NV Chemicals Refresher Tabs

N.V. Chemicals (Aust) P/L

Chemwatch Hazard Alert Code: **2**

Chemwatch: **28-2128**
Version No: **4.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **20/08/2021**
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L.GHS.AUS.EN.E

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

Product Identifier

Product name	NV Chemicals Refresher Tabs
Chemical Name	1,4-dichlorobenzene
Synonyms	Toilet Tabs, Toilet Deodorant Blocks
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains 1,4-dichlorobenzene)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Air freshener, toilet freshener, moth, mildew and insect repellant.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**N.V. Chemicals (Aust) P/L**
Address	24 Lisa Place Coolaroo VIC 3048 Australia
Telephone	+61 3 9351 1100
Fax	+61 3 9351 1077
Website	http://www.nvchemicals.com.au/
Email	info@nvchemicals.com.au

Emergency telephone number

Association / Organisation	**N.V.Chemicals(Aust) P/L**
Emergency telephone numbers	0411 387 097
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

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

Chemwatch Hazard Ratings

	Min	Max
Flammability	1	
Toxicity	2	
Body Contact	2	
Reactivity	1	
Chronic	2	

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	S5
Classification [1]	Acute Toxicity (Oral) Category 4, Serious Eye Damage/Eye Irritation Category 2A, Carcinogenicity Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

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Label elements

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

Hazard statement(s)

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

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	99.2	1,4-dichlorobenzene
Not Available	<1	perfume
Not Available	<0.1	dye
Legend:	*1. Classified by Chemwatch; 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.

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Ingestion	▸ Not considered a normal route of entry. ▸ For advice, contact a Poisons Information Centre or a doctor at once. ▸ Urgent hospital treatment is likely to be needed. ▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

EYES - Stain for evidence of corneal injury. SKIN - Treat as for dermatitis. RESPIRATION - Administer oxygen if available. The use of bronchodilators, expectorants and antitussives may help. There is no antidote for systemic effects. Readily absorbed after oral administration to rats and found in all organs with accumulation in adipose tissues. 90% of the dose is excreted within 48 hours. Two metabolites, 2,5-dichlorophenylmethylsulfone and 2,5-dichlorophenylsulfoxide are detected in the blood (though not the compound itself). Slow release from the adipose tissues is probably responsible for the persistence of these metabolites. 2,5-dichlorophenol is detected in plasma, urine, liver, kidneys and fatty tissues - in humans this metabolite is a useful monitor of exposure. An occupational exposure for 1 week to 7.4 ppm 1,4-DCB produced an increase of 1,4-DCB in the urine as a direct measurement and its use as a biological exposure index has been suggested
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 Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) hydrogen chloride phosgene other pyrolysis products typical of burning organic material.
HAZCHEM	2Z

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ▸ Clean up all spills immediately. ▸ Secure load if safe to do so. ▸ Bundle/collect recoverable product.

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	▸ Collect remaining material in containers with covers for disposal.
Major Spills	Environmental hazard - contain spillage. ▸ Clean up all spills immediately. ▸ Wear protective clothing, safety glasses, dust mask, gloves. ▸ Secure load if safe to do so. Bundle/collect recoverable product. ▸ Use dry clean up procedures and avoid generating dust. ▸ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use). ▸ Water may be used to prevent dusting. ▸ Collect remaining material in containers with covers for disposal. ▸ Flush spill area with water.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Limit all unnecessary personal contact. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	▸ Keep dry. ▸ Store under cover. ▸ Store in a well ventilated area. ▸ Store away from sources of heat or ignition. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	Carton.
Storage incompatibility	▸ 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

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

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. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed 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, vapours, degreasing etc., evaporating from tank (in still air)	0.25-0.5 m/s (50-100 f/min)

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	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 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 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.

Individual protection measures, such as personal protective equipment	
Eye and face protection	No special equipment for minor exposure i.e. when handling small quantities. OTHERWISE: ▸ Safety glasses with side shields. ▸ 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	No special equipment needed when handling small quantities. **OTHERWISE**: Wear chemical protective gloves, e.g. PVC.
Body protection	See Other protection below
Other protection	No special equipment needed when handling small quantities. **OTHERWISE:** ▸ Overalls. ▸ Barrier cream. ▸ Eyewash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
NV Chemicals Refresher Tabs

Material	CPI
NEOPRENE	B
NITRILE	C
PVC	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

Type A-P 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 5 x ES	A-AUS / Class 1 P2	-	A-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	A-2 P2	A-PAPR-2 P2
up to 50 x ES	-	A-3 P2	-
50+ x ES	-	Air-line**	-

^ - Full-face
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)

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Coloured tablet block with perfumed aromatic odour. Sublimes (evaporates) at room temperature. Very slightly soluble in water.

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Physical state	Manufactured	**Relative density (Water = 1)**	1.46
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	560
pH (as supplied)	Not Applicable	**Decomposition temperature (°C)**	>55
Melting point / freezing point (°C)	53.1	**Viscosity (cSt)**	Not Applicable
Initial boiling point and boiling range (°C)	174	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	67	**Taste**	Not Available
Evaporation rate	Not Applicable	**Explosive properties**	Not Available
Flammability	Combustible.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	16	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	2.5	**Volatile Component (%vol)**	100
Vapour pressure (kPa)	1.33 @ 54.8 C.	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	5.08	**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	Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant 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. 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

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	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	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. 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. 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.
Skin Contact	Skin contact with the material may damage the health of the individual; systemic effects may result following absorption. Limited 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. 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. 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.

Continued...

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.
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NV Chemicals 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*

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

Continued...

	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. **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.

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

NV Chemicals 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	96h	Algae or other aquatic plants	1.6mg/L	5
BCF	840h	Fish	33-72	7
EC50	72h	Algae or other aquatic plants	31mg/l	2
EC50	48h	Crustacea	0.7mg/l	2
LC50	96h	Fish	0.88mg/l	4
EC50(ECx)	24h	Algae or other aquatic plants	<0.001mg/L	4

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.
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	‣ Recycle wherever possible or consult manufacturer for recycling options. ‣ Consult State Land Waste Management Authority for disposal. ‣ Bury residue in an authorised landfill. ‣ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

Continued...

	
Marine Pollutant	
HAZCHEM	2Z

Land transport (ADG)

UN number or ID number	3077
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains 1,4-dichlorobenzene)
Transport hazard class(es)	Class: 9 Subsidiary risk: Not Applicable
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: 274 331 335 375 AU01 Limited quantity: 5 kg

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082
are not subject to this Code when transported by road or rail in;
(a) packagings;
(b) IBCs; or
(c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) - ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

UN number	3077
UN proper shipping name	Environmentally hazardous substance, solid, n.o.s. (contains 1,4-dichlorobenzene)
Transport hazard class(es)	ICAO/IATA Class: 9 ICAO / IATA Subrisk: Not Applicable ERG Code: 9L
Packing group	III
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: A97 A158 A179 A197 A215 Cargo Only Packing Instructions: 956 Cargo Only Maximum Qty / Pack: 400 kg Passenger and Cargo Packing Instructions: 956 Passenger and Cargo Maximum Qty / Pack: 400 kg Passenger and Cargo Limited Quantity Packing Instructions: Y956 Passenger and Cargo Limited Maximum Qty / Pack: 30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	3077
UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, SOLID, N.O.S. (contains 1,4-dichlorobenzene)
Transport hazard class(es)	IMDG Class: 9 IMDG Subrisk: Not Applicable
Packing group	III
Environmental hazard	Marine Pollutant
Special precautions for user	EMS Number: F-A, S-F Special provisions: 274 335 966 967 969 Limited Quantities: 5 kg

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

Continued...

Product name	Group
1,4-dichlorobenzene	Not Available

Transport in bulk in accordance with the IGC 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
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
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 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

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	20/08/2021
Initial Date	05/09/2011

SDS Version Summary

Version	Date of Update	Sections Updated
3.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification
4.1	20/08/2021	Classification change due to full database hazard calculation/update.

Other information

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

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

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

Continued...

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

end of SDS