Hematoxylin, Gill 1X

Astral Diagnostics, Inc.

Part Number: **7012-16, 7012-G**
Version No: **1.2**
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Issue Date: **09 NOV 2022**

L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	Hematoxylin, Gill 1X
Synonyms	Not Available
Other means of identification	7012-16, 7012-G

Recommended use of the chemical and restrictions on use

Relevant identified uses	Laboratory Reagent.

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

Registered company name	**Ethos Biosciences, Inc.**
Address	United States
Telephone	800-441-0366 - Technical Service; Available Monday through Friday, 8:00 AM to 4:00 PM, Eastern US Time
Fax	Not Available
Website	http://www.ethosbiosciences.com/
Email	Not Available

Emergency phone number

Association / Organisation	**CHEMTREC (USA)**
Emergency telephone numbers	**800-424-9300, 24 hours per day, 7 days per week**
Other emergency telephone numbers	**Not Available**

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	Sensitisation (Respiratory) Category 1, Specific Target Organ Toxicity - Repeated Exposure Category 2, Serious Eye Damage/Eye Irritation Category 1, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1

Label elements

Hazard pictogram(s)	
Signal word	**Danger**

Hazard statement(s)

H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H373	May cause damage to organs through prolonged or repeated exposure.
H318	Causes serious eye damage.
H302	Harmful if swallowed.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) Prevention

P260	Do not breathe mist/vapors/spray.
P261	Avoid breathing mist/vapors/spray.
P284	[In case of inadequate ventilation] wear respiratory protection.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P272	Contaminated work clothing must not be allowed out of the workplace.

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove person to fresh air and keep 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/doctor/physician/first aider.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.
P314	Get medical advice/attention if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
517-28-2	<1	hematoxylin
7681-55-2	<1	sodium iodate
107-21-1	25	ethylene glycol
7784-31-8	<4	aluminum sulfate, hydrated
64-19-7	<2	acetic acid, glacial
7732-18-5	>65	water

SECTION 4 First-aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with eyes: ‣ Wash out immediately with water. ‣ If irritation continues, seek medical attention. ‣ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ‣ Quickly but gently, wipe material off skin with a dry, clean cloth. ‣ Immediately remove all contaminated clothing, including footwear. ‣ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ‣ Transport to hospital, or doctor.
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, without delay.
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

Treat symptomatically.

- Polyethylene glycols are generally poorly absorbed orally and are mostly unchanged by the kidney.
- Dermal absorption can occur across damaged skin (e.g. through burns) leading to increased osmolality, anion gap metabolic acidosis, elevated calcium, low ionised calcium, CNS depression and renal failure.
- Treatment consists of supportive care.

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 Fire-fighting measures

Extinguishing media

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.

Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.
In such an event consider:

- foam.
- dry chemical powder.
- carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.

Special protective equipment and precautions for fire-fighters

Fire Fighting	• Alert Fire Brigade and tell them location and nature of hazard. • Wear breathing apparatus plus protective gloves in the event of a fire. • Prevent, by any means available, spillage from entering drains or water courses. • Use fire fighting procedures suitable for surrounding area. • **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. • Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	• The material is not readily combustible under normal conditions. • However, it will break down under fire conditions and the organic component may burn. • Not considered to be a significant fire risk. • Heat may cause expansion or decomposition with violent rupture of containers. • Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). • May emit acrid smoke. Decomposes on heating and produces toxic fumes of: carbon dioxide (CO2) metal oxides 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	• Clean up all spills immediately. • Avoid breathing vapors 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	Chemical Class: alcohols and glycols For release onto land: recommended sorbents listed in order of priority. SORBENT TYPE / RANK / APPLICATION / COLLECTION / LIMITATIONS LAND SPILL - SMALL cross-linked polymer - particulate: 1 / shovel / shovel / R, W, SS cross-linked polymer - pillow: 1 / throw / pitchfork / R, DGC, RT sorbent clay - particulate: 2 / shovel / shovel / R,I, P wood fiber - pillow: 3 / throw / pitchfork / R, P, DGC, RT treated wood fiber - pillow: 3 / throw / pitchfork / DGC, RT foamed glass - pillow: 4 / throw / pichfork / R, P, DGC, RT

LAND SPILL - MEDIUM

cross-linked polymer - particulate	1	blower	skiploader	R,W, SS
polypropylene - particulate	2	blower	skiploader	W, SS, DGC
sorbent clay - particulate	2	blower	skiploader	R, I, W, P, DGC
polypropylene - mat	3	throw	skiploader	DGC, RT
expanded mineral - particulate	3	blower	skiploader	R, I, W, P, DGC
polyurethane - mat	4	throw	skiploader	DGC, RT

Legend
DGC: Not effective where ground cover is dense
R; Not reusable
I: Not incinerable
P: Effectiveness reduced when rainy
RT:Not effective where terrain is rugged
SS: Not for use within environmentally sensitive sites
W: Effectiveness reduced when windy
Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;
R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988
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.
- Stop leak if safe to do so.
- Contain spill with sand, earth or vermiculite.
- Collect recoverable product into labelled containers for recycling.
- Neutralise/decontaminate residue (see Section 13 for specific agent).
- Collect solid residues and seal in labelled drums for disposal.
- Wash area and prevent runoff into drains.
- After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	‣ 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. ‣ **DO NOT** allow material to contact humans, exposed food or food utensils. ‣ 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. Launder contaminated clothing before re-use. ‣ 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 are maintained.
Other information	Consider storage under inert gas.

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Polyethylene or polypropylene container. ‣ Packing as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	aluminum sulfate ‣ forms sulfuric acid in water ‣ reacts violently with bases and many other materials ‣ dry material is weakly corrosive to carbon steel; aqueous solution attacks aluminum and other metals forming hydrogen gas

- Glycols and their ethers undergo violent decomposition in contact with 70% perchloric acid. This seems likely to involve formation of the glycol perchlorate esters (after scission of ethers) which are explosive, those of ethylene glycol and 3-chloro-1,2-propanediol being more powerful than glyceryl nitrate, and the former so sensitive that it explodes on addition of water.

Acetic acid:

- vapors forms explosive mixtures with air (above 39 C.)
- reacts violently with bases such as carbonates and hydroxides (giving off large quantities of heat), oxidisers, organic amines, acetaldehyde, potassium tert-butoxide
- reacts (sometimes violently), with strong acids, aliphatic amines, alkanolamines, alkylene oxides, epichlorohydrin, acetic anhydride, 2-aminoethanol, ammonia, ammonium nitrate, bromine pentafluoride, chlorosulfonic acid, chromic acid, chromium trioxide, ethylenediamine, ethyleneimine, hydrogen peroxide, isocyanates, oleum, perchloric acid, permanganates, phosphorus isocyanate, phosphorus trichloride, sodium peroxide, xylene
- attacks cast iron, stainless steel and other metals, forming flammable hydrogen gas
- attacks many forms of rubber, plastics and coatings

Alcohols

- are incompatible with strong acids, acid chlorides, acid anhydrides, oxidising and reducing agents.
- reacts, possibly violently, with alkaline metals and alkaline earth metals to produce hydrogen
- react with strong acids, strong caustics, aliphatic amines, isocyanates, acetaldehyde, benzoyl peroxide, chromic acid, chromium oxide, dialkylzincs, dichlorine oxide, ethylene oxide, hypochlorous acid, isopropyl chlorocarbonate, lithium tetrahydroaluminate, nitrogen dioxide, pentafluoroguanidine, phosphorus halides, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium
- should not be heated above 49 deg. C. when in contact with aluminum equipment

Ethylene glycol:

- reacts violently with oxidisers and oxidising acids, sulfuric acid, chlorosulfonic acid, chromyl chloride, perchloric acid
- forms explosive mixtures with sodium perchlorate
- is incompatible with strong acids, caustics, aliphatic amines, isocyanates, chlorosulfonic acid, oleum, potassium bichromate, phosphorus pentasulfide, sodium chlorite
- Avoid strong acids, bases.

+	x	+	x	+	+	+

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

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-1	hematoxylin	Particulates Not Otherwise Regulated (PNOR)- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	hematoxylin	Particulates Not Otherwise Regulated (PNOR)- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	hematoxylin	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	hematoxylin	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	hematoxylin	Particulates not otherwise regulated	Not Available	Not Available	Not Available	See Appendix D
US NIOSH Recommended Exposure Limits (RELs)	ethylene glycol	Ethylene glycol	Not Available	Not Available	Not Available	See Appendix D
US NIOSH Recommended Exposure Limits (RELs)	aluminum sulfate, hydrated	Aluminum (soluble salts and alkyls, as Al)	2 mg/m3	Not Available	Not Available	Not Available

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-1	acetic acid, glacial	Acetic acid	10 ppm / 25 mg/m3	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	acetic acid, glacial	Acetic acid	10 ppm / 25 mg/m3	37 mg/m3 / 15 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
sodium iodate	0.83 mg/m3	9.1 mg/m3	55 mg/m3
ethylene glycol	30 ppm	150 ppm	900 ppm
aluminum sulfate, hydrated	38 mg/m3	64 mg/m3	380 mg/m3
acetic acid, glacial	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
hematoxylin	Not Available	Not Available
sodium iodate	Not Available	Not Available
ethylene glycol	Not Available	Not Available
aluminum sulfate, hydrated	Not Available	Not Available
acetic acid, glacial	50 ppm	Not Available
water	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
sodium iodate	E	≤ 0.01 mg/m³
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

These exposure guidelines have been derived from a screening level of risk assessment and should not be construed as unequivocally safe limits. ORGS represent an 8-hour time-weighted average unless specified otherwise.
CR = Cancer Risk/10000; UF = Uncertainty factor:
TLV believed to be adequate to protect reproductive health:
LOD: Limit of detection
Toxic endpoints have also been identified as:
D = Developmental; R = Reproductive; TC = Transplacental carcinogen
Jankovic J., Drake F.: A Screening Method for Occupational Reproductive
American Industrial Hygiene Association Journal 57: 641-649 (1996)

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odor Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odor Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odor Threshold Value (OTV) ppm

Classification into classes follows:
ClassOSF Description

A 550 Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities
B 26-550As "A" for 50-90% of persons being distracted
C 1-26 As "A" for less than 50% of persons being distracted
D 0.18-1 10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached
E <0.18 As "D" for less than 10% of persons aware of being tested
NOTE: Detector tubes for sulfuric acid, measuring in excess of 1 mg/m3, are commercially available.
Based on controlled inhalation studies the TLV-TWA is thought to be protective against the significant risk of pulmonary irritation and incorporates a margin of safety so as to prevent injury to the skin and teeth seen in battery workers acclimatised to workplace concentrations of 16 mg/m3. Experimental evidence in normal unacclimated humans indicates the recognition, by all subjects, of odor, taste or irritation at 3 mg/m3 or 5 mg/m3. All subjects reported these levels to be objectionable but to varying degrees.

The TLV is based on the exposures to aluminum chloride and the amount of hydrolysed acid and the corresponding acid TLV to provide the same degree of freedom from irritation. Workers chronically exposed to aluminum dusts and fumes have developed severe pulmonary reactions including fibrosis, emphysema

and pneumothorax. A much rarer encephalopathy has also been described.
for ethylene glycol:
Odor Threshold: 25 ppm
NOTE: Detector tubes for ethylene glycol, measuring in excess of 10 mg/m3, are commercially available.
It appears impractical to establish separate TLVs for ethylene glycol vapor and mists. Atmospheric concentration that do not cause discomfort are unlikely to cause adverse effects. The TLV-C is thought to be protective against throat and respiratory irritation and headache reported in exposed humans. NIOSH has not established a limit for this substance due to the potential teratogenicity associated with exposure and because respiratory irritation reported at the TLV justified a lower value
for acetic acid:
NOTE:Detector tubes for acetic acid, measuring in excess of 1 ppm, are commercially available.
Exposure at or below the TLV-TWA and TLV-STEL is thought to protect the worker against conjunctival, nose and respiratory tract irritation.
Odor Safety Factor(OSF)
OSF21 ("ACETIC ACID, GLACIAL")

Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.

General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in special circumstances. If risk of overexposure exists, wear approved respirator. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. Provide adequate ventilation in warehouses and enclosed storage areas. 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.

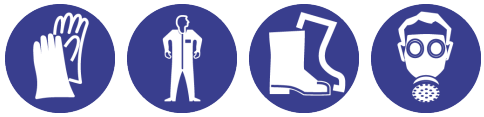
Type of Contaminant:	Air Speed:
solvent, vapors, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, 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.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion)	2.5-10 m/s (500-2000 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 favorable 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. 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 m/s (200-400 f/min) for extraction of solvents generated in a tank 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.

Personal protection



Eye and face protection

- 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	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 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. ‣ Wear chemical protective gloves, e.g. PVC. ‣ Wear safety footwear or safety gumboots, e.g. Rubber
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit.

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AB-AUS	-	AB-PAPR-AUS / Class 1
up to 50 x ES	-	AB-AUS / Class 1	-
up to 100 x ES	-	AB-2	AB-PAPR-2 ^

^ - Full-face
A(All classes) = Organic vapors, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

‣ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapor concentrations or oxygen content.

- The wearer must be warned to leave the contaminated area immediately on detecting any odors through the respirator. The odor may indicate that the mask is not functioning properly, that the vapor 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	Maroon

Physical state	Liquid	**Relative density (Water = 1)**	Not Available
Odor	Vinegar odor	**Partition coefficient n-octanol / water**	Not Available
Odor threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	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 Available
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidizing properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapor pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (Not Available%)**	Not Available
Vapor density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

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	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of

	gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. Inhalation of vapors may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Not normally a hazard due to non-volatile nature of product Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioral changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary edema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapor concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well. The material has **NOT** been classified by EC Directives or other classification systems as "harmful by inhalation". This is because of the lack of corroborating animal or human evidence. In the absence of such evidence, care should be taken nevertheless to ensure exposure is kept to a minimum and that suitable control measures be used, in an occupational setting to control vapors, fumes and aerosols. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact.
Ingestion	Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing. Acute toxic responses to aluminum are confined to the more soluble forms. The toxic effects of glycols (dihydric alcohols), following ingestion are similar to those of alcohol, with depression of the central nervous system (CNS), nausea, vomiting and degenerative changes in liver and kidney. Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased. Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary edema. Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in he case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent. The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. for ethylene glycol: Ingestion symptoms include respiratory failure, central nervous depression, cardiovascular collapse, pulmonary edema, acute kidney failure, and even brain damage. Ingestion of 100 ml has caused death. (ChemInfo) Toxicity of ethylene glycol to human (KB) cell cultures has been reported as less than that of ethanol. (NIOSHTIC) Ethylene glycol produces a three-stage response with the severity of each stage dependent on the amount of ingestion. Hepatic damage is usually minimal. Central nervous system depression characterise the first 12 hours post ingestion. Transient exhilaration occurs without the odor of ethanol. Gastrointestinal complaints include nausea and vomiting. Acidosis, coma, convulsions and myoclonic jerks may also be evident. The optic fundus is usually normal although the presence of papilloedema may confuse the presentation with that produced by methanol. Nystagmus and opthalmoplegias may appear.

	Cardiopulmonary effects are seen 12-24 hours post-ingestion and are characterised by tachycardia, tachypnea, and mild hypertension. Congestive heart failure and circulatory collapse may occur in severe intoxications. Renal effects are seen 24-72 hours post-ingestion and are characterised by oliguria, flank pain, acute tubular necrosis, renal failure, and rarely, bone marrow arrest. Renal damage may be permanent. Toxic effects of ethylene glycol are similar to those produced by ethanol but ethylene glycol produces toxic metabolites. Metabolic acidosis and anion gap result primarily from glycolic acid formation and some lactic acid formation. The citric acid cycle is inhibited as a result of reduced NAD/NADH ratios and to a limited extent, the formation of oxalic acid, and to metabolic acidosis. Oxalate formation produces myocardial depression and acute tubular necrosis. Glycoaldehyde, glycolic acid and glyoxylic acid may contribute to CNS depression and may also produce renal toxicity by producing renal edema. Hypocalcemia may result from chelation by oxalate. Oxalic acid, glycoxalic acid, glycoaldehyde and formic acid appear to form to only a limited degree during intoxication. Oral administration to pregnant mice and rats produced birth defects amongst the off-spring.
Skin Contact	Strong evidence exists that exposure to the material may produce serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. The material is not thought to produce adverse health effects or skin irritation following contact (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man. 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	Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Occupational exposure to aluminum compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminum or its compounds are carcinogenic. Because aluminum competes with calcium for absorption, increased amounts of dietary aluminum may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminum can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminum and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminum, such as deodorants or antacids. In those without allergies, aluminum is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminum cookware has not been shown to lead to aluminum toxicity in general, excessive consumption of antacids containing aluminum compounds and excessive use of aluminum-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminum significantly increases aluminum absorption, and maltol has been shown to increase the accumulation of aluminum in nervous and osseus tissue. Furthermore, aluminum increases estrogen-related gene expression in human breast cancer cells cultured in the laboratory These salts' estrogen-like effects have led to their classification as a metalloestrogen.Some researchers have expressed concerns that the aluminum in antiperspirants may increase the risk of breast cancer. After absorption, aluminum distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminum ion in plasma is the iron binding protein, transferrin. aluminum can enter the brain and reach the placenta and fetus. aluminum may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminum appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans. At high levels of exposure, some aluminum compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminum compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminum potassium sulphate at high levels in the diet. aluminum has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminum. It has been suggested that aluminum is implicated in the aetiology of Alzheimer s disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminum have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminum compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminum/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminum/kg bw per day, respectively. Controversy exists over whether aluminum is the cause of degenerative brain disease (Alzheimer's disease or AD). Several

epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminum in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminum compared with communities where the aluminum level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminum exposure to brain disease. Aluminum concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminum displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.
Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminum hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminum stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminum which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminum has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA). Aluminum enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminum include baking powder, antacids and aluminum products used for general food preparation and storage (over 12 months, aluminum levels in soft drink packed in aluminum cans rose from 0.05 to 0.9 mg/l). [*Walton, J and Bryson-Taylor, D. - Chemistry in Australia, August 1995*]
Human volunteers exposed to ethylene glycol, 20 to 22 hours/day at mean daily concentrations ranging form 1.4 to 27 ppm for about 4 weeks complained of throat irritation, mild headache and low backache. Complaints became marked when the concentration in the exposure chamber was raised above 56 mg/m3 for part of the day. The most common complaint was irritation of the upper respiratory tract. Concentrations above 80 ppm were intolerable with a burning sensation along the trachea and a burning cough. Excessively exposed workers have reported drowsiness.
Repeated minor oral exposure to acetic acid can cause blackening of the skin and teeth, erosion of the teeth, vomiting, diarrhea, nausea. Repeated minor vapor exposure may cause chronic respiratory inflammation and bronchitis.
It is reported that workers exposed for 7 to 12 years at concentrations of 60 ppm acetic acid, plus one hour daily at 100-260 ppm had no injury except slight irritation of the respiratory tract, stomach, and skin although this report is equivocal as in another study different researchers found conjunctivitis, bronchitis, pharyngitis and erosion of exposed teeth apparently in the same workers. Occupational exposures for 7-12 years to concentrations of 80-200 ppm, at peaks, caused blackening and hyperkeratosis of the skin and hands, conjunctivitis (but no corneal damage), bronchitis and pharyngitis and erosion of the exposed teeth (incisors and canines). Digestive disorders with heartburn and constipation have been reported at unspecified prolonged exposures.

Hematoxylin, Gill 1X	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

hematoxylin	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; >=2000 mg/kg[1]	Not Available

sodium iodate	**TOXICITY**	**IRRITATION**
	Oral (Mouse) LD50; 505 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]

ethylene glycol	**TOXICITY**	**IRRITATION**
	dermal (mouse) LD50: >3500 mg/kg[1]	Eye (rabbit): 100 mg/1h - mild
	Oral (Rat) LD50; >2000 mg/kg[2]	Eye (rabbit): 12 mg/m3/3D
		Eye (rabbit): 1440mg/6h-moderate
		Eye (rabbit): 500 mg/24h - mild
		Eye: no adverse effect observed (not irritating)[1]
		Skin (rabbit): 555 mg(open)-mild
		Skin: no adverse effect observed (not irritating)[1]

aluminum sulfate, hydrated	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 370 mg/kg[2]	Not Available

acetic acid, glacial	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 1060 mg/kg[2]	Eye (rabbit): 0.05mg (open)-SEVERE
	Inhalation(Mouse) LC50; 1.405 mg/L4h[2]	Skin (human):50mg/24hr - mild
	Oral (Rat) LD50; 3310 mg/kg[2]	Skin (rabbit):525mg (open)-SEVERE

water	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; >90000 mg/kg[2]	Not Available

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*

Hematoxylin, Gill 1X	For aluminum compounds: aluminum present in food and drinking water is poorly absorbed through the gastrointestinal tract. The bioavailability of aluminum is dependent on the form in which it is ingested and the presence of dietary constituents with which the metal cation can complex Ligands in food can have a marked effect on absorption of aluminum, as they can either enhance uptake by forming absorbable (usually water soluble) complexes (e.g., with carboxylic acids such as citric and lactic), or reduce it by forming insoluble compounds (e.g., with phosphate or dissolved silicate). Considering the available human and animal data it is likely that the oral absorption of aluminum can vary 10-fold based on chemical form alone. Although bioavailability appears to generally parallel water solubility, insufficient data are available to directly extrapolate from solubility in water to bioavailability. For oral intake from food, the European Food Safety Authority (EFSA) has derived a tolerable weekly intake (TWI) of 1 milligram (mg) of aluminum per kilogram of bodyweight. In its health assessment, the EFSA states a medium bioavailability of 0.1 % for all aluminum compounds which are ingested with food. This corresponds to a systemically available tolerable daily dose of 0.143 micrograms (µg) per kilogram (kg) of body weight. This means that for an adult weighing 60 kg, a systemically available dose of 8.6 µg per day is considered safe. Based on a neuro-developmental toxicity study of aluminum citrate administered via drinking water to rats, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg bw (expressed as aluminum) for all aluminum compounds in food, including food additives. The Committee on Toxicity of chemicals in food, consumer products and the environment (COT) considers that the derivation of this PTWI was sound and that it should be used in assessing potential risks from dietary exposure to aluminum. The Federal Institute for Risk Assessment (BfR) of Germany has assessed the estimated aluminum absorption from antiperspirants. For this purpose, the data, derived from experimental studies, on dermal absorption of aluminum from antiperspirants for healthy and damaged skin was used as a basis. At about 10.5 µg, the calculated systemic intake values for healthy skin are above the 8.6 µg per day that are considered safe for an adult weighing 60 kg. If aluminum -containing antiperspirants are used on a daily basis, the tolerable weekly intake determined by the EFSA is therefore exceeded. The values for damaged skin, for example injuries from shaving, are many times higher. This means that in case of daily use of an aluminum-containing antiperspirant alone, the TWI may be completely exhausted. In addition, further aluminum absorption sources such as food, cooking utensils and other cosmetic products must be taken into account Systemic toxicity after repeated exposure No studies were located regarding dermal effects in animals following intermediate or chronic-duration dermal exposure to various forms of aluminum. When orally administered to rats, aluminum compounds (including aluminum nitrate, aluminum sulfate and potassium aluminum sulfate) have produced various effects, including decreased gain in body weight and mild histopathological changes in the spleen, kidney and liver of rats (104 mg Al/kg bw/day) and dogs (88-93 mg Al/kg bw/day) during subchronic oral exposure. Effects on nerve cells, testes, bone and stomach have been reported at higher doses. Severity of effects increased with dose. The main toxic effects of aluminum that have been observed in experimental animals are neurotoxicity and nephrotoxicity. Neurotoxicity has also been described in patients dialysed with water containing high concentrations of aluminum, but epidemiological data on possible adverse effects in humans at lower exposures are inconsistent Reproductive and developmental toxicity: Studies of reproductive toxicity in male mice (intraperitoneal or subcutaneous administration of aluminum nitrate or chloride) and rabbits (administration of aluminum chloride by gavage) have demonstrated the ability of aluminum to cause testicular toxicity, decreased sperm quality in mice and rabbits and reduced fertility in mice. No reproductive toxicity was seen in females given aluminum nitrate by gavage or dissolved in drinking water. Multi-generation reproductive studies in which aluminum sulfate and aluminum ammonium sulfate were administered to rats in drinking water, showed no evidence of reproductive toxicity High doses of aluminum compounds given by gavage have induced signs of embryotoxicity in mice and rats in particular, reduced fetal body weight or pup weight at birth and delayed ossification. Developmental toxicity studies in which aluminum chloride was administered by gavage to pregnant rats showed evidence of foetotoxicity, but it was unclear whether the findings were secondary to maternal toxicity. A twelve-month neuro-development with aluminum citrate administered via the drinking water to Sprague-Dawley rats, was conducted according to Good Laboratory Practice (GLP). aluminum citrate was selected for the study since it is the most soluble and bioavailable aluminum salt. Pregnant rats were exposed to aluminum citrate from gestational day 6 through lactation, and then the offspring were exposed post-weaning until postnatal day 364. An extensive functional observational battery of tests was performed at various times. Evidence of aluminum toxicity was demonstrated in the high (300 mg/kg bw/day of aluminum) and to a lesser extent, the mid-dose groups (100 mg/kg bw/day of aluminum). In the high-dose group, the main effect was renal damage, resulting in high mortality in the male offspring. No major neurological pathology or neurobehavioral effects were observed, other than in the neuromuscular subdomain (reduced grip strength and increased foot splay). Thus, the lowest observed adverse effect level (LOAEL) was 100 mg/kg bw/day and the no observed adverse effect level (NOAEL) was 30 mg/kg bw/day. Bioavailability of aluminum chloride, sulfate and nitrate and aluminum hydroxide was much lower than that of aluminum citrate This study was used by JECFA as key study to derive the PTWI. Genotoxicity aluminum compounds were non-mutagenic in bacterial and mammalian cell systems, but some produced DNA damage and effects on chromosome integrity and segregation in vitro. Clastogenic effects were also observed in vivo when aluminum sulfate was administered at high doses by gavage or by the intraperitoneal route. Several indirect mechanisms have been proposed to explain the variety of genotoxic effects elicited by aluminum salts in experimental systems. Cross-linking of DNA with chromosomal proteins, interaction with microtubule assembly and mitotic spindle functioning, induction of oxidative damage, damage of lysosomal membranes with liberation of DNAase, have been suggested to explain the induction of structural chromosomal aberrations, sister chromatid exchanges, chromosome loss and formation of oxidized bases in experimental systems. The EFSA Panel noted that these indirect mechanisms of genotoxicity, occurring at relatively high levels of exposure, are unlikely to be of relevance for humans exposed to aluminum via the diet. aluminum compounds do not cause gene mutations in either bacteria or mammalian cells. Exposure to aluminum compounds does result in both structural and numerical

	chromosome aberrations both in in-vitro and in-vivo mutagenicity tests. DNA damage is probably the result of indirect mechanisms. The DNA damage was observed only at high exposure levels. Carcinogenicity. The available epidemiological studies provide limited evidence that certain exposures in the aluminum production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. However, the aluminum exposure was confounded by exposure to other agents including polycyclic aromatic hydrocarbons, aromatic amines, nitro compounds and asbestos. There is no evidence of increased cancer risk in non-occupationally exposed persons. Neurodegenerative diseases. Following the observation that high levels of aluminum in dialysis fluid could cause a form of dementia in dialysis patients, a number of studies were carried out to determine if aluminum could cause dementia or cognitive impairment as a consequence of environmental exposure over long periods. aluminum was identified, along with other elements, in the amyloid plaques that are one of the diagnostic lesions in the brain for Alzheimer disease, a common form of senile and pre-senile dementia. some of the epidemiology studies suggest the possibility of an association of Alzheimer disease with aluminum in water, but other studies do not confirm this association. All studies lack information on ingestion of aluminum from food and how concentrations of aluminum in food affect the association between aluminum in water and Alzheimer disease." There are suggestions that persons with some genetic variants may absorb more aluminum than others, but there is a need for more analytical research to determine whether aluminum from various sources has a significant causal association with Alzheimer disease and other neurodegenerative diseases.aluminum is a neurotoxicant in experimental animals. However, most of the animal studies performed have several limitations and therefore cannot be used for quantitative risk assessment. Contact sensitivity: It has been suggested that the body burden of aluminum may be linked to different iseases. Macrophagic myofasciitis and chronic fatigue syndrome can be caused by aluminum-containing adjuvants in vaccines. Macrophagic myofasciitis (MMF) has been described as a disease in adults presenting with ascending myalgia and severe fatigue following exposure to aluminum hydroxide-containing vaccines The corresponding histological findings include aluminum-containing macrophages infiltrating muscle tissue at the injection site. The hypothesis is that the long-lasting granuloma triggers the development of the systemic syndrome. aluminum acts not only as an adjuvant,stimulating the immune system either to fend off infections or to tolerate antigens, it also acts as a sensitisers causing contact allergy and allergic contact dermatitis. In general, metal allergies are very common and aluminum is considered to be a weak allergen. A metal must be ionised to be able to act as a contact allergen, then it has to undergo haptenisation to be immunogenic and to initiate an immune response.Once inside the skin, the metal ions must bind to proteins to become immunologically reactive.The most important routes of exposure and sensitisation to aluminum are through aluminum-containing vaccines. One Swedish study showed a statistically significant association between contact allergy to aluminum and persistent itching nodules in children treated with allergen-specific immunotherapy (ASIT) Nodules were overrepresented in patients with contact allergy to aluminum Other routes of sensitisation reported in the literature are the prolonged use of aluminum-containing antiperspirants, topical medication, and tattooing of the skin with aluminum-containing pigments. Most of the patients experienced eczematous reactions whereas tattooing caused granulomas. Even though aluminum is used extensively in industry, only a low number of cases of occupational skin sensitisation to aluminum have been reported Systemic allergic contact dermatitis in the form of flare-up reactions after re-exposure to aluminum has been documented: pruritic nodules at present and previous injection sites, eczema at the site of vaccination as well as at typically atopic localisations after vaccination with aluminum-containing vaccines and/or patch testing with aluminum, and also after use of aluminum-containing toothpaste
HEMATOXYLIN	May be carcinogenic [Hawleys]
SODIUM IODATE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's edema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure.
ETHYLENE GLYCOL	[Estimated Lethal Dose (human) 100 ml; RTECS quoted by Orica] Substance is reproductive effector in rats (birth defects). Mutagenic to rat cells.
ACETIC ACID, GLACIAL	for acid mists, aerosols, vapors Data from assays for genotoxic activity in vitro suggest that eukaryotic cells are susceptible to genetic damage when the pH falls to about 6.5. Cells from the respiratory tract have not been examined in this respect. Mucous secretion may protect the cells of the airways from direct exposure to inhaled acidic mists, just as mucous plays an important role in protecting the gastric epithelium from its auto-secreted hydrochloric acid. In considering whether pH itself induces genotoxic events in vivo in the respiratory system, comparison should be made with the human stomach, in which gastric juice may be at pH 1-2 under fasting or nocturnal conditions, and with the human urinary bladder, in which the pH of urine can range from <5 to=""> 7 and normally averages 6.2. Furthermore, exposures to low pH in vivo differ from exposures *in vitro* in that, *in vivo*, only a portion of the cell

	surface is subjected to the adverse conditions, so that perturbation of intracellular homeostasis may be maintained more readily than in vitro. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may produce severe 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) thickening of the epidermis. Histologically there may be intercellular edema of the spongy layer (spongiosis) and intracellular edema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration. NOAELs following repeated exposure to acetic acid and its salts range from 210 mg/kg bw/day (2-4 month acetic acid drinking water study; systemic toxicity) to 3600 mg/kg bw/day (acetic acid, sodium salt, 4 week dietary study; no effects reported). Signs of irritation/corrosion at the site of contact as well as systemic toxicity have been reported. Prolonged inhalation exposure to acetic acid results in muscle imbalance, increase in blood cholinesterase activity, decreases in albumins and decreased growth at concentrations greater than 0.01 mg/m3/day. Groups of 20 mice/sex were given 0.025% sodium acetate in drinking water (about 60 mg/kg bw/day) for 1 week before breeding, during a 9-day breeding period and (females only) throughout pregnancy, lactation and until the offspring were weaned at 3 weeks of age. No effects on fertility were observed. The male offspring were given the same solution until they were 5-7 weeks old and were then examined in a 24-hour activity test. Examination of the litters revealed no overt deformities and normal pup weights at day 1 and day 21. The activity of offspring of the treated group was lower than that of controls during the first 12 hours but was similar during the second 12 hours. It is unknown if the decreased activity observed in the sodium acetate treated group to was a result of exposure in utero and/or post-weaning, since the pups were exposed during both time periods.). Acetic acid had no effects on implantation or on maternal or fetal survival in rats, mice or rabbits dosed via gavage during gestation days 6-19 at doses up to 1600 mg/kg/day. The number of abnormalities seen in either soft or skeletal tissues of the test groups did not differ from the number occurring in the controls. Sodium acetate had no effect on pregnant mice or offspring when mice were administered 1000 mg/kg bw, by gavage on days 8-12 of gestation.
Hematoxylin, Gill 1X & HEMATOXYLIN & SODIUM IODATE & ACETIC ACID, GLACIAL	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. 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. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.
Hematoxylin, Gill 1X & ETHYLENE GLYCOL	For ethylene glycol: Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol. dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO2, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested. **Respiratory Effects.** Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning Pulmonary edema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases). **Cardiovascular Effects.** Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol. Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases.Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown. **Gastrointestinal Effects.** Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition. **Musculoskeletal Effects.** Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and

	tetanic contractions associated with hypocalcemia. **Hepatic Effects.** Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. **Renal Effects.** Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria , and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy. **Metabolic Effects.** One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate). **Neurological Effects:** Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolized ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion. Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months. **Reproductive Effects:** Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, fetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration. **Developmental Effects:** The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embyrotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in fetal body weight. **Cancer:** No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol. **Genotoxic Effects**: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol.
HEMATOXYLIN & SODIUM IODATE & aluminum SULFATE, HYDRATED & WATER	No significant acute toxicological data identified in literature search.

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

Hematoxylin, Gill 1X	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available

hematoxylin	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	48h	Crustacea	<20mg/l	2
	EC50	48h	Crustacea	~29.7mg/l	2
	LC50	96h	Fish	>35mg/l	2

sodium iodate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	48h	Crustacea	10.3mg/L	5
	EC50	48h	Crustacea	10.3mg/L	5
	LC50	96h	Fish	160-310mg/l	4
ethylene glycol	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	Not Available	Algae or other aquatic plants	6500-7500mg/l	1
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>10000mg/l	1
	EC50	96h	Algae or other aquatic plants	6500-13000mg/l	1
aluminum sulfate, hydrated	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	0.04mg/l	2
	EC50	48h	Crustacea	0.33mg/l	2
	EC50(ECx)	120h	Fish	<0.001mg/L	5
	LC50	96h	Fish	>0.42mg/l	2
	EC50	96h	Algae or other aquatic plants	0.46mg/l	2
acetic acid, glacial	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	24h	Algae or other aquatic plants	0.08mg/l	2
	EC50	72h	Algae or other aquatic plants	29.23mg/l	2
	EC50	48h	Crustacea	18.9mg/l	2
	LC50	96h	Fish	31.3-67.6mg/l	2
water	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*				

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
For aluminum and its compounds and salts:
Despite its prevalence in the environment, no known form of life uses aluminum salts metabolically. In keeping with its pervasiveness, aluminum is well tolerated by plants and animals.Owing to their prevalence, potential beneficial (or otherwise) biological roles of aluminum compounds are of continuing interest.
Environmental fate:
aluminum occurs in the environment in the form of silicates, oxides and hydroxides, combined with other elements such as sodium, fluorine and arsenic complexes with organic matter.
Acidification of soils releases aluminum as a transportable solution. Mobilisation of aluminum by acid rain results in aluminum becoming available for plant uptake. As an element, aluminum cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. Aluminum in compounds has only one oxidation state (+3), and would not undergo oxidation-reduction reactions under environmental conditions. Aluminum can be complexed by various ligands present in the environment (e.g., fulvic and humic acids). The solubility of aluminum in the environment will depend on the ligands present and the pH. The trivalent aluminum ion is surrounded by six water molecules in solution. The hydrated aluminum ion, $[Al(H_2O)_6]^{3+}$, undergoes hydrolysis, in which a stepwise deprotonation of the coordinated water ligands forms bound hydroxide ligands (e.g., $[Al(H_2O)_5(OH)]^{2+}$, $[Al(H_2O)_4(OH)_2]^{+}$). The speciation of aluminum in water is pH dependent. The hydrated trivalent aluminum ion is the predominant form at pH levels below 4. Between pH 5 and 6, the predominant hydrolysis products are $Al(OH)^{2+}$ and $Al(OH)_2^{+}$, while the solid $Al(OH)_3$ is most prevalent between pH 5.2 and 8.8. The soluble species $Al(OH)_4^-$ is the predominant species above pH 9, and is the only species present above pH 10. Polymeric aluminum hydroxides appear between pH 4.7 and 10.5, and increase in size until they are transformed into colloidal particles of amorphous $Al(OH)_3$, which crystallise to gibbsite in acid waters. Polymerisation is affected by the presence of dissolved silica; when enough silica is present, aluminum is precipitated as poorly crystallised clay mineral species.
Hydroxyaluminum compounds are considered amphoteric (e.g., they can act as both acids and bases in solution). Because of this property, aluminum hydroxides can act as buffers and resist pH changes within the narrow pH range of 4-5.
Monomeric aluminum compounds, typified by aluminum fluoride, chloride, and sulfate, are considered reactive or labile compounds, whereas polymeric aluminum species react much more slowly in the environment. Aluminum has a stronger attraction for fluoride in an acidic environment compared to other inorganic ligand.
The adsorption of aluminum onto clay surfaces can be a significant factor in controlling aluminum mobility in the environment, and these adsorption reactions, measured in one study at pH 3.0-4.1, have been observed to be very rapid. However, clays may act either as a sink or a source for soluble aluminum depending on the degree of aluminum saturation on the clay surface.
Within the pH range of 5-6, aluminum complexes with phosphate and is removed from solution. Because phosphate is a necessary nutrient in ecological systems, this immobilization of both aluminum and phosphate may result in depleted nutrient states in surface water.
Plant species and cultivars of the same species differ considerably in their ability to take up and translocate aluminum to above-ground parts. Tea leaves may

contain very high concentrations of aluminum, >5,000 mg/kg in old leaves. Other plants that may contain high levels of aluminum include Lycopodium (Lycopodiaceae), a few ferns, Symplocos (Symplocaceae), and Orites (Proteaceae). Aluminum is often taken up and concentrated in root tissue. In sub-alpine ecosystems, the large root biomass of the Douglas fir, *Abies amabilis*, takes up aluminum and immobilizes it, preventing large accumulation in above-ground tissue. It is unclear to what extent aluminum is taken up into root food crops and leafy vegetables. An uptake factor (concentration of aluminum in the plant/concentration of aluminum in soil) of 0.004 for leafy vegetables and 0.00065 for fruits and tubers has been reported, but the pH and plant species from which these uptake factors were derived are unclear. Based upon these values, however, it is clear that aluminum is not taken up in plants from soil, but is instead biodiluted.
Aluminum concentrations in rainbow trout from an alum-treated lake, an untreated lake, and a hatchery were highest in gill tissue and lowest in muscle. Aluminum residue analyses in brook trout have shown that whole-body aluminum content decreases as the fish advance from larvae to juveniles. These results imply that the aging larvae begin to decrease their rate of aluminum uptake, to eliminate aluminum at a rate that exceeds uptake, or to maintain approximately the same amount of aluminum while the body mass increases. The decline in whole-body aluminum residues in juvenile brook trout may be related to growth and dilution by edible muscle tissue that accumulated less aluminum than did the other tissues.
The greatest fraction of the gill-associated aluminum was not sorbed to the gill tissue, but to the gill mucus. It is thought that mucus appears to retard aluminum transport from solution to the membrane surface, thus delaying the acute biological response of the fish. It has been reported that concentrations of aluminum in whole-body tissue of the Atlantic salmon exposed to high concentrations of aluminum ranging from 3 ug/g (for fish exposed to 33 ug/L) to 96 ug/g (for fish exposed to 264 ug/L) at pH 5.5. After 60 days of exposure, BCFs ranged from 76 to 190 and were directly related to the aluminum exposure concentration. In acidic waters (pH 4.6-5.3) with low concentrations of calcium (0.5-1.5 mg Ca/L), labile aluminum between 25 and 75 ug/L is toxic. Because aluminum is toxic to many aquatic species, it is not bioaccumulated to a significant degree (BCF <300) in most fish and shellfish; therefore, consumption of contaminated fish does not appear to be a significant source of aluminum exposure in humans.
Bioconcentration of aluminum has also been reported for several aquatic invertebrate species. BCF values ranging from 0.13 to 0.5 in the whole-body were reported for the snail. Bioconcentration of aluminum has also been reported for aquatic insects.
Ecotoxicity:
Freshwater species pH >6.5
Fish: Acute LC50 (48-96 h) 5 spp: 0.6 (*Salmo salar)* - 106 mg/L; Chronic NOEC (8-28 d): 7 spp,NOEC, 0.034-7.1 mg/L. The lowest measured chronic figure was an 8-d LC50 of 0.17 mg/L for *Micropterus* sp.
Amphibian: Acute LC50 (4 d): *Bufo americanus*, 0.86-1.66 mg/L; Chronic LC50 (8-d) 2.28 mg/L
Crustaceans LC50 (48 h): 1 sp 2.3-36 9 mg/L; Chronic NOEC (7-28 d) 3 spp, 0.136-1.72 mg/L
Algae EC50 (96 h): population growth, 0.46-0.57 mg/L; 2 spp, chronic NOEC, 0.8-2.0 mg/L
Freshwater species pH <6.5 (all between pH 4.5 and 6.0)
Fish LC50 (24-96 h): 4 spp, 0.015 (*S. trutta*) - 4.2 mg/L; chronic data on *Salmo trutta*, LC50 (21-42 d) 0.015- 0.105 mg/L
Amphibians LC50 (4-5 d): 2 spp, 0.540-2.670 m/L (absolute range 0.40-5.2 mg/L)
Alga: 1 sp NOEC growth 2.0 mg/L
Among freshwater aquatic plants, single-celled plants are generally the most sensitive to aluminum. Fish are generally more sensitive to aluminum than aquatic invertebrates. Aluminum is a gill toxicant to fish, causing both ionoregulatory and respiratory effects.
The bioavailability and toxicity of aluminum is generally greatest in acid solutions. Aluminum in acid habitats has been observed to be toxic to fish and phytoplankton. Aluminum is generally more toxic over the pH range 4.4.5.4, with a maximum toxicity occurring around pH 5.0.5.2. The inorganic single unit aluminum species (Al(OH)2 +) is thought to be the most toxic. Under very acid conditions, the toxic effects of the high H+ concentration appear to be more important than the effects of low concentrations of aluminum; at approximately neutral pH values, the toxicity of aluminum is greatly reduced. The solubility of aluminum is also enhanced under alkaline conditions, due to its amphoteric character, and some researchers found that the acute toxicity of aluminum increased from pH 7 to pH 9. However, the opposite relationship was found in other studies. The uptake and toxicity of aluminum in freshwater organisms generally decreases with increasing water hardness under acidic, neutral and alkaline conditions. Complexing agents such as fluoride, citrate and humic substances reduce the availability of aluminum to organisms, resulting in lower toxicity. Silicon can also reduce aluminum toxicity to fish.
Drinking Water Standards:
aluminum: 200 ug/l (UK max.)
200 ug/l (WHO guideline)
chloride: 400 mg/l (UK max.)
250 mg/l (WHO guideline)
fluoride: 1.5 mg/l (UK max.)
1.5 mg/l (WHO guideline)
nitrate: 50 mg/l (UK max.)
50 mg/l (WHO guideline)
sulfate: 250 mg/l (UK max.)
Soil Guideline: none available.
Air Quality Standards: none available.
Acetic acid and its salts (the acetates) can be grouped together because of their close structural relationships, their natural occurrence in plants and animals, and their fundamental role in cell metabolism, particularly in the tricarboxylic acid cycle (also known as the citric acid or Kreb s cycle), which is where humans get their energy.

- Acetic acid is degraded photochemically in the atmosphere to produce hydroxyl radicals (estimated typical half-life of 22 days). Physical removal of acetates on atmospheric particulates may occur via wet or dry deposition.
- Natural water will neutralise dilute solutions of acetic acid.
- Spills of acetic acid on soil will readily biodegrade - the biodegradation rate for acetic acid after 14 days under aerobic conditions is 74 days.
- In invertebrates the toxicity of acetic acid (EC50 = 50-450 mg/L, depending on test species) -under static conditions, the 48 hour EC50 value for acetic acid is 65 mg/L for aquatic invertebrates (the test media was not neutralised). When the test solutions are neutralised, to form acetates, the static 48 hour EC50 for acetic acid is 6000 mg/L. In renewal systems with aquatic invertebrates, 48 hour EC50s for acetic acid are 100 mg/L and 180 mg/L.
- Fish LC50 (96 h): 75-88 mg/L.
- Acetic acid is not expected to bioconcentrate in the aquatic system.
- Low concentrations of acetic acid are harmful to fish.
- Drinking water standards: none available.
- Soil Guidelines: none available.
- Air Quality Standards: none available

for ethylene glycol:
log Kow : -1.93- -1.36
Half-life (hr) air : 24

Henry's atm m3 /mol: 6.00E-08
BOD 5 : 0.15-0.81,12%
COD : 1.21-1.29
ThOD : 1.26
BCF : 10-190
In the atmosphere ethylene glycol exists mainly in the vapor phase. It is degraded in the atmosphere by reaction with photochemically produced hydroxy radicals (estimated half-life 24-50 hours).
Ethylene glycol does not concentrate in the food chain.
Environmental fate:
Ethylene glycol has a low vapor pressure (7.9 Pa at 20 C); it is expected to exist almost entirely in the vapor phase if released to the atmosphere. The Henry's law constant for ethylene glycol is 1.41 × 10-3 or 6.08 × 10-3 Pa.m3/mol, depending on method of calculation, indicating a low capacity for volatilization from water bodies or soil surfaces.
Ethylene glycol adsorbed onto silica gel and irradiated with light (wavelength >290 nm) degraded by 12.1% over 17 h . Photodegradation is not expected, as the molecule should not absorb at these wavelengths; the mechanism of this breakdown is, therefore, unknown. Estimated half-life in the atmosphere for reaction with hydroxyl radicals from various reports is 2.1 days , 8-84 h or 1 day.
Ethylene glycol released to the atmosphere will be degraded by reaction with hydroxyl radicals; the half-life for the compound in this reaction has been estimated at between 0.3 and 3.5 days. No hydrolysis of ethylene glycol is expected in surface waters.
The compound has little or no capacity to bind to particulates and will be mobile in soil. Soil partition coefficients (log Koc) of 0-0.62 were determined. Migration rates in five soil types were measured at between 4 and 27 cm per 12 h
The low octanol/water partition coefficient (log Kow -1.93 to -1.36)and measured bioconcentration factors in a few organisms indicate low capacity for bioaccumulation. Bioconcentration factors of 190 for the green algae (Chlorella fusca) , up to 0.27 in specific tissues of the crayfish (Procambarus sp.) , and 10 for the golden orfe (Leuciscus idus melanotus) confirm low bioaccumulation.
Ethylene glycol is readily biodegradable in standard tests using sewage sludge. Many studies show biodegradation under both aerobic and anaerobic conditions. Some studies suggest a lag phase before degradation, but many do not. Degradation occurs in both adapted and unadapted sludges. Rapid degradation has been reported in surface waters (less in salt water than in fresh water), groundwater, and soil inocula. Several strains of microorganisms capable of utilizing ethylene glycol as a carbon source have been identified.
Ethylene glycol has been identified as a metabolite of the growth regulator ethylene in a number of higher plants and as naturally occurring in the edible fungus Tricholoma matsutake
Ecotoxicity:
Fish LC50 (96 h):118-550 mg/L
Ethylene glycol has generally low toxicity to aquatic organisms. Toxic thresholds for microorganisms are above 1000 mg/liter. EC50s for growth in microalgae are 6500 mg/liter or higher. Acute toxicity tests with aquatic invertebrates where a value could be determined show LC50s above 20 000 mg/liter, and those with fish show LC50s above 17 800 mg/liter. An amphibian test showed an LC50 for tadpoles at 17 000 mg/litre. A no-observed-effect concentration (NOEC) for chronic tests on daphnids of 8590 mg/liter (for reproductive end-points) has been reported. A NOEC following short-term exposure of fish has been reported at 15 380 mg/litre for growth. Tests using deicer containing ethylene glycol showed greater toxicity to aquatic organisms than observed with the pure compound, indicating other toxic components of the formulations. Laboratory tests exposing aquatic organisms to stream water receiving runoff from airports have demonstrated toxic effects and death. Field studies in the vicinity of an airport have reported toxic signs consistent with ethylene glycol poisoning, fish kills, and reduced biodiversity. These effects cannot definitively be ascribed to ethylene glycol. Terrestrial organisms are much less likely to be exposed to ethylene glycol and generally show low sensitivity to the compound. Concentrations above 100 000 mg/litre were needed to produce toxic effects on yeasts and fungi from soil. Very high concentrations and soaking of seeds produced inhibition of germination in some experiments; these are not considered of environmental significance. A no-observed-effect level (NOEL) for orally dosed ducks at 1221 mg/kg body weight and reported lethal doses for poultry at around 8000 mg/kg body weight indicate low toxicity to birds.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
hematoxylin	HIGH	HIGH
sodium iodate	HIGH	HIGH
ethylene glycol	LOW (Half-life = 24 days)	LOW (Half-life = 3.46 days)
aluminum sulfate, hydrated	HIGH	HIGH
acetic acid, glacial	LOW	LOW
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
hematoxylin	LOW (LogKOW = 0.7145)
sodium iodate	LOW (LogKOW = -4.6296)
ethylene glycol	LOW (BCF = 200)
aluminum sulfate, hydrated	LOW (LogKOW = -2.2002)
acetic acid, glacial	LOW (LogKOW = -0.17)

Mobility in soil

Ingredient	Mobility
hematoxylin	LOW (KOC = 9846)
sodium iodate	LOW (KOC = 35.04)

Ingredient	Mobility
ethylene glycol	HIGH (KOC = 1)
aluminum sulfate, hydrated	LOW (KOC = 6.124)
acetic acid, glacial	HIGH (KOC = 1)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	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. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO

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

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

Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
hematoxylin	Not Available
sodium iodate	Not Available
ethylene glycol	Not Available
aluminum sulfate, hydrated	Not Available
acetic acid, glacial	Not Available
water	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
hematoxylin	Not Available
sodium iodate	Not Available
ethylene glycol	Not Available
aluminum sulfate, hydrated	Not Available

Product name	Ship Type
acetic acid, glacial	Not Available
water	Not Available

SECTION 15 Regulatory information

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

hematoxylin is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US OSHA Permissible Exposure Limits (PELs) Table Z-3
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

sodium iodate is found on the following regulatory lists

US DOE Temporary Emergency Exposure Limits (TEELs)
US List of Active Substances Exempt from the TSCA Inventory Notifications (Active-Inactive) Rule
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

ethylene glycol is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List
US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants
US - California Proposition 65 - Maximum Allowable Dose Levels (MADLs) for Chemicals Causing Reproductive Toxicity
US - California Proposition 65 - Reproductive Toxicity
US - California Safe Drinking Water and Toxic Enforcement Act of 1986 - Proposition 65 List
US - Massachusetts - Right To Know Listed Chemicals
US ATSDR Minimal Risk Levels for Hazardous Substances (MRLs)
US Clean Air Act - Hazardous Air Pollutants
US DOE Temporary Emergency Exposure Limits (TEELs)
US EPA Integrated Risk Information System (IRIS)
US EPCRA Section 313 Chemical List
US NIOSH Recommended Exposure Limits (RELs)
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

aluminum sulfate, hydrated is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals
US CWA (Clean Water Act) - List of Hazardous Substances
US DOE Temporary Emergency Exposure Limits (TEELs)
US NIOSH Recommended Exposure Limits (RELs)
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

acetic acid glacial is found on the following regulatory lists

US - Massachusetts - Right To Know Listed Chemicals
US CWA (Clean Water Act) - List of Hazardous Substances
US DOE Temporary Emergency Exposure Limits (TEELs)
US NIOSH Recommended Exposure Limits (RELs)
US OSHA Permissible Exposure Limits (PELs) Table Z-1
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Chemical Substance Inventory - Interim List of Active Substances

water is found on the following regulatory lists

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
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	Yes
Respiratory or Skin Sensitization	Yes
Serious eye damage or eye irritation	Yes
Specific target organ toxicity (single or repeated exposure)	Yes
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)

Name	Reportable Quantity in Pounds (lb)	Reportable Quantity in kg
ethylene glycol	5000	2270
aluminum sulfate, hydrated	5000	2270
acetic acid glacial	5000	2270

State Regulations

US. California Proposition 65

⚠ **WARNING:** This product can expose you to chemicals including **ethylene glycol**, which is known to the State of California to cause birth defects or other reproductive harm. For more information, go to www.P65Warnings.ca.gov.

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (hematoxylin; sodium iodate; ethylene glycol; aluminum sulfate, hydrated; acetic acid glacial; water)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (hematoxylin; sodium iodate)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	*Yes = All CAS declared ingredients are on the inventory* *No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	09 NOV 2022
Initial Date	11 DEC 2013

Other information

Ingredients with multiple cas numbers

Name	CAS No
ethylene glycol	107-21-1, 1371582-33-0, 2088100-90-5, 37221-95-7, 71767-64-1
aluminum sulfate, hydrated	7784-31-8, 25102-19-6, 57292-32-7, 16828-11-8, 17927-65-0, 10043-01-3, 16828-12-9

Classification of the preparation and its individual components has drawn on official and authoritative sources 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.

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit。
IDLH: Immediately Dangerous to Life or Health Concentrations
ES: Exposure Standard
OSF: Odor Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odor Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances