported through inhalation. [AIHA Journal] Polyglycols at 200 mg/l were easily inhaled with no adverse effects Inhalation hazard is increased at higher temperatures.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Although the polyethylene glycols (PEGs) are extremely low in acute oral toxicity, the LD50s decrease as the molecular weights increase. PEGs of average molecular weights 4000 to 6000 are not absorbed from the rat intestine within 5 hours whilst the lower molecular weight variety (1000 to 1540) are absorbed to only a slight extent Large oral doses of salicylates may cause mild burning pain in the throat, stomach and usually prompt vomiting. Several hours may elapse before the development of deep and rapid breathing, lassitude, anorexia, nausea, vomiting, thirst and occasional diarrhoea. Common derivatives of salicylic acid produce substantially the same toxic syndrome, ("salicylism"). Major signs and symptoms arise from stimulation and terminal depression of the central nervous system. Stimulation produces vomiting, hyperpnea (abnormal increase in rate and depth of respiration), headache, tinnitus (ringing in the ears) confusion, bizarre behaviour or mania, generalised convulsions. Death is due to respiratory failure or cardiovascular collapse. Severe sensory disturbances such as deafness and dimness of vision are common. Less common features include sweating, skin eruptions, gastrointestinal and other hemorrhages, renal failure and pancreatitis. A tendency to bleed may be manifest by blood in the vomitus (haematemesis), bloody stools (melena) or purplish-red spots (petechiae) on the skin. Many of the toxic effects detailed here are due to or aggravated by severe disturbance of acid-base balance with the chief cause being prolonged hyperventilation from central stimulation. An assessment of acute salicylate intoxication based on dose suggests; 500 mg/kg: Potentially lethal Non-steroidal anti-inflammatory drugs (NSAID) can cause serious gastrointestinal (GI) adverse events including inflammation, bleeding, ulceration, and perforation of the stomach, small intestine, or large intestine, which can be fatal. These serious adverse events can occur at any time, with or without warning symptoms, in patients treated with NSAIDs. Only one in five patients, who develop a serious upper GI adverse event on NSAID therapy, is symptomatic. Upper GI ulcers, gross bleeding, or perforation caused by NSAIDs occur in approximately 1% of patients treated for 3-6 months, and in about 2-4% of patients treated for one year. These trends continue with longer duration of use, increasing the likelihood of developing a serious GI event at some time during the course of therapy. Anaphylactoid (allergic) reactions may occur. This typically occurs in asthmatic patients who experience rhinitis with or without nasal polyps, or who exhibit severe, potentially fatal bronchospasm after taking aspirin or other NSAIDs. NSAIDs, can cause serious skin adverse events such as exfoliative dermatitis, Stevens-Johnson Syndrome (SJS), and toxic epidermal necrolysis (TEN), which can be fatal Non-steroidal anti-inflammatory drug (NSAID) overdose may produce nausea, vomiting, indigestion and epigastric pain. Central nervous system effects may include drowsiness, dizziness, mental confusion, disorientation, lethargy, paraesthesia, numbness, intense headache, blurred vision, tinnitus, decreased auditory acuity, ataxia, muscle twitching, convulsions, stupor and coma. Other reported effects include sweating, oliguria or anuria, tachycardia and hypo- or hypertension. Renal damage may also occur. A number of materials such as cyanamide, calcium cyanamide, cyanates, isocyanates, isonitrile, thiocyanates, ferricyanide and ferrocyanide, other complex metallocyanides and cyanoacetates do not exhibit the same toxicology as cyanides and nitriles. Complex cyanides are compounds in which the cyanide anion is incorporated into a complex or complexes; these compounds are different in chemical and toxicologic properties from simple cyanides. In solution, the stability of the cyanide complex varies with the type of cation and the complex that it forms. Some of these are dissociable in weak acids to give free cyanide and a cation, while other complexes require much stronger acidic conditions for dissociation. The least-stable complex metallocyanides include [Zn(CN)4]2-, Cd(CN)3-, and [Cd(CN)4]2-; moderately stable complexes include Cu(CN)2-, [Cu(CN)3]2-, [Ni(CN)4]2-, and Ag(CN)2-; and the most stable complexes include [Fe(CN)6]4-and [Co(CN)6]4-**.** The toxicity of complex cyanides is usually related to their ability to release cyanide ions in solution, which

Continued...

	then enter into an equilibrium with HCN; relatively small fluctuations in pH significantly affect their biocidal properties.
Skin Contact	Skin contact with the material may be harmful; systemic effects may result following absorption. The material is not thought to be a skin irritant (i.e. is unlikely to produce irritant dermatitis as described in EC Directives using animal models). Temporary discomfort, however, may result from prolonged dermal exposures. Good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. The polyethylene glycols (PEGs)may be absorbed by the skin but no toxic effects have been noted and sensitisation does not occur. This material may increase the absorption activity or toxicity of other ingredients in a mixture. (Source: Genium) Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals. Repeated or prolonged eye contact may cause inflammation (similar to windburn) characterised by a temporary redness of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. On eye contact the polyethylene glycols will cause slight transient pain and conjunctival irritation although no permanent damage. The effects are described as similar to those produced by mild soap
Chronic	Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. Polyethylene glycols appear to act as slow-acting parasympathomimetic-like compounds. When given intravenously they may increase the tendency of blood to clot and if given rapidly may cause cell clotting and death from embolism. Ethylene glycol is not believed to be a metabolite NSAIDs may cause an increased risk of serious cardiovascular thrombotic events, myocardial infarction, and stroke, which can be fatal. NSAIDs cause an increased risk of serious gastrointestinal adverse events including bleeding, ulceration, and perforation of the stomach or intestines, which can be fatal. These events can occur at any time during use and without warning symptoms. Prolonged treatment with non-steroidal anti-inflammatory drugs (NSAIDs) has been associated with gastrointestinal irritation, erosion, ulceration, perforation, frank or occult bleeding, diarrhoea, constipation, and blood in the vomit or stool. Kidney damage may result in haematuria (blood in the urine), pyuria (white blood cells in the urine), proteinuria (protein in the urine), urinary casts (cylindrical aggregations of particles that form in the distal nephron, dislodge, and pass into the urine), nocturia (excessive night time urination) , polyuria (production of large volumes of pale urea), dysuria (painful or difficult urination), oliguria (production of abnormally small volumes of urea), or anuria (inability to urinate), renal insufficiency (insufficient excretion of wastes by the kidney), nephrosis and nephrotic syndrome (conditions characterized by oedema and large amounts of protein in the urine and usually increased blood cholesterol), and glomerular and interstitial nephritis. Liver effects, although rare, include jaundice, hepatocellular injury, possible fatal hepatitis, and abnormal liver function tests. Clinical trials of several COX-2 selective and nonselective NSAIDS of up to three years duration have shown an increased risk of serious cardiovascular (CV) thrombotic events, myocardial infarction, and stroke, which can be fatal. All NSAIDs, both COX-2 selective and nonselective, may have a similar risk. NSAIDs, can lead to onset of new hypertension or worsening of preexisting hypertension, either of which may contribute to the increased incidence of CV events. Long-term administration of NSAIDs has resulted in renal papillary necrosis and other renal injury. Acute interstitial nephritis with haematuria, proteinuria, and occasionally nephritic syndrome have been reported. Anaphylactoid reactions may occur in patients with known prior exposure to other NSAIDs. NSAIDs have produced ocular changes in animals and there have been reports of adverse eye findings in patients. Anaemia is sometimes seen in patients receiving NSAIDs.. This may be due to fluid retention, occult or gross GI blood loss, or an incompletely described effect upon erythropoiesis. In rat studies with NSAIDs, as with other drugs known to inhibit prostaglandin synthesis, an increased incidence of dystocia, delayed parturition, and decreased pup survival occurred. Because of the known effects of NSAIDs on the foetal cardiovascular system (closure of ductus arteriosus), use during pregnancy (particularly late pregnancy) should be avoided.. NSAIDs inhibit enzymes collectively described as "COXs". In the course of the early search for a specific inhibitor of the negative effects of prostaglandins which spared the positive effects, it was discovered that prostaglandins could indeed

Continued...

be separated into two general classes which could loosely be regarded as "good prostaglandins" and "bad prostaglandins", according to the structure of a particular enzyme involved in their biosynthesis, cyclooxygenase (COX).
Prostaglandins whose synthesis involves the cyclooxygenase-I enzyme, or COX-1, are responsible for maintenance and protection of the gastrointestinal tract, while prostaglandins whose synthesis involves the cyclooxygenase-II enzyme, or COX-2, are responsible for inflammation and pain.

The existing non-steroidal anti-inflammatory drugs (NSAIDs) differ in their relative specificities for COX-2 and COX-1
There has been much concern about the possibility of increased risk for heart attack and stroke in users of NSAID drugs, particularly COX-2 selective NSAIDs. The cardiovascular risks associated with NSAIDs are controversial, with apparently contradictory data produced from different clinical trials and in published meta-analyses. Cardiovascular risk of COX-2 specific inhibitors is not surprising since prostaglandins are involved in regulation of blood pressure by the kidneys.
COX-inhibitors produce blood dyscrasias (abnormal conditions of the blood), and interfere with platelet function.
Phototoxic or photoallergic skin reactions may also occur. Anaphylactoid reactions characterised by maculopapular rash, urticaria, pruritus, bronchospasm, and syncope have been described. Other effects include oedema, metabolic acidosis, hyperkalaemia, azotemia, cystitis and urinary tract infections, visual and hearing disturbances, conjunctivitis, corneal deposits, retinal degeneration, ear pain and occasionally, deafness. Idiosyncratic responses include asthma, allergic interstitial nephritis, hypersensitivity hepatitis, aplastic anaemia and exfoliative dermatitis.

Non-steroidal anti-inflammatory drugs with an inhibitory effect on prostaglandin synthesis, when given during the latter stages of pregnancy, cause premature closure of the foetal ductus arteriosus (1). When given at term they prolong labour and delay parturition. Evidence (1) from animal experimental studies, clinical investigations in humans, and epidemiological studies supports the hypothesis that NSAIDs are chemopreventative agents against colon cancer. This is corroborated by knowledge of the underlying pathophysiological mechanisms and the effects of arachidonic metabolites, i.e prostaglandins, on the carcinogenic process and the influence of cyclooxygenase (COX) inhibitors such as NSAIDs on these metabolites. 1. Berkel et al; Epidemiol Rev., Vol 18, No. 2, 1996

Because of the known effects of NSAIDs drugs on the foetal cardiovascular system (closure of ductus arteriosus), use during pregnancy (particularly late pregnancy) should be avoided. In rat studies with NSAIDs, as with other drugs known to inhibit prostaglandin synthesis, an increased incidence of dystocia, delayed parturition, and decreased pup survival occurred
Aspirin and NSAIDs may cause anaphylactic or anaphylactoid reactions. Constitutively-expressed cyclooxygenase (COX-1) inhibition is likely to be responsible for the cross-reactions and side effects associated with these drugs, as well as the anaphylactoid reactions sometimes seen in aspirin-sensitive respiratory disease. Though anaphylactic and anaphylactoid reactions may be clinically indistinguishable, they involve different mechanisms. Anaphylactic reactions are due to immediate hypersensitivity involving cross-linking of drug-specific IgE. Regardless of COX selectivity pattern, NSAIDs may function as haptens capable of inducing allergic sensitization. Unlike anaphylaxis, anaphylactoid reactions are most likely related to inhibition of COX-1 by NSAIDS. Thus, an anaphylactoid reaction caused by a particular COX-1 inhibiting NSAID will occur with a chemically unrelated NSAID which also inhibits COX-1 enzymes. Selective COX-2 inhibitors appear to be safe in patients with a history of NSAID-related anaphylactoid reactions but can function as haptens, with resulting sensitisation and anaphylaxis upon next exposure. Eva A Berkes Clinical Reviews in Allergy and Immunology 24, pp 137-147 2003.

COX-2 inhibitors reduce inflammation (and pain) while minimising gastrointestinal adverse drug reactions (e.g. stomach ulcers) that are common with non-selective NSAIDs. COX-1 is involved in synthesis of prostaglandins and thromboxane, but COX-2 is only involved in the synthesis of prostaglandin. Therefore, inhibition of COX-2 inhibits only prostaglandin synthesis without affecting thromboxane and thus has no effect on platelet aggregation or blood clotting.

Chronic abuse of analgesics has been associated with nephropathy. Patients invariably have a history of regular ingestion of substantial or excessive doses over a period of years. In mild cases the condition is reversible. The initial renal lesion is papillary necrosis proceeding to secondary atrophic changes in the renal cortex body. An abnormally high incidence of transitional cell carcinoma of the renal pelvis and bladders has been reported in patients with analgesic nephropathy.
Mild chronic salicylate intoxication, or "salicylism", may occur after repeated exposures to large doses. Symptoms include dizziness, tinnitus, deafness, sweating, nausea and vomiting, headache and mental confusion. Symptoms of more severe intoxication include hyperventilation, fever, restlessness, ketosis, and respiratory alkalosis and metabolic acidosis. Depression of the central nervous system may lead to coma, cardiovascular collapse and respiratory failure.
Chronic exposure to the salicylates (o-hydroxybenzoates) may produce metabolic and central system disturbances or damage to the kidneys. Persons with pre-existing skin disorders, eye problems or impaired kidney function may be more susceptible to the effects of these substances. Certain individuals (atopics), notably asthmatics, exhibit significant hyper-sensitivity to salicylic acid derivatives. Reactions include urticaria and other skin eruptions, rhinitis and severe (even fatal) bronchospasm and dyspnea. Chronic exposure to the p-hydroxybenzoates (parabens) is associated with hypersensitivity reactions following application of these to the skin. Hypersensitivity reactions have also been reported following parenteral or oral administration. Cross-sensitivity occurs between the p-hydroxybenzoates Hypersensitivity reactions may include by acute bronchospasm, hives (urticaria), deep dermal wheals (angioneurotic oedema), running nose (rhinitis) and blurred vision. Anaphylactic shock and skin rash (non- thrombocytopenic purpura) may also occur. Any individual may be predisposed to such anti-body mediated reaction if other chemical agents have caused prior sensitisation (cross-sensitivity).
Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

API Pond Ammonia Test Solution #1

TOXICITY	IRRITATION
Not Available	Not Available

Continued...

polyethylene glycol	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit): 500mg/24h - mild.
	Oral (rat) LD50: 600 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): 500mg/24h - mild.
		Skin: no adverse effect observed (not irritating)[1]
SOD NITROPRUSSIDE SOLUTION	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: 69.8 mg/kg[2]	Not Available
SODIUM SALICYLATE-USP	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: 930 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

POLYETHYLENE GLYCOL

for polyethylene glycols
Pure polyethylene glycols have essentially similar toxicity, with toxicity being inverse to molecular weights. Absorption from the gastrointestinal tract decreases with increasing molecular weight
The G.I. absorption of a series of polyethylene glycols has been studied. Polyethylene glycols having average molecular weights of 4000 and 6000 showed no absorption from the rat intestine over a five-hour period, while polyethylene glycols of 1000 and 1540 molecular weights showed a slight absorption amounting to less than 2% of the total dose during the same period. When 1 g doses of polyethylene glycols of molecular weight 1000 (PEG 1000) and 6000 (PEG 6000) were given intravenously to six human subjects, 85% of PEG 1000 and 96% of PEG 6000 were excreted in the urine in 12 hours. When these two same materials in 10 g doses were given orally to five human subjects, none of the PEG 6000 was found in the urine in the following 24 hours, whereas about 8% of PEG 1000 administered was found to excrete in urine within 24 hours When PEG 400 was given intravenously to three human subjects, an average of 77% recovery of this material was found in the urine in 12 hours. However, when the same substance was given orally to the same three human subjects, a recovery of between 40 and 50% of the dose was determined in the urine in the course of the following 24 hours. Single oral doses of PEG 400 were incompletely recovered from urine and faeces of rabbits even when collection of excreta was continued as long as four days following the dose. Evidence from all these and other studies indicate that ethylene glycol is not formed as a metabolite of PEG 400
Prolonged skin contact of PEG 1500 and 4000 upon the skin of rabbits in dosages of 10 g/kg bw showed no deleterious effects on internal organs and little, if any, of the materials was absorbed through the skin.
Although early reports indicated that skin sensitization was observed among a few human subjects and in guinea pigs tested with certain polyethylene glycols, later studies showed that currently produced materials were without irritating or sensitizing properties. However, recent report (Fischer, 1978) demonstrated that four patients showed allergic reactions to lower molecular weight liquid polyethylene glycols in topical medications. Two had immediate urticarial reactions to PEG 400. Two other patients had delayed allergic eczematous reactions, one to PEG 200, and one to PEG 300.
Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.
Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture .
On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However,
their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.
Allergic Contact Dermatitis––Formation, Structural Requirements,and Reactivity of Skin Sensitizers.
Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69
Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.
PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations.
Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between

Continued...

	ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used Safety Evaluation of Polyethyene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology http://doi.org/10.5487/TR.2015.31.2.105 The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. for molecular weights (200-8000) * Oral (rat) LD50: 31000->50000 mg/kg Oral (mice) LD50: 38000->50000 mg/kg Oral (g.pig) LD50: 17000->50000 mg/kg Oral (rabbit) LD50: 14000->50000 mg/kg * AIHA WEEL Guides Intraperitoneal (mice) LD50: 3100-12900 mg/kg
SODIUM SALICYLATE-USP	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production. Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

Acute Toxicity	✔	**Carcinogenicity**	✘
Skin Irritation/Corrosion	✘	**Reproductivity**	✘
Serious Eye Damage/Irritation	✔	**STOT - Single Exposure**	✘
Respiratory or Skin sensitisation	✘	**STOT - Repeated Exposure**	✘
Mutagenicity	✘	**Aspiration Hazard**	✘

Legend: ✘ *– Data either not available or does not fill the criteria for classification*
✔ *– Data available to make classification*

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

API Pond Ammonia Test Solution #1

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
Not Available	Not Available	Not Available	Not Available	Not Available

polyethylene glycol

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	20-mg/L	2
EC50	72	Algae or other aquatic plants	15.915mg/L	2

SOD NITROPRUSSIDE SOLUTION

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
Not Available	Not Available	Not Available	Not Available	Not Available

SODIUM SALICYLATE-USP

ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	1-370mg/L	2
EC50	72	Algae or other aquatic plants	48.29mg/L	2
EC0	24	Crustacea	80mg/L	4
NOEC	504	Crustacea	4.126mg/L	2

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity*

Continued...

	Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
polyethylene glycol	LOW	LOW
SODIUM SALICYLATE-USP	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
polyethylene glycol	LOW (LogKOW = -1.1996)
SODIUM SALICYLATE-USP	LOW (LogKOW = 2.2447)

Mobility in soil

Ingredient	Mobility
polyethylene glycol	HIGH (KOC = 1)
SODIUM SALICYLATE-USP	LOW (KOC = 23.96)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO

Land transport (DOT): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Continued...

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Transport in bulk according to Annex II of MARPOL and the IBC code

Not Applicable

SECTION 15 REGULATORY INFORMATION

Safety, health and environmental regulations / legislation specific for the substance or mixture

POLYETHYLENE GLYCOL(25322-68-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

GESAMP/EHS Composite List - GESAMP Hazard Profiles
IMO IBC Code Chapter 17: Summary of minimum requirements
IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances
US AIHA Workplace Environmental Exposure Levels (WEELs)
US DOE Temporary Emergency Exposure Limits (TEELs)
US DOT Coast Guard Bulk Hazardous Materials - List of Flammable and Combustible Bulk Liquid Cargoes
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US Toxicology Excellence for Risk Assessment (TERA) Workplace Environmental Exposure Levels (WEEL)
US TSCA Chemical Substance Inventory - Interim List of Active Substances

SOD NITROPRUSSIDE SOLUTION(14402-89-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations
US - Alaska Limits for Air Contaminants
US - California Permissible Exposure Limits for Chemical Contaminants
US - Hawaii Air Contaminant Limits
US - Idaho - Limits for Air Contaminants
US - Idaho Toxic Air Pollutants Non- Carcinogenic Increments - Occupational Exposure Limits
US - Michigan Exposure Limits for Air Contaminants
US - Minnesota Permissible Exposure Limits (PELs)
US - Oregon Permissible Exposure Limits (Z-1)
US - Pennsylvania - Hazardous Substance List
US - Rhode Island Hazardous Substance List
US - Tennessee Occupational Exposure Limits - Limits For Air Contaminants
US - Vermont Permissible Exposure Limits Table Z-1-A Final Rule Limits for Air Contaminants
US - Vermont Permissible Exposure Limits Table Z-1-A Transitional Limits for Air Contaminants
US - Washington Permissible exposure limits of air contaminants
US - Wyoming Toxic and Hazardous Substances Table Z1 Limits for Air Contaminants
US ACGIH Threshold Limit Values (Spanish)
US ACGIH Threshold Limit Values (TLV)
US Clean Air Act - Hazardous Air Pollutants
US CWA (Clean Water Act) - Toxic Pollutants
US Department of Transportation (DOT) Marine Pollutants - Appendix B
US Department of Transportation (DOT), Hazardous Material Table
US DOE Temporary Emergency Exposure Limits (TEELs)
US EPCRA Section 313 Chemical List
US NIOSH Recommended Exposure Limits (RELs) (Spanish)
US OSHA Permissible Exposure Levels (PELs) - Table Z1
US OSHA Permissible Exposure Limits - Annotated Table Z-1 (Spanish)
US Postal Service (USPS) Hazardous Materials Table: Postal Service Mailability Guide
US Postal Service (USPS) Numerical Listing of Proper Shipping Names by Identification (ID) Number
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

SODIUM SALICYLATE-USP(54-21-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances
US DOE Temporary Emergency Exposure Limits (TEELs)
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

Federal Regulations

Superfund Amendments and Reauthorization Act of 1986 (SARA)

SECTION 311/312 HAZARD CATEGORIES

Flammable (Gases, Aerosols, Liquids, or Solids)	No
Gas under pressure	No
Explosive	No
Self-heating	No
Pyrophoric (Liquid or Solid)	No
Pyrophoric Gas	No
Corrosive to metal	No
Oxidizer (Liquid, Solid or Gas)	No
Organic Peroxide	No

Continued...

Self-reactive	No
In contact with water emits flammable gas	No
Combustible Dust	No
Carcinogenicity	No
Acute toxicity (any route of exposure)	Yes
Reproductive toxicity	No
Skin Corrosion or Irritation	No
Respiratory or Skin Sensitization	No
Serious eye damage or eye irritation	Yes
Specific target organ toxicity (single or repeated exposure)	No
Aspiration Hazard	No
Germ cell mutagenicity	No
Simple Asphyxiant	No
Hazards Not Otherwise Classified	No

US. EPA CERCLA HAZARDOUS SUBSTANCES AND REPORTABLE QUANTITIES (40 CFR 302.4)

None Reported

State Regulations

US. CALIFORNIA PROPOSITION 65

None Reported

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (polyethylene glycol; SOD NITROPRUSSIDE SOLUTION; SODIUM SALICYLATE-USP)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (SOD NITROPRUSSIDE SOLUTION)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (polyethylene glycol; SOD NITROPRUSSIDE SOLUTION; SODIUM SALICYLATE-USP)
Vietnam - NCI	Yes
Russia - ARIPS	Yes
Thailand - TECI	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 OTHER INFORMATION

Revision Date	11/12/2018
Initial Date	09/06/2005

SDS Version Summary

Version	Issue Date	Sections Updated
6.1.1.1	04/16/2014	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Advice to Doctor, Chronic Health, Classification, Disposal, Engineering Control, Environmental, First Aid (inhaled), First Aid (swallowed), Ingredients, Personal Protection (other), Personal Protection (Respirator), Personal

Continued...

		Protection (eye), Personal Protection (hands/feet), Spills (major), Spills (minor), Storage (storage incompatibility), Storage (suitable container), Toxicity and Irritation (Other)
7.1.1.1	11/12/2018	Name

Other information

Ingredients with multiple cas numbers

Name	CAS No
polyethylene glycol	25322-68-3, 8038-37-7, 9081-95-2, 9085-02-3, 9085-03-4, 12676-74-3, 12770-93-3, 25104-58-9, 25609-81-8, 34802-42-1, 37361-15-2, 50809-04-6, 50809-59-1, 54510-95-1, 54847-64-2, 59763-40-5, 60894-12-4, 61840-14-0, 64441-68-5, 64640-28-4, 67411-64-7, 70926-57-7, 75285-02-8, 75285-03-9, 77986-38-0, 79964-26-4, 80341-53-3, 85399-22-0, 85945-29-5, 88077-80-9, 88747-22-2, 90597-70-9, 99264-61-6, 99333-89-8, 101677-86-5, 106186-24-7, 107502-63-6, 107529-96-4, 109550-27-8, 112384-37-9, 112895-21-3, 114323-93-2, 116549-90-7, 119219-06-6, 125223-68-9, 133573-31-6, 134919-43-0, 150872-82-5, 154394-38-4, 156948-19-5, 169046-53-1, 174460-08-3, 174460-09-4, 188364-77-4, 188924-03-0, 189154-62-9, 191743-71-2, 196696-84-1, 201163-43-1, 206357-86-0
SOD NITROPRUSSIDE SOLUTION	14402-89-2, 13755-38-9

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.