HEROGRIP EnviroMight Canister Adhesive

QUIN GLOBAL ASIA PACIFIC

Chemwatch Hazard Alert Code: 4

Version No: **4.6**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **18/07/2024**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	HEROGRIP EnviroMight Canister Adhesive
Synonyms	Not Available
Proper shipping name	CHEMICAL UNDER PRESSURE, FLAMMABLE, N.O.S.
Other means of identification	Not Available

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

Relevant identified uses	Adhesive

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**QUIN GLOBAL ASIA PACIFIC**
Address	63 Hincksman Street Queanbeyan, NSW 2620 Australia
Telephone	+61 2 6175 0574
Fax	Not Available
Website	www.quinglobal.com
Email	sales@quinglobal.com.au

Emergency telephone number

Association / Organisation	**CHEMWATCH EMERGENCY RESPONSE (24/7)**
Emergency telephone numbers	+61 1800 951 288
Other emergency telephone numbers	+61 3 9573 3188

Once connected and if the message is not in your preferred language then please dial 01

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Flammable Gases Category 1A, Gases Under Pressure (Liquefied Gas), Serious Eye Damage/Eye Irritation Category 2B
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Label elements

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

Hazard statement(s)

H220	Extremely flammable gas.

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H280	Contains gas under pressure; may explode if heated.
H320	Causes eye irritation.
AUH044	Risk of explosion if heated under confinement.

Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P377	Leaking gas fire: Do not extinguish, unless leak can be stopped safely.
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.
P381	In case of leakage, eliminate all ignition sources.

Precautionary statement(s) Storage

P410+P403	Protect from sunlight. Store in a well-ventilated place.

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
68476-85-7.	35	LPG (liquefied petroleum gas)
110-71-4	48.75	1,2-dimethoxyethane
64742-49-0.	2.6	naphtha petroleum, light, hydrotreated
66070-58-4	13.65	styrene/ butadiene copolymer, hydrogenated
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.
Ingestion	▸ Not considered a normal route of entry.

SECTION 5 Firefighting measures

Extinguishing media

DO NOT EXTINGUISH BURNING GAS UNLESS LEAK CAN BE STOPPED SAFELY:
OTHERWISE: LEAVE GAS TO BURN.
FOR SMALL FIRE:
▸ Dry chemical, CO2 or water spray to extinguish gas (only if absolutely necessary and safe to do so).
▸ **DO NOT** use water jets.
FOR LARGE FIRE:
▸ Cool cylinder by direct flooding quantities of water onto upper surface until well after fire is out.

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- **DO NOT** direct water at source of leak or venting safety devices as icing may occur.

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. ▸ May be violently or explosively reactive. ▸ Wear breathing apparatus plus protective gloves. ▸ Consider evacuation ▸ Fight fire from a safe distance, with adequate cover. ▸ If safe, switch off electrical equipment until vapour fire hazard removed. ▸ Use water delivered as a fine spray to control fire and cool adjacent area. ▸ **DO NOT** approach cylinders suspected to be hot. ▸ Cool fire-exposed cylinders with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	Combustion products include: carbon monoxide (CO) carbon dioxide (CO_2) metal oxides other pyrolysis products typical of burning organic material. **Contains low boiling substance:** Closed containers may rupture due to pressure buildup under fire conditions. **WARNING**: Long standing in contact with air and light may result in the formation of potentially explosive peroxides. ▸ Vented gas is more dense than air and may collect in pits, basements.
HAZCHEM	2YE

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	▸ Avoid breathing vapour and any contact with liquid or gas. Protective equipment including respirator should be used. ▸ **DO NOT** enter confined spaces where gas may have accumulated. ▸ Shut off all sources of possible ignition and increase ventilation. ▸ Clear area of personnel. ▸ Stop leak only if safe to so do. ▸ Remove leaking cylinders to safe place. release pressure under safe controlled conditions by opening valve. ▸ Orientate cylinder so that the leak is gas, not liquid, to minimise rate of leakage ▸ Keep area clear of personnel until gas has dispersed.
Major Spills	▸ Clear area of all unprotected personnel and move upwind. ▸ Alert Emergency Authority and advise them of the location and nature of hazard. ▸ May be violently or explosively reactive. ▸ Wear full body clothing with breathing apparatus. ▸ Prevent by any means available, spillage from entering drains and water-courses. ▸ Consider evacuation. ▸ Shut off all possible sources of ignition and increase ventilation. ▸ No smoking or naked lights within area. ▸ Use extreme caution to prevent violent reaction. ▸ Stop leak only if safe to so do. ▸ Water spray or fog may be used to disperse vapour. ▸ DO NOT enter confined space where gas may have collected. ▸ Keep area clear until gas has dispersed. ▸ Remove leaking cylinders to a safe place. ▸ Fit vent pipes. Release pressure under safe, controlled conditions ▸ Burn issuing gas at vent pipes. ▸ DO NOT exert excessive pressure on valve; DO NOTattempt to operate damaged valve.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Test for leakage with brush and detergent **-NEVER** use a naked flame. ▸ Do NOT heat cylinder by any means to increase the discharge rate of product from cylinder. ▸ Leaking gland nuts may be tightened if necessary. ▸ If a cylinder valve will not close completely, remove the cylinder to a well ventilated location (e.g. outside) and, when empty, tag as FAULTY and return to supplier. ▸ Obtain a work permit before attempting any repairs. ▸ DO NOT attempt repair work on lines, vessels under pressure. ▸ Atmospheres must be tested and O.K. before work resumes after leakage.

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	▸ Avoid generation of static electricity. Earth all lines and equipment. ▸ **DO NOT** transfer gas from one cylinder to another. ▸ Containers, even those that have been emptied, may contain explosive vapours. ▸ Do NOT cut, drill, grind, weld or perform similar operations on or near containers. The tendency of many ethers to form explosive peroxides is well documented. Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe ▸ **DO NOT** concentrate by evaporation, or evaporate extracts to dryness, as residues may contain explosive peroxides with DETONATION potential. ▸ Any static discharge is also a source of hazard. ▸ Before any distillation process remove trace peroxides by shaking with excess 5% aqueous ferrous sulfate solution or by percolation through a column of activated alumina. ▸ Distillation results in uninhibited ether distillate with considerably increased hazard because of risk of peroxide formation on storage. ▸ Add inhibitor to any distillate as required. ▸ When solvents have been freed from peroxides by percolation through columns of activated alumina, the absorbed peroxides must promptly be desorbed by treatment with polar solvents such as methanol or water, which should then be disposed of safely.
Other information	

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Cylinder: ▸ Ensure the use of equipment rated for cylinder pressure. ▸ Ensure the use of compatible materials of construction. ▸ Valve protection cap to be in place until cylinder is secured, connected. ▸ Cylinder must be properly secured either in use or in storage. ▸ Cylinder valve must be closed when not in use or when empty. ▸ Segregate full from empty cylinders. **WARNING:** Suckback into cylinder may result in rupture. Use back-flow preventive device in piping.
Storage incompatibility	Acetals and ketals: ▸ slowly form peroxides on reaction with air. ▸ are generally stable and lack reactivity in neutral to strongly basic environments (acetals hydrolyse uncatalysed in acidic gastric juices and intestinal fluids to yield acetaldehydes) ▸ exhibit all the lack of reactivity associated with ethers in general so long as they are not treated with acids, especially aqueous acids. Formation of an acetal occurs when the hydroxyl group of a hemiacetal becomes protonated and is lost as water. Acetals are stable compared to hemiacetals but their formation is a reversible equilibrium as with esters. Methylal ▸ forms unstable peroxides on storage ▸ reacts violently with strong oxidisers ▸ is incompatible with acids ▸ attacks some plastics, rubber and coatings ▸ may generate electrostatic charges due to low conductivity Propane: ▸ reacts violently with strong oxidisers, barium peroxide, chlorine dioxide, dichlorine oxide, fluorine etc. ▸ liquid attacks some plastics, rubber and coatings ▸ may accumulate static charges which may ignite its vapours Ethers · may react violently with strong oxidising agents and acids. · can act as bases.- they form salts with strong acids and addition complexes with Lewis acids; the complex between diethyl ether and boron trifluoride is an example. · are generally stable to water under neutral conditions and ambient temperatures. · are hydrolysed by heating in the presence of halogen acids, particularly hydrogen iodide · are relatively inert In other reactions, which typically involve the breaking of the carbon-oxygen bond ▸ The tendency of many ethers to form explosive peroxides is well documented. ▸ Ethers lacking non-methyl hydrogen atoms adjacent to the ether link are thought to be relatively safe. ▸ When solvents have been freed from peroxides (by percolation through a column of activated alumina for example), the absorbed peroxides must promptly be desorbed by treatment with the polar solvents methanol or water, which should be discarded safely. ▸ Compressed gases may contain a large amount of kinetic energy over and above that potentially available from the energy of reaction produced by the gas in chemical reaction with other substances

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	LPG (liquefied petroleum gas)	LPG (liquified petroleum gas)	1000 ppm / 1800 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
LPG (liquefied petroleum gas)	65,000 ppm	2.30E+05 ppm	4.00E+05 ppm
1,2-dimethoxyethane	13 ppm	140 ppm	840 ppm
naphtha petroleum, light, hydrotreated	1,000 mg/m3	11,000 mg/m3	66,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
LPG (liquefied petroleum gas)	Not Available	Not Available
1,2-dimethoxyethane	Not Available	Not Available

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Ingredient	Original IDLH	Revised IDLH
naphtha petroleum, light, hydrotreated	Not Available	Not Available
styrene/ butadiene copolymer, hydrogenated	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
1,2-dimethoxyethane	E	≤ 0.1 ppm
naphtha petroleum, light, hydrotreated	E	≤ 0.1 ppm
Notes:	***Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.***	

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.
· Areas where cylinders are stored require good ventilation and, if enclosed need discrete/ controlled exhaust ventilation.
· Secondary containment and exhaust gas treatment may be required by certain jurisdictions.
· Local exhaust ventilation (explosion proof) is usually required in workplaces.
· Automated controls should ensure that workplace atmospheres do not exceed 25% of the lower explosive limit (LEL) (if available).
· Monitor the work area and secondary containments for release of gas.
· Automated alerting systems with automatic shutdown of gas-flow may be appropriate and may in fact be mandatory in certain jurisdictions.
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:
gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
· Adequate ventilation is typically taken to be that which limits the average concentration to no more than 25% of the LEL within the building, room or enclosure containing the dangerous substance.
· Ventilation for plant and machinery is normally considered adequate if it limits the average concentration of any dangerous substance that might potentially be present to no more than 25% of the LEL. However, an increase up to a maximum 50% LEL can be acceptable where additional safeguards are provided to prevent the formation of a hazardous explosive atmosphere. For example, gas detectors linked to emergency shutdown of the process might be used together with maintaining or increasing the exhaust ventilation on solvent evaporating ovens and gas turbine enclosures.
· Temporary exhaust ventilation systems may be provided for non-routine higher-risk activities, such as cleaning, repair or maintenance in tanks or other confined spaces or in an emergency after a release. The work procedures for such activities should be carefully considered.. The atmosphere should be continuously monitored to ensure that ventilation is adequate and the area remains safe. Where workers will enter the space, the ventilation should ensure that the concentration of the dangerous substance does not exceed 10% of the LEL (irrespective of the provision of suitable breathing apparatus)

Individual protection measures, such as personal protective equipment

Eye and face protection

- Chemical goggles.
- Full face shield may be required for supplementary but never for primary protection of eyes.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. 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

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Hands/feet protection	▸ When handling sealed and suitably insulated cylinders wear cloth or leather gloves. ▸ Insulated gloves: ▸ NOTE: Insulated gloves should be loose fitting so that may be removed quickly if liquid is spilled upon them. Insulated gloves are not made to permit hands to be placed in the liquid; they provide only short-term protection from accidental contact with the liquid.
Body protection	See Other protection below
Other protection	▸ The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton. ▸ Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost. BRETHERICK: Handbook of Reactive Chemical Hazards. ▸ Protective overalls, closely fitted at neck and wrist. ▸ Eye-wash unit. **IN CONFINED SPACES:** ▸ Non-sparking protective boots ▸ Static-free clothing. ▸ Ensure availability of lifeline. Staff should be trained in all aspects of rescue work. Rescue gear: Two sets of SCBA breathing apparatus Rescue Harness, lines etc. ▸ Some plastic personal protective equipment (PPE) (e.g. gloves, aprons, overshoes) are not recommended as they may produce static electricity. ▸ For large scale or continuous use wear tight-weave non-static clothing (no metallic fasteners, cuffs or pockets). ▸ Non sparking safety or conductive footwear should be considered. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.

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:
HEROGRIP EnviroMight Canister Adhesive

Material	CPI
BUTYL	A

* 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 AX-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	Air-line*	AX-2 P2	AX-PAPR-2 P2 ^
up to 10 x ES	-	AX-3 P2	-
10+ x ES	-	Air-line**	-

* - Continuous Flow; ** - Continuous-flow or positive pressure demand
^ - 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)

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used
- Positive pressure, full face, air-supplied breathing apparatus should be used for work in enclosed spaces if a leak is suspected or the primary containment is to be opened (e.g. for a cylinder change)
- Air-supplied breathing apparatus is required where release of gas from primary containment is either suspected or demonstrated.

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	AX-AUS / Class 1	-
up to 50	1000	-	AX-AUS / Class 1
up to 50	5000	Airline *	-
up to 100	5000	-	AX-2
up to 100	10000	-	AX-3
100+		-	Airline**

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** - Continuous-flow or positive pressure demand.
A(All classes) = Organic vapours, B AUS or B1 = Acid gases, 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 deg C)

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Not Available

Physical state	Liquified Gas	**Relative density (Water = 1)**	0.838
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	495
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	-97	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	-40	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	-104	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	HIGHLY FLAMMABLE.	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	9.1	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	2.2	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	46.86	**Gas group**	Not Available
Solubility in water	Partly miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	2.93	**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. ▸ Presence of heat source ▸ Presence of an ignition source
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. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of acetals may produce a transitory ether-like anaesthesia. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.

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	Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure. The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation. Guinea pigs to very high levels of methylal vapour for 2 to 6 hours showed extensive bronchopneumonia and moderate to severe fatty degeneration of the liver and kidney, and in some cases, moderate fatty degeneration of the myocardium. Liver and kidney injury may follow high exposures
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal. Considered an unlikely route of entry in commercial/industrial environments The liquid may produce considerable gastrointestinal discomfort and may be harmful or toxic if swallowed. Ingestion may result in nausea, pain and vomiting. Vomit entering the lungs by aspiration may cause potentially lethal chemical pneumonitis
Skin Contact	The material may accentuate any pre-existing dermatitis condition Alkyl ethers may defat and dehydrate the skin producing dermatoses. Absorption may produce headache, dizziness, and central nervous system depression. 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. Skin contact with the material may be harmful; systemic effects may result following absorption. The material produces severe skin irritation; evidence exists, or practical experience predicts, that the material either: ▸ produces severe inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant and severe 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. **NOTE:** Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
Eye	Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to 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. Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures.. The vapour when concentrated has pronounced eye irritation effects and this gives some warning of high vapour concentrations. If eye irritation occurs seek to reduce exposure with available control measures, or evacuate area.
Chronic	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. 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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/ormemory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses. Surface cracking and erosion may also increase susceptibility to infection by microorganisms. Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties. Animal studies: No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human. Principal route of occupational exposure to the gas is by inhalation. Chronic exposure to alkyl ethers may result in loss of appetite, excessive thirst, fatigue, and weight loss Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]
HEROGRIP EnviroMight Canister Adhesive	

TOXICITY	IRRITATION

Continued...

	Not Available	Not Available

LPG (liquefied petroleum gas)

TOXICITY	IRRITATION
Inhalation (Rat) LC50: 658 mg/l4h[2]	Not Available

1,2-dimethoxyethane

TOXICITY	IRRITATION
Dermal (Guinea Pig) LD50: 5 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Inhalation (Rat) LC50: 3000 ppm4h[2]	Skin: adverse effect observed (irritating)[1]
Oral (Rabbit) LD50; 320 mg/kg[2]	

naphtha petroleum, light, hydrotreated

TOXICITY	IRRITATION
dermal (rat) LD50: 3.35 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
Inhalation (Rat) LC50: 0.26 mg/L4h[2]	Skin: adverse effect observed (irritating)[1]
Oral (Rat) LD50: 16.75 mg/kg[2]	

styrene/ butadiene copolymer, hydrogenated

TOXICITY	IRRITATION
Not Available	Eye (rabbit): 500 mg/24h - mild

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***

LPG (LIQUEFIED PETROLEUM GAS)

for Petroleum Hydrocarbon Gases:
In many cases, there is more than one potentially toxic constituent in a refinery gas. In those cases, the constituent that is most toxic for a particular endpoint in an individual refinery stream is used to characterize the endpoint hazard for that stream. The hazard potential for each mammalian endpoint for each of the petroleum hydrocarbon gases is dependent upon each petroleum hydrocarbon gas constituent endpoint toxicity values (LC50, LOAEL, etc.) and the relative concentration of the constituent present in that gas. It should also be noted that for an individual petroleum hydrocarbon gas, the constituent characterizing toxicity may be different for different mammalian endpoints, again, being dependent upon the concentration of the different constituents in each, distinct petroleum hydrocarbon gas.
All Hydrocarbon Gases Category members contain primarily hydrocarbons (i.e., alkanes and alkenes) and occasionally asphyxiant gases like hydrogen. The inorganic components of the petroleum hydrocarbon gases are less toxic than the C1 - C4 and C5 - C6 hydrocarbon components to both mammalian and aquatic organisms. Unlike other petroleum product categories (***e.g.*** gasoline, diesel fuel, lubricating oils, etc.), the inorganic and hydrocarbon constituents of hydrocarbon gases can be evaluated for hazard individually to then predict the screening level hazard of the Category members
Acute toxicity: No acute toxicity LC50 values have been derived for the C1 -C4 and C5- C6 hydrocarbon (HC) fractions because no mortality was observed at the highest exposure levels tested (~ 5 mg/l) for these petroleum hydrocarbon gas constituents. The order of acute toxicity of petroleum hydrocarbon gas constituents from most to least toxic is:
C5-C6 HCs (LC50 > 1063 ppm) > C1-C4 HCs (LC50 > 10,000 ppm) > benzene (LC50 = 13,700 ppm) > butadiene (LC50 = 129,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).
Repeat dose toxicity: With the exception of the asphyxiant gases, repeated dose toxicity has been observed in individual selected petroleum hydrocarbon gas constituents. Based upon LOAEL values, the order of order of repeated-dose toxicity of these constituents from most toxic to the least toxic is:
Benzene (LOAEL .>=10 ppm) >C1-C4 HCs (LOAEL = 5,000 ppm; assumed to be 100% 2-butene) > C5-C6 HCs (LOAEL = 6,625 ppm) > butadiene (LOAEL = 8,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).
Genotoxicity:
In vitro: The majority of the Petroleum Hydrocarbon Gases Category components are negative for ***in vitro*** genotoxicity. The exceptions are: benzene and 1,3-butadiene, which are genotoxic in bacterial and mammalian ***in vitro*** test systems.
In vivo: The majority of the Petroleum Hydrocarbon Gases Category components are negative for ***in vivo*** genotoxicity**.** The exceptions are benzene and 1,3-butadiene, which are genotoxic in ***in vivo*** test systems
Developmental toxicity: Developmental effects were induced by two of the petroleum hydrocarbon gas constituents, benzene and the C5 - C6 hydrocarbon fraction. No developmental toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for developmental toxicity. Based on LOAEL and NOAEL values, the order of acute toxicity of these constituents from most to least toxic is:
Benzene (LOAEL = 20 ppm) > butadiene (NOAEL .>=1,000 ppm) > C5-C6 HCs (LOAEL = 3,463 ppm) > C1-C4 HCs (NOAEL >=5,000 ppm; assumed to be 100% 2-butene) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen)**.**
Reproductive toxicity: Reproductive effects were induced by only two petroleum hydrocarbon gas constituents, benzene and isobutane (a constituent of the the C1-C4 hydrocarbon fraction). No reproductive toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for reproductive toxicity. Based on LOAEL and NOAEL values, the order of reproductive toxicity of these constituents from most to least toxic is:
Benzene (LOAEL = 300 ppm) > butadiene (NOAEL .>=6,000 ppm) > C5-C6 HCs (NOAEL .>=6,521 ppm) > C1-C4 HCs (LOAEL = 9,000 ppm; assumed to be 100% isobutane) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen)

1,2-DIMETHOXYETHANE

Animal testing shows material is a reproductive effector:
Studies with some ethylene glycol ethers and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of the glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decrease significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats. This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure. Ethylene glycol ethers and acetates are mainly metabolised to alkoxyacetic acids but there is also a minor pathway through ethylene glycol to oxalic acid. The main pathway of ethylene glycol ethers is associated with significant clinical or experimental health effects, but the minor pathway is also interesting because formation of urinary stones depends principally upon urinary concentration of oxalate and calcium. In one study (1) the tendency to form urinary stones was 2.4 times higher amongst silk-screen printers exposed to ethylene glycol ethers, than among office workers. (1) Laitinen J., et al: Occupational Environmental Medicine 1996, 53 595-600

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NAPHTHA PETROLEUM, LIGHT, HYDROTREATED	DHC Solvent Chemie (for EC No.: 926-605-8) For Low Boiling Point Naphthas (LBPNs): Acute toxicity: LBPNs generally have low acute toxicity by the oral (median lethal dose [LD50] in rats > 2000 mg/kg-bw), inhalation (LD50 in rats > 5000 mg/m3) and dermal (LD50 in rabbits > 2000 mg/kg-bw) routes of exposure Most LBPNs are mild to moderate eye and skin irritants in rabbits, with the exception of heavy catalytic cracked and heavy catalytic reformed naphthas, which have higher primary skin irritation indices. Sensitisation: LBPNs do not appear to be skin sensitizers, but a poor response in the positive control was also noted in these studies Repeat dose toxicity: The lowest-observed-adverse-effect concentration (LOAEC) and lowest-observed-adverse-effect level (LOAEL) values identified following short-term (2-89 days) and subchronic (greater than 90 days) exposure to the LBPN substances. These values were determined for a variety of endpoints after considering the toxicity data for all LBPNs in the group. Most of the studies were carried out by the inhalation route of exposure. Renal effects, including increased kidney weight, renal lesions (renal tubule dilation, necrosis) and hyaline droplet formation, observed in male rats exposed orally or by inhalation to most LBPNs, were considered species- and sex-specific These effects were determined to be due to a mechanism of action not relevant to humans -specifically, the interaction between hydrocarbon metabolites and alpha-2-microglobulin, an enzyme not produced in substantial amounts in female rats, mice and other species, including humans. The resulting nephrotoxicity and subsequent carcinogenesis in male rats were therefore not considered in deriving LOAEC/LOAEL values. Only a limited number of studies of short-term and subchronic duration were identified for site-restricted LBPNs. The lowest LOAEC identified in these studies, via the inhalation route, is 5475 mg/m3, based on a concentration-related increase in liver weight in both male and female rats following a 13-week exposure to light catalytic cracked naphtha. Shorter exposures of rats to this test substance resulted in nasal irritation at 9041 mg/m3 No systemic toxicity was reported following dermal exposure to light catalytic cracked naphtha, but skin irritation and accompanying histopathological changes were increased, in a dose-dependent manner, at doses as low as 30 mg/kg-bw per day when applied 5 days per week for 90 days in rats No non-cancer chronic toxicity studies (= 1 year) were identified for site-restricted LBPNs and very few non-cancer chronic toxicity studies were identified for other LBPNs. An LOAEC of 200 mg/m3 was noted in a chronic inhalation study that exposed mice and rats to unleaded gasoline (containing 2% benzene). This inhalation LOAEC was based on ocular discharge and ocular irritation in rats. At the higher concentration of 6170 mg/m3, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m3). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m3 A LOAEL of 714 mg/kg-bw was identified for dermal exposure based on local skin effects (inflammatory and degenerative skin changes) in mice following application of naphtha for 105 weeks. No systemic toxicity was reported. Genotoxicity: Although few genotoxicity studies were identified for the site-restricted LBPNs, the genotoxicity of several other LBPN substances has been evaluated using a variety of in vivo and in vitro assays. While in vivo genotoxicity assays were negative overall, the in vitro tests exhibited mixed results. For in vivo genotoxicity tests, LBPNs exhibited negative results for chromosomal aberrations and micronuclei induction, but exhibited positive results in one sister chromatid exchange assay although this result was not considered definitive for clastogenic activity as no genetic material was unbalanced or lost. Mixtures that were tested, which included a number of light naphthas, displayed mixed results (i.e., both positive and negative for the same assay) for chromosomal aberrations and negative results for the dominant lethal mutation assay. Unleaded gasoline (containing 2% benzene) was tested for its ability to induce unscheduled deoxyribonucleic acid (DNA) synthesis (UDS) and replicative DNA synthesis (RDS) in rodent hepatocytes and kidney cells. UDS and RDS were induced in mouse hepatocytes via oral exposure and RDS was induced in rat kidney cells via oral and inhalation exposure. Unleaded gasoline (benzene content not stated) exhibited negative results for chromosomal aberrations and the dominant lethal mutation assay and mixed results for atypical cell foci in rodent renal and hepatic cells. For in vitro genotoxicity studies, LBPNs were negative for six out of seven Ames tests, and were also negative for UDS and for forward mutations LBPNs exhibited mixed or equivocal results for the mouse lymphoma and sister chromatid exchange assays, as well as for cell transformation and positive results for one bacterial DNA repair assay. Mixtures that were tested, which included a number of light naphthas, displayed negative results for the Ames and mouse lymphoma assays Gasoline exhibited negative results for the Ames test battery, the sister chromatid exchange assay and for one mutagenicity assay . Mixed results were observed for UDS and the mouse lymphoma assay. While the majority of in vivo genotoxicity results for LBPN substances are negative, the potential for genotoxicity of LBPNs as a group cannot be discounted based on the mixed in vitro genotoxicity results. Carcinogenicity: Although a number of epidemiological studies have reported increases in the incidence of a variety of cancers, the majority of these studies are considered to contain incomplete or inadequate information. Limited data, however, are available for skin cancer and leukemia incidence, as well as mortality among petroleum refinery workers. It was concluded that there is limited evidence supporting the view that working in petroleum refineries entails a carcinogenic risk (Group 2A carcinogen). IARC (1989a) also classified gasoline as a Group 2B carcinogen; it considered the evidence for carcinogenicity in humans from gasoline to be inadequate and noted that published epidemiological studies had several limitations, including a lack of exposure data and the fact that it was not possible to separate the effects of combustion products from those of gasoline itself. Similar conclusions were drawn from other reviews of epidemiological studies for gasoline (US EPA 1987a, 1987b). Thus, the evidence gathered from these epidemiological studies is considered to be inadequate to conclude on the effect s of human exposure to LBPN substances. No inhalation studies assessing the carcinogenicity of the site-restricted LBPNs were identified. Only unleaded gasoline has been examined for its carcinogenic potential, in several inhalation studies. In one study, rats and mice were exposed to 0, 200, 870 or 6170 mg/m3 of a 2% benzene formulation of the test substance, via inhalation, for approximately 2 years. A statistically significant increase in hepatocellular adenomas and carcinomas, as well as a non-statistical increase in renal tumours, were observed at the highest dose in female mice. A dose-dependent increase in the incidence of primary renal neoplasms was also detected in male rats, but this was not considered to be relevant to humans, as discussed previously.Carcinogenicity was also assessed for unleaded gasoline, via inhalation, as part of initiation/promotion studies. In these studies, unleaded gasoline did not appear to initiate tumour formation, but did show renal cell and hepatic tumour promotion ability, when rats and mice were exposed, via inhalation, for durations ranging from 13 weeks to approximately 1 year using an initiation/promotion protocol However, further examination of data relevant to the composition of unleaded gasoline demonstrated that this is a highly-regulated substance; it is expected to contain a lower percentage of benzene and has a discrete component profile when compared to other substances in the LBPN group. Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPN substances as carcinogenic. All of these substances were classified by the European Commission (2008) as Category 2 (R45: may cause cancer) (benzene content = 0.1% by weight). IARC has classified gasoline, an LBPN, as a Group 2B carcinogen (possibly carcinogenic to humans) and “occupational exposures in petroleum refining” as Group 2A carcinogens (probably carcinogenic to humans). Several studies were conducted on experimental animals to investigate the dermal carcinogenicity of LBPNs. The majority of these studies were conducted through exposure of mice to doses ranging from 694-1351 mg/kg-bw, for durations ranging from 1 year to the animals lifetime or until a tumour persisted for 2 weeks. Given the route of exposure, the studies specifically examined the formation of skin tumours. Results for carcinogenicity via dermal exposure are mixed. Both malignant and benign skin tumours were induced with heavy catalytic cracked naphtha, light catalytic cracked naphtha, light straight-run naphtha and naphtha Significant increases in squamous cell carcinomas were also observed when mice were dermally treated with Stoddard solvent, but the latter was administered as a mixture (90% test substance), and the details of the study were not available. In contrast, insignificant increases in tumour formation or no tumours were observed when light alkylate naphtha, heavy catalytic reformed naphtha, sweetened naphtha, light catalytically cracked naphtha or unleaded gasoline was dermally applied to mice. Negative results for skin tumours were also observed in male mice dermally exposed to sweetened naphtha using an initiation/promotion protocol. Reproductive/ Developmental toxicity: No reproductive or developmental toxicity was observed for the majority of LBPN substances evaluated. Most of these studies were carried out by inhalation exposure in rodents. NOAEC values for reproductive toxicity following inhalation exposure ranged from 1701 mg/m3 (CAS RN 8052-41-3) to 27 687 mg/m3 (CAS RN 64741-63-5) for the LBPNs group evaluated, and from 7690 mg/m3 to 27 059 mg/m3 for the site-restricted light catalytic cracked and full-range catalytic reformed naphthas. However, a decreased number of pups per litter and higher frequency of post-implantation loss were observed following inhalation exposure of female rats to hydrotreated heavy naphtha (CAS RN 64742-48-9) at a concentration of 4679 mg/m3, 6 hours per day, from gestational days 7-20. For dermal exposures, NOAEL values of 714 mg/kg-bw (CAS RN 8030-30-6) and 1000 mg/kg-bw per day (CAS RN 68513-02-0) were noted . For oral exposures, no adverse effects on reproductive parameters were reported when rats were given site-restricted light catalytic cracked naphtha at 2000 mg/kg on gestational day 13 . For most LBPNs, no treatment-related developmental effects were observed by the different routes of exposure However, developmental toxicity was observed for a few naphthas. Decreased foetal body weight and an increased incidence of ossification variations were observed when rat dams were exposed to light aromatized solvent naphtha, by gavage, at 1250 mg/kg-bw per day. In addition, pregnant rats exposed by inhalation to hydrotreated heavy naphtha at 4679 mg/m3 delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring. Low Boiling Point Naphthas [Site-Restricted]

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	Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length,with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent that iso- or cyclo-paraffins. The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption,is known as the 'hydrocarbon continuum hypothesis', and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver. The High Benzene Naphthas (HBNs; Lower Olefins and Aromatics -LOA - CAT H) Category was developed for the HPV Program by grouping ethylene manufacturing streams (products) that exhibit commonalities from both manufacturing process and compositional perspectives. intermediates. The category includes hydrocarbon product streams associated with the ethylene industry that contain significant levels of benzene, generally with a benzene content greater than 10% and averaging about 55%. This grouping of CAS numbers represents hydrocarbon streams with a carbon number distribution that is predominantly C5- C11, through components boiling at 350 C or higher.. The high benzene naphthas category contains hydrocarbons (aliphatic, aromatic and olefinic) with carbon numbers predominantly in the C5-C10 range and boiling from approximately 30 deg C to 300 deg C. Members of this category contain >0.1% benzene and contain varying amounts of toluene, xylenes and n-hexane. Some category members contain naphthalenes, isoprene and 1,3-butadiene and this has been quantified where possible All the streams in this category are complex UVCBs containing = 50% paraffins, = 60% isoparaffins, = 90% olefins, = 90% naphthenics, =100% aromatics, and above 0.1% benzene. All streams within this category are expected to have the following classifications H304, H315 and H336, H340, H350 (given their composition) and a flammability classification (either H224 or H226, depending on the flash point and / or the boiling point) Benzene, as the predominant component in most streams, is expected to be the key driver with respect to health effects endpoints within the SIDS battery of tests. However, as the concentration of benzene is decreased and the concentrations of other components are increased, the observed effects of benzene are expected to diminish and the effects of other components are expected to increase. The existing epidemiology and toxicology database for the components other than benzene and for mixtures containing the components is extensive. All components present in the streams at concentrations greater than 5% have been tested in at least one toxicity study. Those components having only limited data lack structural alerts for mammalian toxicity and data exist for their structural analogs. The C5 and C6 alkanes and alkenes present in the streams are not expected to significantly contribute to the toxicity profile as these substances are present in the streams at low concentrations and, with the exception of hexane, generally have a low level of toxicity. The toxic effects of hexane (present at < 15%) are unlikely to be observed due to the presence of the other components. **Genotoxicity:** When tested as pure substances, some of the components other than benzene have caused genetic damage and adverse target organ effects in repeated-dose animal studies. When tested as pure substances, some of the components other than benzene have caused genetic damage and adverse target organ effects in repeated-dose animal studies. However, since the biologically active components of the High Benzene Naphthas streams are metabolized through a common P450 metabolic pathway, it is anticipated that multiple components will compete for the same active enzyme sites. Component toxicities, which are dependent on the formation of biologically active metabolites, may be reduced as less metabolite(s) will be produced through competition for these sites. Direct support for reduction or elimination of toxicities of individual components is provided by results of an existing mouse bone marrow micronucleus test with one of the High Benzene Naphthas streams, Hydrotreated C6-8 Fraction. This stream, containing approximately 55% benzene, was negative in a mouse bone marrow micronucleus test when administered by oral gavage at 5000 mg/kg to male and female CD-1 mice. Several studies have shown that benzene administered orally to CD-1 mice induces high frequencies of micronuclei in bone marrow erythrocytes at doses as low as 110 mg/kg . The presence in the Hydrotreated C6-8 Fraction of other components (approximately 25% toluene, 10% xylene, 7% pentane, 7% ethylbenzene, 3% cyclohexane, and 2% hexane) apparently inhibited the expected clastogenicity of benzene. Other similar interactions between components of the category have also been reported. **Repeat dose toxicity:** Repeated oral or inhalation exposures to many of the components of the streams in the category have been shown to cause adverse health effects in a variety of organs. However, existing data also show that antagonistic and synergistic interactions occur between some components comprising the streams. **Developmental toxicity:** Developmental toxicity data exist for most components present in this category at concentrations greater than 5% . In these studies, no convincing evidence was seen for teratogenicity in the absence of maternal toxicity. Foetotoxicity has been reported for some components, but mostly in the presence of maternal toxicity. A Pyrolysis Gasoline Fraction stream similar to the Pyrolysis Gasoline streams in the HBNs Category has been tested in an oral developmental toxicity study in rabbits. No developmental effects were seen. **Reproductive toxicity:** Some data for benzene indicates adverse gonadal effects (e.g., atrophy/degeneration, decrease in spermatozoa, moderate increases in abnormal sperm forms), data on reproductive outcomes are either inconclusive or conflicting. However, most studies indicate no effects on reproductive indices, even at high doses. Reproductive organ effects were seen after inhalation exposure to isoprene and hexane. **Gene Mutation**: Of the identified category components present at concentrations greater than 5%, only 1,3-butadiene and benzene have consistently caused gene mutations in genetic toxicity tests . 1,3- Butadiene was positive in several ***in vivo*** and ***in vitro*** tests. Benzene was negative in several standard tests but was positive in an ***in vivo*** HPRT gene mutation test in mouse spleenocytes. Based on the data for components, the streams in the category are predicted to be negative in the HPV gene mutation test (Ames Test). Negative Ames Tests conducted with two streams (one from this category and one similar to category streams) support this prediction **Chromosome Aberration:**: Benzene has caused chromosome aberrations in ***in vitro*** and ***in vivo*** tests. The other most prevalent component in streams in this category, toluene, is negative in both ***in vitro*** and ***in vivo*** tests. Of the remaining identified category components present at concentrations greater than 5%, only vinyl acetate, 1,3-butadiene, isoprene, hexane, and naphthalene have been reported to cause chromosome aberrations. For petroleum: This product contains benzene, which can cause acute myeloid leukaemia, and n-hexane, which can be metabolized to compounds which are toxic to the nervous system. This product contains toluene, and animal studies suggest high concentrations of toluene lead to hearing loss. This product contains ethyl benzene and naphthalene, from which animal testing shows evidence of tumour formation. Cancer-causing potential: Animal testing shows inhaling petroleum causes tumours of the liver and kidney; these are however not considered to be relevant in humans. Mutation-causing potential: Most studies involving gasoline have returned negative results regarding the potential to cause mutations, including all recent studies in living human subjects (such as in petrol service station attendants). Reproductive toxicity: Animal studies show that high concentrations of toluene (>0.1%) can cause developmental effects such as lower birth weight and developmental toxicity to the nervous system of the foetus. Other studies show no adverse effects on the foetus. Human effects: Prolonged or repeated contact may cause defatting of the skin which can lead to skin inflammation and may make the skin more susceptible to irritation and penetration by other materials. Animal testing shows that exposure to gasoline over a lifetime can cause kidney cancer, but the relevance in humans is questionable.
HEROGRIP EnviroMight Canister Adhesive & 1,2-DIMETHOXYETHANE	For ethylene glycol monoalkyl ethers and their acetates (EGMAEs): Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates. EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.

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Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE ***in vitro*** than those of rats.
Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA ***in vitro*** and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA ***in vitro***.
Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in ***S. typhimurium*** strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. ***In vitro*** cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.
Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity
Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).
Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic.
The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE).
For 1,2-dimethoxyethane (monoglyme):
Monoglyme, an ethylene glycol ether, demonstrates a low order of toxicity with an oral LD50 of greater than 4000 mg/kg in rats. The acute inhalation toxicity of monoglyme was determined in a two-dose study in which the six-hour inhalation LC50 was found to be between 20 and 63 mg/L. The vapors produced some irritation and anesthesia at the high level. All high dose animals survived the exposure but died within 72 hours post-exposure.
A great deal of information is available on the repeated-dose toxicity of the biologically indicated metabolite of monoglyme, 2-methoxyethanol (2ME). In one study testicular degeneration was a prominent finding in rats even at the lowest dose tested (750 ppm, about 70 mg/kg/day) and in females, at this level, thymic atrophy was a finding. Thus, a NOEL was not found for rats of either sex. In the case of mice, the NOAEL for testicular degeneration and increased haematopoiesis in the spleen was 2000 ppm in males. A NOAEL was not reached for female mice since adrenal gland hypertrophy and increased haematopoiesis in the spleen occurred at the lowest concentration administered (2000 ppm, about 300 mg/kg/day).
Repeated-dose studies Repeated dose exposure in the drinking water was also associated with progressive anemia in rats and mice and increased mortality in rats at the two highest doses. The target organs can be identified as testes, bone marrow, spleen (hematopoiesis), thymus and adrenal. In general, the testes is considered a sensitive, if no the most sensitive, target organ.
A complete spectrum of toxicity can be confidently predicted from monoglyme s metabolism and studies on related compounds by all routes of exposure. Toxic responses include thymic atrophy, bone marrow suppression and testicular degeneration. Although direct chemical evidence identifying the primary and secondary metabolites of monoglyme was not found, the metabolic pathways for this class of chemicals are well known. Additionally, there is very strong biological evidence linking adverse effects of monoglyme with a common active metabolite of methoxy glycol ethers.
It has been demonstrated that most of the toxic effects of the monoalkyl glycol ethers arise as a result of the metabolic conversion of the glycol ether into a substituted acetic acid derivative. In humans it is established that 2-methoxyacetic acid is the defining toxic metabolite and studies have shown that the level of 2-methoxyacetic acid in urine is an excellent. marker for exposure Demethylation by mixed-function oxidases, a relatively slow reaction, accounts for some metabolic conversion to ethylene glycol which is converted to oxalate.
Dehydrogenase enzymes initially convert the free alcohol to the aldehyde and then the carboxylic acid . This is a very rapid conversion and it is known that a teratogenic dose of 2-methoxyethanol is completely oxidised, to 2-methoxyacetic acid, in a period of one-hour in rats. The competing reaction, demethylation of 2-methoxyethanol to ethylene glycol is comparatively slow as it is accomplished by the mixed-function oxidase system.
There is general agreement that 2-methoxyacetic acid is the proximate toxin. It is also established the clearance of 2-methoxyacetic acid is relatively slow as compared to its formation and the clearance in man may be much longer than in rats.
Genotoxicity has been evaluated using multiple ***in vitro*** experimental procedures covering both mutation and chromosomal aberration. Results of genotoxicity studies are mixed.
In ***in vitro*** studies, positive results are cited for the A ***S. typhimurium*** reverse mutation assay . Monoglyme is known to be cytotoxic to Salmonella typhimurium (30) at concentrations as low as 500 microliters per plate.
Monoglyme produced no evidence of genotoxicity in the presence or absence of S9 metabolic activation in mammalian cell point mutation tests using the HGPRT assay in CHO cells
Clastogenic activity was assessed ***in vitro*** using the Sister Chromatid Exchange in Chinese Hamster Ovary Cells Test (SCE). In this test, the material produced numerous indications of statistically significant effects on the frequency of SCE over the range of concentrations tested with and without addition of an active S9 metabolic system. A high number of cells were also observed with significant types of chromosomal aberrations suggesting that material was a clastogenic agent, especially in the presence of S9 activation
DNA damage was assessed using an ***in vitro*** unscheduled DNA synthesis (UDS) Assay. rat hepatocytes were treated with a wide concentration range of monoglyme up to concentrations demonstrating cytotoxicity in the assay system. Treatment did not produce either statistically significant or dose-related increases in the amount of UDS activity as measured by radioactive thymidine uptake
Reproductive toxicity: Adverse effects to reproduction are based on metabolism of monoglyme to 2-methoxy acetic acid, a compound that is known to interfere with sperm production. Adverse effects on the conceptus, including embryo lethality are also caused by this metabolite. Direct specific effects on female reproduction are not known to result from 2-methoxyacetic acid and thus are not expected from monoglyme exposure
Developmental toxicity:, Monoglyme appears to be a specific developmental toxin in rats and mice. Results in rats show that 120 mg/kg/day or more was associated with 100% foetal death and doses of 30 or 60 mg/kg
were foetotoxic but did not produce major malformations . This suggests that monoglyme is a specific developmental toxin as would be anticipated based on its metabolism to 2-methoxy acetic acid.

LPG (LIQUEFIED PETROLEUM GAS) &	No significant acute toxicological data identified in literature search.

Continued...

STYRENE/ BUTADIENE COPOLYMER, HYDROGENATED			
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

HEROGRIP EnviroMight Canister Adhesive

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

LPG (liquefied petroleum gas)

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

1,2-dimethoxyethane

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	9120mg/l	2
NOEC(ECx)	504h	Crustacea	320mg/l	2
EC50	48h	Crustacea	4000mg/l	2
LC50	96h	Fish	>500mg/l	2

naphtha petroleum, light, hydrotreated

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.64mg/l	2
NOEC(ECx)	504h	Crustacea	0.17mg/l	2
LC50	96h	Fish	0.11mg/l	2
EC50	96h	Algae or other aquatic plants	64mg/l	2

styrene/ butadiene copolymer, hydrogenated

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

Legend: ***Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data***

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
1,2-dimethoxyethane	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
1,2-dimethoxyethane	LOW (LogKOW = -0.21)

Mobility in soil

Ingredient	Mobility
1,2-dimethoxyethane	HIGH (Log KOC = 1)

SECTION 13 Disposal considerations

Waste treatment methods

Continued...

Product / Packaging disposal	▸ **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. ▸ Evaporate or incinerate residue at an approved site. ▸ Return empty containers to supplier. ▸ Ensure damaged or non-returnable cylinders are gas-free before disposal.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	2YE

Land transport (ADG)

14.1. **UN number or ID number**	3501
14.2. **UN proper shipping name**	CHEMICAL UNDER PRESSURE, FLAMMABLE, N.O.S.
14.3. **Transport hazard class(es)**	Class: 2.1 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Special provisions: 274 362 Limited quantity: 0

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3501
14.2. **UN proper shipping name**	Chemical under pressure, flammable, n.o.s. *
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 2.1 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 10L
14.4. **Packing group**	Not Applicable
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Special provisions: A1 A187 Cargo Only Packing Instructions: 218 Cargo Only Maximum Qty / Pack: 75 kg Passenger and Cargo Packing Instructions: Forbidden Passenger and Cargo Maximum Qty / Pack: Forbidden Passenger and Cargo Limited Quantity Packing Instructions: Forbidden Passenger and Cargo Limited Maximum Qty / Pack: Forbidden

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	3501
14.2. **UN proper shipping name**	CHEMICAL UNDER PRESSURE, FLAMMABLE, N.O.S.
14.3. **Transport hazard class(es)**	IMDG Class: 2.1 IMDG Subsidiary Hazard: Not Applicable
14.4. **Packing group**	Not Applicable
14.5 **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	EMS Number: F-D , S-U Special provisions: 274 362 Limited Quantities: 0

Continued...

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

Not Applicable

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

Product name	Group
LPG (liquefied petroleum gas)	Not Available
1,2-dimethoxyethane	Not Available
naphtha petroleum, light, hydrotreated	Not Available
styrene/ butadiene copolymer, hydrogenated	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
LPG (liquefied petroleum gas)	Not Available
1,2-dimethoxyethane	Not Available
naphtha petroleum, light, hydrotreated	Not Available
styrene/ butadiene copolymer, hydrogenated	Not Available

SECTION 15 Regulatory information

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

LPG (liquefied petroleum gas) is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

1,2-dimethoxyethane is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

naphtha petroleum, light, hydrotreated is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
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 - Not Classified as Carcinogenic

styrene/ butadiene copolymer, hydrogenated is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (LPG (liquefied petroleum gas); 1,2-dimethoxyethane; naphtha petroleum, light, hydrotreated; styrene/ butadiene copolymer, hydrogenated)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (styrene/ butadiene copolymer, hydrogenated)
Japan - ENCS	No (naphtha petroleum, light, hydrotreated)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	No (styrene/ butadiene copolymer, hydrogenated)
Legend:	***Yes = All CAS declared ingredients are on the inventory***

Continued...

National Inventory	Status
	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	18/07/2024
Initial Date	18/05/2022

SDS Version Summary

Version	Date of Update	Sections Updated
3.6	18/07/2024	Hazards identification - Classification, Composition / information on ingredients - Ingredients, Name

Other information

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
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration

- 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