Refresher Tabs

CHEMITAB Pty Ltd

Part Number: **CHE 01-02-03**
Version No: **2.3**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **21/11/2022**
Print Date: **21/11/2022**
L.GHS.AUS.EN

SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Refresher Tabs
Chemical Name	1,4-dichlorobenzene
Synonyms	Not Available
Other means of identification	CHE 01-02-03

Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	Toilet urinal blocks

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**CHEMITAB Pty Ltd**
Address	7/14 Anvil Road Seven Hills NEW SOUTH WALES 2147 Australia
Telephone	+61296749995
Fax	Not Available
Website	www.chemitab.com.au
Email	george@chemitab.com.au

Emergency telephone number

Association / Organisation	**Not Available**
Emergency telephone numbers	**Not Available**
Other emergency telephone numbers	**0402144114**

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. NON-DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

Poisons Schedule	Not Applicable
Classification [1]	Serious Eye Damage/Eye Irritation Category 2A, Acute Toxicity (Oral) Category 4, Hazardous to the Aquatic Environment Long-Term Hazard Category 1, Carcinogenicity Category 2
Legend:	*1. Classification by vendor; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

Hazard pictogram(s)	

Signal word	**Warning**

Hazard statement(s)

H319	Causes serious eye irritation.
H302	Harmful if swallowed.
H410	Very toxic to aquatic life with long lasting effects.
H351	Suspected of causing cancer.

Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P330	Rinse mouth.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
106-46-7	<99	1,4-dichlorobenzene

Legend:	*1. Classification by vendor; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ‣ Wash out immediately with fresh running water. ‣ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ‣ Seek medical attention without delay; if pain persists or recurs seek medical attention. ‣ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ‣ Immediately remove all contaminated clothing, including footwear.

	‣ Flush skin and hair with running water (and soap if available). ‣ Seek medical attention in event of irritation.
Inhalation	‣ If fumes or combustion products are inhaled remove from contaminated area. ‣ Lay patient down. Keep warm and rested. ‣ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ‣ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ‣ Transport to hospital, or doctor, without delay.
Ingestion	‣ Immediately give a glass of water. ‣ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
Chlorobenzenes are readily adsorbed from the gastrointestinal tract; they are distributed into highly perfused tissues and accumulate in lipid tissues. Lipid accumulation is greatest for the more highly chlorinated chlorobenzene compounds. Chlorobenzenes are metabolised by microsomal oxidation to form arene oxide intermediates and then further to their corresponding chlorophenols which are excreted in the urine as mercapturic acids after conjugation with glutathione or as glucuronic acid or sulfate conjugates. A small percentage are eliminated unchanged in expired air or faeces.
The material may induce methaemoglobinaemia following exposure.

- Initial attention should be directed at oxygen delivery and assisted ventilation if necessary. Hyperbaric oxygen has not demonstrated substantial benefits.
- Hypotension should respond to Trendelenburg's position and intravenous fluids; otherwise dopamine may be needed.
- Symptomatic patients with methaemoglobin levels over 30% should receive methylene blue. (Cyanosis, alone, is not an indication for treatment). The usual dose is 1-2 mg/kg of a 1% solution (10 mg/ml) IV over 50 minutes; repeat, using the same dose, if symptoms of hypoxia fail to subside within 1 hour.
- Thorough cleansing of the entire contaminated area of the body, including the scalp and nails, is of utmost importance.

BIOLOGICAL EXPOSURE INDEX - BEI
These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comment
1. Methaemoglobin in blood	1.5% of haemoglobin	During or end of shift	B, NS, SQ

B: Background levels occur in specimens collected from subjects **NOT** exposed
NS: Non-specific determinant; also observed after exposure to other materials
SQ: Semi-quantitative determinant - Interpretation may be ambiguous; should be used as a screening test or confirmatory test.

SECTION 5 Firefighting measures

Extinguishing media

- Alcohol stable foam.
- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	‣ 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 courses. ‣ Use water delivered as a fine spray to control fire and cool adjacent 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	‣ Combustible solid which burns but propagates flame with difficulty; it is estimated that most organic dusts are combustible (circa 70%) - according to the circumstances under which the combustion process occurs, such materials may cause fires and / or dust explosions. ‣ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions). ‣ Avoid generating dust, particularly clouds of dust in a confined or unventilated space as dusts may form an explosive mixture with air, and any source of ignition, i.e. flame or spark, will cause fire or explosion. Dust clouds generated by the fine grinding

	of the solid are a particular hazard; accumulations of fine dust (420 micron or less) may burn rapidly and fiercely if ignited - particles exceeding this limit will generally not form flammable dust clouds; once initiated, however, larger particles up to 1400 microns diameter will contribute to the propagation of an explosion. ▸ In the same way as gases and vapours, dusts in the form of a cloud are only ignitable over a range of concentrations; in principle, the concepts of lower explosive limit (LEL) and upper explosive limit (UEL) are applicable to dust clouds but only the LEL is of practical use; - this is because of the inherent difficulty of achieving homogeneous dust clouds at high temperatures (for dusts the LEL is often called the "Minimum Explosible Concentration", MEC). ▸ When processed with flammable liquids/vapors/mists,ignitable (hybrid) mixtures may be formed with combustible dusts. Ignitable mixtures will increase the rate of explosion pressure rise and the Minimum Ignition Energy (the minimum amount of energy required to ignite dust clouds - MIE) will be lower than the pure dust in air mixture. The Lower Explosive Limit (LEL) of the vapour/dust mixture will be lower than the individual LELs for the vapors/mists or dusts. ▸ A dust explosion may release of large quantities of gaseous products; this in turn creates a subsequent pressure rise of explosive force capable of damaging plant and buildings and injuring people. ▸ Usually the initial or primary explosion takes place in a confined space such as plant or machinery, and can be of sufficient force to damage or rupture the plant. If the shock wave from the primary explosion enters the surrounding area, it will disturb any settled dust layers, forming a second dust cloud, and often initiate a much larger secondary explosion. All large scale explosions have resulted from chain reactions of this type. ▸ Dry dust can be charged electrostatically by turbulence, pneumatic transport, pouring, in exhaust ducts and during transport. ▸ Build-up of electrostatic charge may be prevented by bonding and grounding. ▸ Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting. ▸ All movable parts coming in contact with this material should have a speed of less than 1-meter/sec. ▸ A sudden release of statically charged materials from storage or process equipment, particularly at elevated temperatures and/ or pressure, may result in ignition especially in the absence of an apparent ignition source. ▸ One important effect of the particulate nature of powders is that the surface area and surface structure (and often moisture content) can vary widely from sample to sample, depending of how the powder was manufactured and handled; this means that it is virtually impossible to use flammability data published in the literature for dusts (in contrast to that published for gases and vapours). ▸ Autoignition temperatures are often quoted for dust clouds (minimum ignition temperature (MIT)) and dust layers (layer ignition temperature (LIT)); LIT generally falls as the thickness of the layer increases. Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) hydrogen chloride phosgene other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	Not Applicable

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 waste regularly and abnormal spills immediately. ▸ Avoid breathing dust and contact with skin and eyes. ▸ Wear protective clothing, gloves, safety glasses and dust respirator. ▸ Use dry clean up procedures and avoid generating dust. ▸ Vacuum up or sweep up. **NOTE:** Vacuum cleaner must be fitted with an exhaust micro filter (HEPA type) (consider explosion-proof machines designed to be grounded during storage and use). ▸ Dampen with water to prevent dusting before sweeping. ▸ Place in suitable containers for disposal. Environmental hazard - contain spillage.
Major Spills	Environmental hazard - contain spillage. Moderate hazard. ▸ **CAUTION**: Advise personnel in area. ▸ Alert Emergency Services and tell them location and nature of hazard. ▸ Control personal contact by wearing protective clothing. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Recover product wherever possible. ▸ **IF DRY:** Use dry clean up procedures and avoid generating dust. Collect residues and place in sealed plastic bags or other

	containers for disposal. **IF WET:** Vacuum/shovel up and place in labelled containers for disposal. ▸ **ALWAYS:** Wash area down with large amounts of water and prevent runoff into drains. ▸ 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

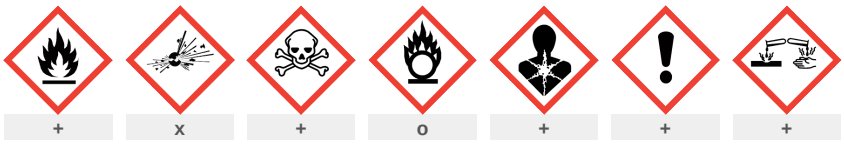
Precautions for safe handling

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. ▸ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions) ▸ Minimise airborne dust and eliminate all ignition sources. Keep away from heat, hot surfaces, sparks, and flame. ▸ Establish good housekeeping practices. ▸ Remove dust accumulations on a regular basis by vacuuming or gentle sweeping to avoid creating dust clouds. ▸ Use continuous suction at points of dust generation to capture and minimise the accumulation of dusts. Particular attention should be given to overhead and hidden horizontal surfaces to minimise the probability of a "secondary" explosion. According to NFPA Standard 654, dust layers 1/32 in.(0.8 mm) thick can be sufficient to warrant immediate cleaning of the area. ▸ Do not use air hoses for cleaning. ▸ Minimise dry sweeping to avoid generation of dust clouds. Vacuum dust-accumulating surfaces and remove to a chemical disposal area. Vacuums with explosion-proof motors should be used. ▸ Control sources of static electricity. Dusts or their packages may accumulate static charges, and static discharge can be a source of ignition. ▸ Solids handling systems must be designed in accordance with applicable standards (e.g. NFPA including 654 and 77) and other national guidance. ▸ Do not empty directly into flammable solvents or in the presence of flammable vapors. ▸ The operator, the packaging container and all equipment must be grounded with electrical bonding and grounding systems. Plastic bags and plastics cannot be grounded, and antistatic bags do not completely protect against development of static charges. Empty containers may contain residual dust which has the potential to accumulate following settling. Such dusts may explode in the presence of an appropriate ignition source. ▸ Do NOT cut, drill, grind or weld such containers. ▸ In addition ensure such activity is not performed near full, partially empty or empty containers without appropriate workplace safety authorisation or permit.
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry area protected from environmental extremes. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. For major quantities: ▸ Consider storage in bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams}. ▸ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Avoid contact with aluminium and its alloys (including storage containers). Formation of aluminium chloride may catalyse further self-accelerating attack on the metal (Friedel-Crafts reaction) leading to violent explosion. ▸ Glass container is suitable for laboratory quantities ▸ **DO NOT** use aluminium or galvanised containers

	‣ Polyethylene or polypropylene container. ‣ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Haloaryl compounds (halogenated aromatics), though normally not very reactive, may be sufficiently activated by other substituents or by a few specific reaction conditions, to undergo violent reactions. BRETHERICK L.: Handbook of Reactive Chemical Hazards ‣ Avoid contact with aluminium and its alloys (including storage containers). Formation of aluminium chloride may catalyse further self-accelerating attack on the metal (Friedel-Crafts reaction) leading to violent explosion. ‣ Avoid reaction with oxidising agents



+	x	+	o	+	+	+

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
Australia Exposure Standards	1,4-dichlorobenzene	p-Dichlorobenzene	25 ppm / 150 mg/m3	300 mg/m3 / 50 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
1,4-dichlorobenzene	30 ppm	170 ppm	1,000 ppm

Ingredient	Original IDLH	Revised IDLH
1,4-dichlorobenzene	150 ppm	Not Available

MATERIAL DATA

It is the goal of the ACGIH (and other Agencies) to recommend TLVs (or their equivalent) for all substances for which there is evidence of health effects at airborne concentrations encountered in the workplace.
At this time no TLV has been established, even though this material may produce adverse health effects (as evidenced in animal experiments or clinical experience). Airborne concentrations must be maintained as low as is practically possible and occupational exposure must be kept to a minimum.
NOTE: The ACGIH occupational exposure standard for Particles Not Otherwise Specified (P.N.O.S) does NOT apply.
Sensory irritants are chemicals that produce temporary and undesirable side-effects on the eyes, nose or throat. Historically occupational exposure standards for these irritants have been based on observation of workers' responses to various airborne concentrations. Present day expectations require that nearly every individual should be protected against even minor sensory irritation and exposure standards are established using uncertainty factors or safety factors of 5 to 10 or more. On occasion animal no-observable-effect-levels (NOEL) are used to determine these limits where human results are unavailable. An additional approach, typically used by the TLV committee (USA) in determining respiratory standards for this group of chemicals, has been to assign ceiling values (TLV C) to rapidly acting irritants and to assign short-term exposure limits (TLV STELs) when the weight of evidence from irritation, bioaccumulation and other endpoints combine to warrant such a limit. In contrast the MAK Commission (Germany) uses a five-category system based on intensive odour, local irritation, and elimination half-life. However this system is being replaced to be consistent with the European Union (EU) Scientific Committee for Occupational Exposure Limits (SCOEL); this is more closely allied to that of the USA.
OSHA (USA) concluded that exposure to sensory irritants can:
‣ cause inflammation
‣ cause increased susceptibility to other irritants and infectious agents
‣ lead to permanent injury or dysfunction
‣ permit greater absorption of hazardous substances and
‣ acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.

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)

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.

- Local exhaust ventilation is required where solids are handled as powders or crystals; even when particulates are relatively large, a certain proportion will be powdered by mutual friction.
- Exhaust ventilation should be designed to prevent accumulation and recirculation of particulates in the workplace.
- If in spite of local exhaust an adverse concentration of the substance in air could occur, respiratory protection should be considered. Such protection might consist of:

(a): particle dust respirators, if necessary, combined with an absorption cartridge;
(b): filter respirators with absorption cartridge or canister of the right type;
(c): fresh-air hoods or masks

- Build-up of electrostatic charge on the dust particle, may be prevented by bonding and grounding.
- Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting.

Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to efficiently remove the contaminant.

Type of Contaminant:	Air Speed:
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 ft/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 ft/min)

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. 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 4-10 m/s (800-2000 ft/min) for extraction of crusher dusts generated 2 metres 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

NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

	The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades 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. Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present. ‣ polychloroprene. ‣ nitrile rubber. ‣ butyl rubber. ‣ fluorocaoutchouc. ‣ polyvinyl chloride. Gloves should be examined for wear and/ or degradation constantly.
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit.

Respiratory protection

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	P1 Air-line*	- -	PAPR-P1 -
up to 50 x ES	Air-line**	P2	PAPR-P2
up to 100 x ES	-	P3	-
		Air-line*	-
100+ x ES	-	Air-line**	PAPR-P3

* - Negative pressure demand ** - Continuous flow
A(All classes) = Organic vapours, 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)

· Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
· The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
· Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
· Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
· Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
· Use approved positive flow mask if significant quantities of dust becomes airborne.
· Try to avoid creating dust conditions.
Class P2 particulate filters are used for protection against mechanically and thermally generated particulates or both.
P2 is a respiratory filter rating under various international standards, Filters at least 94% of airborne particles
Suitable for:
· Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
· Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
· Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Not Available

Physical state	Solid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour 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	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (1%)**	Not Available
Vapour 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. Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further disability if excessive concentrations of particulate are inhaled. If prior damage to the circulatory or nervous systems has occurred or if kidney damage has been sustained, proper screenings should be conducted on individuals who may be exposed to further risk if handling and use of the material result in excessive exposures. The physiological response to 1,4-dichlorobenzene (DCB)is primarily injury to the liver and secondarily to the kidneys. Central nervous system depression will occur at concentrations that are extremely objectionable to the eyes and nose. Individuals exposed to higher concentrations may show weakness, dizziness and weight loss. Vomiting may occur. Acute haemolytic anaemia with methaemoglobinaemia has been reported. Prolonged inhalation exposure may cause dizziness, headache nausea, vomiting, central nervous system depression and damage to liver and kidneys. In two human fatalities believed to be caused by 1,4-DCB inhalation, the subjects died of massive hepatic (liver) necrosis; the exposure concentrations are not known. A 3 year-old child who had been playing with crystals containing 1,4-DCB for 4-5 days was jaundiced with pale mucous membranes, indicative of liver damage. A case of pulmonary granulomatosis was reported to have occurred in a 53-year-old woman who for 12-15 years had been inhaling 1,4-DCB crystals that were scattered on a weekly basis on the carpets and furniture of her home. A lung biopsy revealed the presence of 1,4-DCB crystals with the surrounding lung parenchyma being distorted (by fibrosis, thickening of the alveolar walls, and marked infiltrates of lymphocytes and mononuclear phagocytes). These effects are most likely related to the physical interaction of 1,4-DCB crystals (or any crystals when inhaled) with lung tissue, rather than to chemical toxicity. A health survey of 58 men occupationally exposed to 1,4-DCB for 8 hours/day, 5 days/week for 8 months to 25 years (average, 4.75 years) found the odor to be faint at 15-30 ppm and strong at 30-60 ppm, with painful irritation of the nose and eyes usually occurring at concentrations ranging from 80 to 160 ppm. At levels >160 ppm, the air was considered not breathable for unacclimated persons. Rabbits exposed 8 hours/day for a total of 62 exposures in 83 days at 770-800 ppm exhibited tremors, weakness, and death along with oedema of the cornea and opacity of the lens. In male mice exposed to 1,2-DCB in mean concentrations of 0, 64, or 163 ppm for 6 hours/day, 5 days/week for 4, 9, or 14 days, histopathologic lesions were observed in the olfactory epithelium of the nasal cavity at >64 ppm. The olfactory epithelial lesions were graded as very severe following the 4-day exposure and moderate after the 14 day exposure, indicating to the study authors that repair may occur despite continued exposure. The more severe cases were characterized by a complete loss of olfactory epithelium, which left only partially denuded basement membrane. No histological alterations were observed in the respiratory epithelium of the nasal cavity, or in the trachea or lungs. Mouse exposed to the saturated vapour (calculated as between 2000 and 3000 ppm) showed prompt narcosis, followed by central respiratory depression and cyanosis - death occurred within 24 hours. 8000 ppm produced sedation in dogs exposed for 1 hour. Rats exposed at a concentration of 450 ppm, 6 hours/day for up to 13 days showed pale, discoloured kidneys Rats survived inhalation exposure for 2 hours at 977 ppm but died after 7 hour exposure. Rats surviving a 7 hour exposure at 539 ppm showed liver necrosis and kidney tubule damage. Liver damage was evident in other rats exposed from 50 to 800 ppm and during exposures lasting 0.5 and 1 hour at 390 ppm. Following a single or multiple 3-hour inhalation exposures of radiolabelled 1,4-DCB in rats, label was detected in all evaluated tissues (liver, kidneys, lungs, muscle, fat, and blood plasma), indicating that considerable absorption had occurred. Levels of label in tissues did not appreciably increase with increasing the number of exposures beyond one. Similarly, following a single 24-hour inhalation exposure in rats, 1,4-DCB levels in the liver, kidney, fat, and blood increased sharply during the first 6-hour evaluation period, then rose slowly but steadily for the remainder of the exposure period, indicating an initial rapid absorption, followed by a slower total absorption as equilibration of body and blood levels is approached.

Ingestion	The substance and/or its metabolites may bind to haemoglobin inhibiting normal uptake of oxygen. This condition, known as "methaemoglobinemia", is a form of oxygen starvation (anoxia). Symptoms include cyanosis (a bluish discolouration skin and mucous membranes) and breathing difficulties. Symptoms may not be evident until several hours after exposure. At about 15% concentration of blood methaemoglobin there is observable cyanosis of the lips, nose and earlobes. Symptoms may be absent although euphoria, flushed face and headache are commonly experienced. At 25-40%, cyanosis is marked but little disability occurs other than that produced on physical exertion. At 40-60%, symptoms include weakness, dizziness, lightheadedness, increasingly severe headache, ataxia, rapid shallow respiration, drowsiness, nausea, vomiting, confusion, lethargy and stupor. Above 60% symptoms include dyspnea, respiratory depression, tachycardia or bradycardia, and convulsions. Levels exceeding 70% may be fatal. 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. Acute-, intermediate,- and chronic-duration inhalation and oral studies of dichlorobenzenes (DCBs) clearly identify the liver as a sensitive target of oral exposure, inducing increases in liver weight at low levels of exposure and histological changes such as cloudy swelling and centrilobular degeneration and necrosis at higher levels in rats and mice. In rats exposed to 455 mg/kg/day for 15 days 1,2-DCB, severe liver damage, characterised by intense necrosis and fatty changes and porphyria, were reported. Large doses have caused tremor in exposed animals; insects exhibit symptoms resembling DDT poisoning. 1,2-DCB is a strong central nervous system depressant. 1,2-DCB is quickly and extensively absorbed through both the gastrointestinal tract and the respiratory tract; studies measuring the absorption of 1,2-DCB following dermal exposure are not available. Following absorption, 1,2-dichlorobenzene (1,2-DCB) is distributed throughout the body, but tends to be found in greatest levels in the fat, kidney, and liver. Metabolism is believed to occur mainly in the liver, but may occur at lower levels in other tissues, such as the kidney or lung. Elimination of 1,2-DCB from the body is rapid, with the majority of a single dose being removed within the first 75 hours postexposure; elimination occurs primarily in the urine as metabolites Information on the oral toxicity of 1,3-DCB in animals is available from one 90-day systemic toxicity study and one developmental toxicity study. The intermediate-duration study found effects in the thyroid, pituitary, and liver of rats, with thyroid lesions occurring at dose levels lower than those inducing pituitary and liver effects. Hepatic porphyria was produced in rats following seven consecutive doses of 770 mg 1,4-DCB/kg. Slight to moderate corneal opacity was noted in rabbits following 3 weeks of daily dosing with 5000 mg/kg 1,4-DCB. Rats receiving a daily dose of 500 mg/kg 1,4-DCB for 20 days showed cloudy swelling and necrosis in the central areas of the liver lobules and swelling of the renal tubular epithelium. 100 mg/kg daily doses did not reproduce this finding. Pale and mottled kidneys were seen in rats given oral doses of 70 to 428 mg/kg/day, 1,4-DCB for 28 days. Rats given 1200 mg/kg for 13 weeks showed degeneration and necrosis of hepatocytes, hypoplasia of the bone marrow, lymphoid depletion of the spleen and thymus, and epithelial necrosis of the nasal turbinates and small intestinal mucosa. At doses of 300 mg/kg 1,4-DCB male rats showed kidney damage characterised by degeneration or necrosis of the renal cortical tubular epithelial cells. Female rats did not show these lesions even at doses of 1500 mg/kg Oral doses of 500 mg 1,2-DCB given over 13- weeks to mice and rats produced necrosis and hepatocellular degeneration and depletion of lymphocytes in both the spleen and thymus and renal tubular degeneration in rats. Multifocal mineralisation of the myocardial fibers of the heart and skeletal muscle was seen in mice. Necrosis of individual hepatocytes was seen in female mice given 250 mg/kg. At 125 mg/kg a few rats exhibited minimal hepatocellular necrosis. Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. 1,2-dichlorobenzene (DCB) was irritating when applied to the skin of human subjects for 15-60 minutes. One worker developed a dermatitis following hand contact that was reported as sensitisation after a follow-up patch test. Two subjects reported a burning sensation during a 1 hour exposure. A diffuse redness of the treated area progressed to a darker red colour with blister formation within 24 hours. A brown pigment formed at the site which was apparent 3 months postexposure 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. Absorption by skin may readily exceed vapour inhalation exposure. Symptoms for skin absorption are the same as for inhalation.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

	Undiluted 1,2-dichlorobenzene (DCB) applied to rabbit eye caused pain and slight conjunctival irritation. Irritation cleared within 5 days without residual injury. Vapours from heated 1,4-DCB may cause mild corneal damage. Solid particles of 1,4-CB in the eye are reported to be very painful. At workplace concentrations ranging from 50-170 ppm 1,4-DCB, periodic medical examination found no evidence of adverse effects in workers with particular reference to ocular lesions including cataracts. Painful irritation of eyes and nose has been recorded at 80-160 ppm 1,4-DCB
Chronic	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Chronic inhalation exposure to dichlorobenzenes (DCBs) may cause changes to liver and kidney and haematological (blood) disorders. There is some evidence to suggest a link between leukaemia and exposure to dichlorobenzenes. [NIOSHTIC]. Workers who were chronically exposed to 1,4-DCB vapor experienced irritation of the nose and eyes and case reports of people who inhaled or ingested 1,4-DCB suggest that the liver, nervous system, and haematopoietic system are systemic targets in humans. The available limited information on these systemic effects in humans is consistent with findings in animals exposed to 1,4-DCB. In individuals exposed chronically to 1,4-DCB, liver effects including jaundice, cirrhosis, and possible death may occur. Chronic exposure may also produce weakness, headache, rhinitis, twitching of the facial muscles. A woman who consumed 4 to 5 moth ball pellets daily for 2.5 years developed unsteady gait, tremors of the hand and general mental sluggishness which disappeared 4 months after exposure ceased. Eight workers manufacturing 1,4-DCB based moth-proofing agents for 1 to 7 months developed neural disorders including intensified muscle reflexes, mild clonus of the ankle and tremors of the fingers. They reported loss of appetite and haemopoietic changes. An evaluation of 953 adult participants in the Third National Health and Nutrition Examination Survey of the general U.S. population found that exposure to 1,4-DCB may possibly contribute to decreases in lung function. Little human data is available about developmental effects. A 21-year-old woman who had eaten 1-2 blocks of 1,4-DCB toilet freshener per week for the first 38 weeks of pregnancy gave birth to an apparently normal child. Rats treated 1,4-DCB for 2 years, by gastric intubation, showed kidney lesion and in the male, hyperplasia of the thyroid at dose rates of 150 mg/kg. Mice treated with 300 mg/kg 1,4-DCB, in a similar 2 year gavage study, showed liver changes characterised by hepatocellular degeneration. Thyroid follicular cell hyperplasia was increased in male but not female mice. Nephropathy consisting primarily of degeneration of the cortical tubular epithelium was seen and was more pronounced in males. Rats, guinea pigs, rabbits, mice and monkeys exposed by inhalation to 1,4-DCB, 7 hours/day, 5 days/week for 140 exposures at 800 ppm exhibited tremor, weight loss and liver changes, including swelling and central necrosis in female rats, and swelling of the kidney epithelium. A 2 year study with rats and mice treated with oral doses of 1,2-DCB at either 60 or 120 mg 5 days/ week produced a lower survival time of male rats receiving the higher dose. An increase in the incidence of tubular regeneration in the male mouse kidney was the only compound-related, non-neoplastic, histologic lesion observed and no evidence of carcinogenicity was seen during the study In rabbits exposed to 300 ppm, but not those exposed to 800 ppm, there was a significant increase in the number of resorptions and the percentages of resorbed implantations per litter; the fact that the effect did not occur in the rabbits exposed to the higher exposure level suggests that it was not treatment-related. A 2-generation oral study in rats found toxicity in the offspring at doses .90 mg/kg/day; effects included reduced birth weight in F1 pups, increased mortality on postnatal day 4 in F1 and F2 pups,

	clinical manifestations of dry and scaly skin (until approximately postnatal day 7) in F1 and F2 pups, and reduced neurobehavioral performance (draw-up reflex evaluated at weaning) in F2 pups. No exposure-related changes occurred at 30 mg/kg/day. Other evaluations of developmental effects of 1,4-DCB following oral exposure have been negative. Data on the carcinogenic effects of 1,4-DCB in humans are not available. Four cases involving cancer and exposure to 1,2-DCB have been reported. These involved the development of peripheral leukoblastosis, chronic lymphoid leukaemia and myeloblastic leukaemia. 1,4-DCB has been shown to be carcinogenic in chronic animal studies by both the inhalation and oral routes. Following lifetime oral exposure, hepatic tumors (hepatocellular adenomas and carcinomas and histiocytic sarcomas) were increased in mice of both sexes, but not in either sex of rats. The oral bioassay also found that the male rats exposed to 1,4-DCB developed renal tubular cell adenocarcinomas, but these are believed to be the result of interaction with a2u-globulin, a renal protein not present in humans. Data on the possible carcinogenic effects of 1,4-DCB following dermal exposure are not available. An increase in liver tumours (e.g. renal tubular cell adenocarcinomas) was seen in male rats treated with 1,4-DCB, by gastric intubation doses of 150 mg/kg for 2 years. No evidence of carcinogenicity was seen in female rats. An increase incidence of hepatocellular carcinomas and adenomas was seen in mice treated with gavage doses of 300 mg/kg/day for 2 years. A positive dose-trend for adrenal gland pheochromocytomas in male mice was also reported. .

Refresher Tabs	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
1,4-dichlorobenzene	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (human): 80 ppm
	Inhalation(Rat) LC50: >5.07 mg/l4h[1]	
	Oral (Rat) LD50; 500 mg/kg[2]	
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*	

Refresher Tabs	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. 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 oedema. 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.
1,4-DICHLOROBENZENE	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*] Eye effects, respiratory tract changes, diarrhoea, specific developmental effects (cardiovascular system) recorded.
Refresher Tabs & 1,4-DICHLOROBENZENE	During the manufacture and use of chlorobenzenes, clinical symptoms and signs of excessive exposure include: central nervous system effects and irritation of the eyes and upper respiratory tract (MCB); haematological disorders (1,2-DCB); and central nervous system effects, hardening of the skin, and haematological disorders including anaemia (1,4-DCB). All chlorobenzenes appear to be absorbed readily from the gastrointestinal and respiratory tracts in humans and experimental animals, with absorption influenced by the position of the chlorine in different isomers of the same congener. The chlorobenzenes are less readily absorbed through the skin. After rapid distribution to highly perfused organs in experimental animals, absorbed chlorobenzenes accumulate primarily in the fatty tissue, with smaller amounts in the liver and other organs. Chlorobenzenes have been shown to cross the placenta, and have been found in the foetal brain. In general, accumulation is greater for the more highly chlorinated congeners. There is considerable variation, however, in the accumulation of different isomers of the same congener. In both humans and experimental animals, the metabolism of chlorobenzenes proceeds via microsomal oxidation to the corresponding chlorophenol. These chlorophenols can be excreted in the urine as mercapturic acids, or as glucuronic acid or sulfate conjugates. Tetrachlorobenzenes (TeCB) and pentachlorobenzene (PeCB) are metabolized at a slower rate and remain in the tissues for longer periods than the monochloro- to trichloro- congeners. Some of the chlorobenzenes induce a wide range of enzyme systems including those involved in oxidative, reductive, conjugation, and hydrolytic pathways. In general, elimination of

	the higher chlorinated benzenes is slower than that of the MCB and DCB congeners, and a greater proportion of the tri- to penta-congeners are eliminated unchanged in the faeces. With few exceptions, the chlorobenzenes are only moderately toxic for experimental animals, on an acute basis, and, generally, have oral LD50s greater than 1000 mg/kg body weight; from the limited data available, dermal LD50s are higher. The ingestion of a lethal dose leads to respiratory paralysis, while the inhalation of high doses causes local irritation and depression of the central nervous system. Acute exposures to non-lethal doses of chlorobenzenes induce toxic effects on the liver, kidneys, adrenal glands, mucous membranes, and brain, and effects on metabolizing enzymes. Studies on skin and eye irritation caused by chlorobenzenes have been restricted to 1,2,4-TCB and 1,2-DCB. Both produce severe discomfort, but no permanent damage was noted after direct application to the rabbit eye. 1,2,4-TCB is mildly irritating to the skin and may lead to dermatitis after repeated or prolonged contact. No evidence of sensitization was found. Short-term exposures (5-21 days) of rats and mice to MCB and DCBs at hundreds of mg/kg body weight resulted in liver damage and haematological changes indicative of bone marrow damage. Liver damage was also the major adverse effect noted after the short-term exposure of rats or rabbits to other chlorobenzenes (TCB-PeCB), at doses slightly lower than those for MCB and DCBs. Several of the chlorobenzene isomers studied induced porphyria, the isomers with *para* chlorine atoms being the most active (i.e., 1,4-DCB, 1,2,4-TCB, 1,2,3,,4-TeCB, and PeCB). The general order of toxicity noted for TeCBs and PeCB after short-term exposure was: 1,2,4,5-TeCB >PeCB>1,2,3,4- and 1,2,3,5-TeCB, which correlated well with the levels found in fat and liver. Long-term exposure studies (up to 6 months) on several species of experimental animals indicated a trend for the toxicity of chlorobenzenes to increase with increased ring chlorination. However, there was considerable variation in the long-term toxicities of different isomers of the same congener. For example, 1,4-DCB appeared to be much less toxic than 1,2-DCB. There was a good correlation between toxicity and the degree of accumulation of the compound in the body tissues, female animals being less sensitive than males. Major target organs were the liver and kidney; at higher doses, effects on the haematopoietic system were reported and thyroid toxicity was noted in studies on 1,2,4,5-TeCB and PeCB. There has been no evidence that chlorobenzenes are teratogenic in rats and rabbits. High doses produce embryotoxic and fetotoxic effects. However, such doses were clearly toxic to the mother. Although there is some evidence that TCBs, TeCBs, and PeCB are embryotoxic and fetotoxic at doses that are not toxic for the mother, available data are inconsistent. 1,2-DCB is quickly and extensively absorbed through both the gastrointestinal tract and the respiratory tract; studies describing the absorption of 1,2-DCB following dermal exposure are not available. Following absorption, 1,2-DCB is distributed throughout the body, but tends to be found in greatest levels in the fat, kidney, and liver. 1,2-DCB is initially metabolized by cytochrome P-450 enzymes, specifically P4502E1, to an active epoxide followed by hydrolysis to 2,3-dichlorophenol or 3,4-dichlorophenol. The dichlorophenols may be further oxidised or, more often, be conjugated to glutathione, sulfate, or to form the glucuronide; conjugation occurs extensively, with virtually no unconjugated metabolites reported in the available studies. Metabolism is believed to occur mainly in the liver, but may occur at lower levels in other tissues, such as the kidney or lung. Elimination of 1,2-DCB from the body is rapid, with the majority of a single dose being removed within the first 75 hours postexposure; elimination occurs primarily in the urine as metabolites. Absorption of 1,3-DCB can be inferred from studies that have detected 1,3-DCB or metabolites in the breast milk, blood, and fat of humans and in the bile and urine of exposed animals. Distribution is believed to be similar to the other DCB isomers. Similar to the other DCB isomers, 1,3-DCB is initially metabolised by cytochrome P-450 enzymes, followed by extensive conjugation, primarily to glutathione, has been reported. 1,3-DCB is eliminated mainly in the urine, similar to the other DCB isomers. Absorption of 1,4-DCB is rapid and essentially complete following inhalation or oral exposure. Dermal absorption is believed to be very low, based on a very high (>6 g/kg) dermal LD50 for 1,4-DCB in rats, and on a lack of systemic effects in humans who held solid 1,4-DCB in their hands. Similar to the other dichlorobenzene isomers, 1,4-DCB is distributed throughout the body, but tends to be found in greatest levels in fat, liver, and kidney. Metabolism of 1,4-DCB is similar to that of 1,2-DCB, with an initial oxidation to an epoxide, followed by hydrolysis to 2,5-dichlorophenol. Extensive phase II metabolism occurs subsequently, with eliminated metabolites found mainly as the sulfate, glucuronide, or mercapturic acid. 1,4-DCB is eliminated almost exclusively in the urine, primarily as conjugates of 2,5-dichlorophenol.

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

Refresher Tabs

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

1,4-dichlorobenzene

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	31mg/l	2
BCF	840h	Fish	33-72	7

	EC50	48h	Crustacea	0.7mg/l	2
	EC50(ECx)	24h	Algae or other aquatic plants	<0.001mg/L	4
	LC50	96h	Fish	1mg/l	4
	EC50	96h	Algae or other aquatic plants	1.6mg/L	5
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*				

Very toxic 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.
Chlorobenzenes are removed from the environment principally by biological, and, to a lesser extent, by non-biological mechanisms; however, they are considered moderately persistent in water, air, and sediments. Residence times in water of 1 day in rivers and over 100 days in ground water have been reported. In air, chemical and photolytic reactions are presumed to be the predominant pathways for chlorobenzene degradation, with residence times in the range of 13-116 days.
Many microorganisms from sediments and sewage sludge have been shown to degrade chlorobenzenes. It would appear that the higher chlorinated compounds are less readily degraded, and such degradation occurs only under aerobic conditions. Under anaerobic conditions in soil and ground water, dichlorobenzenes (DCB), trichlorobenzenes (TCBs), and pentachlorobenzenes (PeCBs) are usually resistant to microbial degradation. The major mechanism of aerobic degradation is via oxidative dechlorination, usually initiated by dioxygenative hydroxylation, leading to the formation of hydroxylated aromatic compounds (mainly catechols), which undergo ring fission and subsequent mineralisation to carbon dioxide and water.
Chlorobenzenes as a family can persist in soil for several months, in air for 3.5 days and in water for 1 day or less. Chlorobenzenes released into water environments will evaporate form water to the atmosphere.
Henry's Law constant, the soil sorption constant, and the octanol-water (Kow) constant predict movement and fate in the environment. The distribution from water to air will decrease with increasing chlorination of the compound. Ninety-six percent of monochlorobenzene (MCB) is released to the atmosphere from aquatic environments. In other studies 99% of MCB, 1,2-dichlorobenzene (DCB) and 1,2,4-trichlorobenzene (TCB) evaporated from water solutions within 4 hours.
Chlorobenzenes released into the aquatic environment will be redistributed preferentially to the air and to sediment (particularly organically rich sediments).
Limited information has shown that levels 1000 times those found in water have been detected in sediments, particularly in highly industrialised regions. Retention of chlorobenzenes in soil increases with the organic content of the soil; there is a positive correlation between the degree of chlorination of the compound and its adsorption on organic matter. Limited evidence is available showing that sediment-bound residues are bioavailable to organisms; i.e., aquatic invertebrates can take up residues from sediment, and plants, from soil.
There is some indication that concentrations of chlorobenzenes in freshwater fish increase with increasing degree of chlorination of the compound. The few studies available indicate levels of 1,4-DCB in some marine fish of 0.05 mg/kg (wet weight). In the available studies on chlorobenzene levels in meat and milk, limited primarily to samples from contaminated areas, concentrations of 0.02-5 ug/kg have been reported.Available information on the effects of chlorobenzenes on the environment is mainly focused on acute effects on aquatic organisms. In general, toxicity increases with the degree of chlorination of the benzene ring.
While MCB, 1,2-DCB, 1,3-DCB, 1,2,4-TCB, 1,3,5-TCB, and 1,2,4,5-TeCB all exhibit a low toxicity for microorganisms, the toxicity of the TCBs and TeCBs is, with the exception of 1,2,4,5-TeCB, slightly higher than that of the other compounds; in unicellular aquatic algae, EC50 values for 96-h cell growth or chlorophyll a production ranged from over 300 mg/litre for MCB to approximately 1 mg/litre for 1,2,3,5-TeCB. Some aquatic invertebrates appear more sensitive to chlorobenzenes, but levels required for 48- or 96-h lethality are still near, or well above, 1 mg/litre (e.g., *Daphnia magna* at 2.4 mg/litre for 1,2-DCB, and up to 530 mg/litre for 1,2,4,5-TeCB). The 96-h LC50 for bluegill sunfish ranged between 0.3 mg/litre for PeCB and 24 mg/litre for MCB. In embryo-larval assays, the chronic toxicity limits for DCBs varied between 0.76 and 2.0 mg/litre for the fathead minnow; in the estuarine sheepshead minnow, the chronic toxicity limits for 1,2,4-TCB and 1,2,4,5-TeCB were 0.22 and 0.13 mg/litre, respectively. Newly-hatched goldfish and large-mouth bass were the most susceptible life-stage with LC50s (96-h) of 1 and 0.05 mg/litre, respectively, for MCB.
The bioaccumulation of chlorobenzenes by aquatic organisms is determined by their relative water and lipid solubility (thus reflecting the octanol/water partition coefficients) and the number of chlorine substitutions. Uptake from water increases with increasing chlorination. The rate of elimination of chlorobenzenes decreases with increasing chlorination. BCFs ranging from 270 for 1,2-DCB to 20 000 for PeCB were reported for laboratory studies on rainbow trout (*Oncorhynchus mykiss*). BCFs for a variety of fish species ranged from 7000 to 24000 (lipid weight) for 1,2,4-TCB, with a positive correlation between bioaccumulation and lipid content.
However, the prediction of BCFs is more difficult for terrestrial plants than for aquatic organisms because of the complex nature of the root soil interface combined with gaseous uptake by aerial parts .
A negative correlation was demonstrated between the BCF and the soil adsorption coefficient (based on soil organic matter content) for the uptake into the roots of barley. The adsorption of chlorobenzenes onto soil organic matter increased with increasing chlorination. However, expression of uptake in barley roots in relation to the soil interstitial water concentration of the chlorobenzenes produced a positive correlation between the BCF and the octanol/water partition coefficients.
Higher chlorinated chlorobenzenes, therefore, are most readily taken up by the plant roots when they are available in soil interstitial water. This will occur particularly in sandy soils with low organic matter content.
Reduction of the widespread use and disposal of chlorobenzenes should, however, be considered because:

- Chlorobenzenes may act as precursors for the formation of polychlorinated dibenzodioxins/polychlorinated dibenzofurans (PCDDs/PCDFs), e.g., in incineration processes.
- These chemicals can lead to taste and odour problems in drinking-water and fish.
- Residues persist in organically-rich anaerobic sediments and soils, and ground water.

Whereas 1,2- and 1,3-dichlorobenzene (DCB) are liquids at room temperature, 1,4-DCB is a solid that sublimes readily. Sublimation rates of 1,4-DCB from consumer products were measured at 1.6x10-3 to 4.6x10-3 g/minute at temperatures ranging from 21 to 24 °C during a 19-day test period. DCBs tend to volatilise to the atmosphere from soil and water at a relatively rapid rate. Volatilisation from surface soil may be an important transport mechanism for DCBs but adsorption to soil particulates may inhibit volatilisation.
Since DCBs are slightly soluble in water (80.0–156 mg/L) , partitioning to clouds, rain, or surface water may occur. Henry's Law constant values ranging from 1.74x10-3 to 2.63x10-3 atm-m3/mol at 25 °C indicate that partitioning from air to water is likely to be minor relative to the reverse process of volatilisation of the compound from water to air. These substances can be transported over long distances through the atmosphere.
Based on measured soil organic carbon partition coefficient (Koc) values, which range from 275 to 1,833 in different soils, DCBs are expected to sorb moderately to soils and sediments. Sorption is primarily to the soil organic phase and, therefore, depends on the organic content of the soil. However, sorption is likely to be reversible; therefore, DCBs may leach from hazardous waste sites and be transported to groundwater, or may migrate from surface water through the soil to groundwater. In a sandy soil with low organic matter, 26-49% of 1,4-DCB percolated through the soil to a depth of 140 cm. Transformation of DCBs by

biodegradation, photolysis, chemical hydrolysis, and oxidation appear to be relatively minor processes. Leaching of DCBs to groundwater from subsurface soils under certain conditions may occur.
DCBs are expected to bioconcentrate in aquatic organisms. High log octanol-water partition coefficient (log Kow) values of 3.43-3.53 also suggest that DCBs have a moderate to high potential for bioaccumulation. A calculated bioconcentration factor (BCF) of 267 was reported for the fathead minnow *(Pimephales promelas)*. Measured mean BCF values of 370 and 720 were experimentally determined at equilibrium for rainbow trout exposed to water concentrations of 28 ng/L (ppb) and 670 ng/L (ppb), respectively, of 1,4-DCB for up to 119 days in laboratory aquaria. BCF values measured in this study for 1,2-DCB were 270 (47 ng/L in water) and 560 (940 ng/L in water), while BCF values measured for 1,3-DCB were 420 (28 ng/L in water) and 740 (690 ng/L in water). A study of chlorobenzenes in sediments, water, and selected fish from the Great Lakes indicated that many chlorobenzenes are bioconcentrated by fish, but that DCBs are concentrated to a smaller extent than some of the more highly chlorinated chlorobenzene compounds such as pentachlorobenzene and hexachlorobenzene.
Chlorobenzenes are lipophilic and volatile compounds that can be taken up by plants by both root and foliage pathways. Carrots were grown for 100 days in control soil, chemically-spiked soil, and in low and high rate sludge-amended soils.Concentrations of 1,4-DCB in carrot foliage and the corresponding bioconcentration factors (BCFs) were 13 ppb (BCF=3.1) for the control, 17 ppb (BCF=1.3) for the spiked soil, 22 ppb (BCF=2.5) for the low rate sewage-amended soil, and 49 ppb (BCF=1.5) for the high rate sewage amended soils. The authors concluded that foliar uptake of all chlorobenzenes tested, including the DCBs, was an important bioaccumulation pathway.
The main degradation pathway for DCBs in air is reaction with photochemically generated hydroxyl radicals. Reactions with ozone or other common atmospheric species are not expected to be significant. Therefore, the atmospheric lifetime of the DCBs may be predicted from an assumed hydroxyl radical concentration in air and the rate of reaction. The reported rate for reaction of hydroxyl radicals with DCBs is 3.2-7.2x10-13 cm3/mol-sec), and the estimated atmospheric half-life for DCBs is about 14-31 days. Since this degradation process is relatively slow, DCBs may become widely dispersed, but are not likely to accumulate in the atmosphere. Reports of smog chamber studies of chlorobenzene degradation have indicated degradation after 5 hours of 21.5% of 1,2-DCB. Chloronitrobenzenes and chloronitrophenols were identified as degradation products. Irradiation of chlorobenzenes with natural sunlight was reported to produce polychlorinated biphenyls (PCBs). Whether this occurs under natural atmospheric conditions is unknown, but it would appear to be unlikely because of the normally low concentrations of chlorobenzenes in ambient air.
Biodegradation may be an important transformation process for DCBs in water under aerobic, but not anaerobic, conditions
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
1,4-dichlorobenzene	HIGH (Half-life = 360 days)	MEDIUM (Half-life = 83.58 days)

Bioaccumulative potential

Ingredient	Bioaccumulation
1,4-dichlorobenzene	LOW (BCF = 190)

Mobility in soil

Ingredient	Mobility
1,4-dichlorobenzene	LOW (KOC = 434)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	‣ Containers may still present a chemical hazard/ danger when empty. ‣ Return to supplier for reuse/ recycling if possible. Otherwise: ‣ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ‣ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ‣ **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.

SECTION 14 Transport information

Labels Required

Marine Pollutant	
HAZCHEM	Not Applicable

Land transport (ADG): 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
1,4-dichlorobenzene	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
1,4-dichlorobenzene	Not Available

SECTION 15 Regulatory information

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

1,4-dichlorobenzene is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
Australian Inventory of Industrial Chemicals (AIIC)	International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)
Chemical Footprint Project - Chemicals of High Concern List	

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (1,4-dichlorobenzene)
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	Yes
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	21/11/2022
Initial Date	21/11/2022

Other information

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: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour 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