

# TensorGrip X41 Canister Spray Adhesive

## QUIN GLOBAL ASIA PACIFIC

Version No: 5.5

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Chemwatch Hazard Alert Code: 4

Initial Date: 27/04/2022

Revision Date: 03/07/2025

Print Date: 03/07/2025

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

#### Product Identifier

Product name	TensorGrip X41 Canister Spray Adhesive
Synonyms	Not Available
Proper shipping name	CHEMICAL UNDER PRESSURE, FLAMMABLE, N.O.S. (contains dimethyl ether)
Other means of identification	Not Available

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

Relevant identified uses	Adhesive
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#### Details of the manufacturer or importer 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	<a href="http://www.quinglobal.com">www.quinglobal.com</a>
Email	sales@quinglobal.com.au

#### Emergency telephone number

Association / Organisation	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone number(s)	+61 1800 951 288 (ID#: 9-985004)
Other emergency telephone number(s)	+61 3 9573 3188

### SECTION 2 Hazards identification

#### Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification <sup>[1]</sup>	Flammable gases, Hazard Category 1A, Gases Under Pressure (Liquefied Gas), Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 2
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.
H280	Contains gas under pressure; may explode if heated.
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H336	May cause drowsiness or dizziness.
H411	Toxic to aquatic life with long lasting effects.
AUH044	Risk of explosion if heated under confinement.

## TensorGrip X41 Canister Spray Adhesive

## Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P271	Use only outdoors or in a well-ventilated area.
P261	Avoid breathing gas.
P273	Avoid release to the environment.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
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.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P381	In case of leakage, eliminate all ignition sources.
P391	Collect spillage.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

## Precautionary statement(s) Storage

P405	Store locked up.
P410+P403	Protect from sunlight. Store in a well-ventilated place.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

## Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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No further product hazard information.

## SECTION 3 Composition / information on ingredients

## Substances

See section below for composition of Mixtures

## Mixtures

CAS No	%[weight]	Name
67-64-1	20	<u>acetone</u>
79-20-9	5	<u>methyl acetate</u>
64742-49-0.	30	<u>naphtha petroleum, light, hydrotreated</u>
9003-55-8	15	<u>styrene/ butadiene rubber</u>
115-10-6	30	<u>dimethyl ether</u>
<b>Legend:</b>	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

<b>Eye Contact</b>	<ul style="list-style-type: none"> <li>▶ If product comes in contact with eyes remove the patient from gas source or contaminated area.</li> <li>▶ Take the patient to the nearest eye wash, shower or other source of clean water.</li> <li>▶ Open the eyelid(s) wide to allow the material to evaporate.</li> <li>▶ Gently rinse the affected eye(s) with clean, cool water for at least 15 minutes. Have the patient lie or sit down and tilt the head back. Hold the eyelid(s) open and pour water slowly over the eyeball(s) at the inner corners, letting the water run out of the outer corners.</li> <li>▶ The patient may be in great pain and wish to keep the eyes closed. It is important that the material is rinsed from the eyes to prevent further damage.</li> <li>▶ Ensure that the patient looks up, and side to side as the eye is rinsed in order to better reach all parts of the eye(s)</li> <li>▶ Transport to hospital or doctor.</li> <li>▶ Even when no pain persists and vision is good, a doctor should examine the eye as delayed damage may occur.</li> <li>▶ If the patient cannot tolerate light, protect the eyes with a clean, loosely tied bandage.</li> <li>▶ Ensure verbal communication and physical contact with the patient.</li> </ul> <p><b>DO NOT</b> allow the patient to rub the eyes  <b>DO NOT</b> allow the patient to tightly shut the eyes  <b>DO NOT</b> introduce oil or ointment into the eye(s) without medical advice  <b>DO NOT</b> use hot or tepid water.</p>
<b>Skin Contact</b>	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> <li>▶ Immediately remove all contaminated clothing, including footwear.</li> <li>▶ Flush skin and hair with running water (and soap if available).</li> <li>▶ Seek medical attention in event of irritation.</li> </ul> <p>In case of cold burns (frost-bite):</p> <ul style="list-style-type: none"> <li>▶ Move casualty into warmth before thawing the affected part; if feet are affected carry if possible</li> <li>▶ Bathe the affected area immediately in luke-warm water (not more than 35 deg C) for 10 to 15 minutes, immersing if possible and without rubbing</li> <li>▶ <b>DO NOT</b> apply hot water or radiant heat.</li> </ul>

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## TensorGrip X41 Canister Spray Adhesive

	<ul style="list-style-type: none"> <li>▶ Apply a clean, dry, light dressing of 'fluffed-up' dry gauze bandage</li> <li>▶ If a limb is involved, raise and support this to reduce swelling</li> <li>▶ If an adult is involved and where intense pain occurs provide pain killers such as paracetamol</li> <li>▶ Transport to hospital, or doctor</li> <li>▶ Subsequent blackening of the exposed tissue indicates potential of necrosis, which may require amputation.</li> </ul> <p>For thermal burns:</p> <ul style="list-style-type: none"> <li>▶ Decontaminate area around burn.</li> <li>▶ Consider the use of cold packs and topical antibiotics.</li> </ul> <p>For first-degree burns (affecting top layer of skin)</p> <ul style="list-style-type: none"> <li>▶ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides.</li> <li>▶ Use compresses if running water is not available.</li> <li>▶ Cover with sterile non-adhesive bandage or clean cloth.</li> <li>▶ Do NOT apply butter or ointments; this may cause infection.</li> <li>▶ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur.</li> </ul> <p>For second-degree burns (affecting top two layers of skin)</p> <ul style="list-style-type: none"> <li>▶ Cool the burn by immerse in cold running water for 10-15 minutes.</li> <li>▶ Use compresses if running water is not available.</li> <li>▶ Do NOT apply ice as this may lower body temperature and cause further damage.</li> <li>▶ Do NOT break blisters or apply butter or ointments; this may cause infection.</li> <li>▶ Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape.</li> </ul> <p>To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort):</p> <ul style="list-style-type: none"> <li>▶ Lay the person flat.</li> <li>▶ Elevate feet about 12 inches.</li> <li>▶ Elevate burn area above heart level, if possible.</li> <li>▶ Cover the person with coat or blanket.</li> <li>▶ Seek medical assistance.</li> </ul> <p>For third-degree burns</p> <p>Seek immediate medical or emergency assistance.</p> <p>In the mean time:</p> <ul style="list-style-type: none"> <li>▶ Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound.</li> <li>▶ Separate burned toes and fingers with dry, sterile dressings.</li> <li>▶ Do not soak burn in water or apply ointments or butter; this may cause infection.</li> <li>▶ To prevent shock see above.</li> <li>▶ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway.</li> <li>▶ Have a person with a facial burn sit up.</li> <li>▶ Check pulse and breathing to monitor for shock until emergency help arrives.</li> </ul>
Inhalation	<ul style="list-style-type: none"> <li>▶ Following exposure to gas, remove the patient from the gas source or contaminated area.</li> <li>▶ NOTE: Personal Protective Equipment (PPE), including positive pressure self-contained breathing apparatus may be required to assure the safety of the rescuer.</li> <li>▶ Prostheses such as false teeth, which may block the airway, should be removed, where possible, prior to initiating first aid procedures.</li> <li>▶ If the patient is not breathing spontaneously, administer rescue breathing.</li> <li>▶ If the patient does not have a pulse, administer CPR.</li> <li>▶ If medical oxygen and appropriately trained personnel are available, administer 100% oxygen.</li> <li>▶ Summon an emergency ambulance. If an ambulance is not available, contact a physician, hospital, or Poison Control Centre for further instruction.</li> <li>▶ Keep the patient warm, comfortable and at rest while awaiting medical care.</li> <li>▶ <b>MONITOR THE BREATHING AND PULSE, CONTINUOUSLY.</b></li> <li>▶ Administer rescue breathing (preferably with a demand-valve resuscitator, bag-valve mask-device, or pocket mask as trained) or CPR if necessary.</li> </ul>
Ingestion	<ul style="list-style-type: none"> <li>▶ Not considered a normal route of entry.</li> <li>▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.</li> </ul>

## Indication of any immediate medical attention and special treatment needed

For petroleum distillates

· In case of ingestion, gastric lavage with activated charcoal can be used promptly to prevent absorption - decontamination (induced emesis or lavage) is controversial and should be considered on the merits of each individual case; of course the usual precautions of an endotracheal tube should be considered prior to lavage, to prevent aspiration.

· Individuals intoxicated by petroleum distillates should be hospitalized immediately, with acute and continuing attention to neurologic and cardiopulmonary function.

· Positive pressure ventilation may be necessary.

· Acute central nervous system signs and symptoms may result from large ingestions of aspiration-induced hypoxia.

· After the initial episode, individuals should be followed for changes in blood variables and the delayed appearance of pulmonary oedema and chemical pneumonitis. Such patients should be followed for several days or weeks for delayed effects, including bone marrow toxicity, hepatic and renal impairment. Individuals with chronic pulmonary disease will be more seriously impaired, and recovery from inhalation exposure may be complicated.

· Gastrointestinal symptoms are usually minor and pathological changes of the liver and kidneys are reported to be uncommon in acute intoxications.

· Chlorinated and non-chlorinated hydrocarbons may sensitize the heart to epinephrine and other circulating catecholamines so that arrhythmias may occur. Careful consideration of this potential adverse effect should precede administration of epinephrine or other cardiac stimulants and the selection of bronchodilators.

for lower alkyl ethers:

## BASIC TREATMENT

- ▶ Establish a patent airway with suction where necessary.
- ▶ Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- ▶ Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- ▶ A low-stimulus environment must be maintained.
- ▶ Monitor and treat, where necessary, for shock.
- ▶ Anticipate and treat, where necessary, for seizures.
- ▶ **DO NOT use emetics.** Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.

## ADVANCED TREATMENT

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- ▶ Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Hypotension without signs of hypovolaemia may require vasopressors.
- ▶ Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

## EMERGENCY DEPARTMENT

## TensorGrip X41 Canister Spray Adhesive

- ▶ Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- ▶ Ethers may produce anion gap acidosis. Hyperventilation and bicarbonate therapy might be indicated.
- ▶ Haemodialysis might be considered in patients with impaired renal function.
- ▶ Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L.

EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For gas exposures:

## BASIC TREATMENT

- ▶ Establish a patent airway with suction where necessary.
- ▶ Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- ▶ Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- ▶ Monitor and treat, where necessary, for pulmonary oedema.
- ▶ Monitor and treat, where necessary, for shock.
- ▶ Anticipate seizures.

## ADVANCED TREATMENT

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- ▶ Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- ▶ Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

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## SECTION 5 Firefighting measures

## Extinguishing media

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

**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.
- ▶ **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

## FOR FIRES INVOLVING MANY GAS CYLINDERS:

- ▶ To stop the flow of gas, specifically trained personnel may inert the atmosphere to reduce oxygen levels thus allowing the capping of leaking container(s).
- ▶ Reduce the rate of flow and inject an inert gas, if possible, before completely stopping the flow to prevent flashback.
- ▶ **DO NOT extinguish the fire until the supply is shut off** otherwise an explosive re-ignition may occur.
- ▶ If the fire is extinguished and the flow of gas continues, used increased ventilation to prevent build-up, of explosive atmosphere.
- ▶ Use non-sparking tools to close container valves.
- ▶ Be CAUTIOUS of a Boiling Liquid Evaporating Vapour Explosion, *BLEVE*, if fire is impinging on surrounding containers.
- ▶ Direct 2500 litre/min (500 gpm) water stream onto containers above liquid level with the assistance remote monitors.

## GENERAL

- ▶ 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 FIGHTING PROCEDURES:

- ▶ The only safe way to extinguish a flammable gas fire is to stop the flow of gas.
- ▶ If the flow cannot be stopped, allow the entire contents of the cylinder to burn while cooling the cylinder and surroundings with water from a suitable distance.
- ▶ Extinguishing the fire without stopping the gas flow may permit the formation of ignitable or explosive mixtures with air. These mixtures may propagate to a source of ignition.

## SPECIAL HAZARDS

- ▶ Excessive pressures may develop in a gas cylinder exposed in a fire; this may result in explosion.
- ▶ Cylinders with pressure relief devices may release their contents as a result of fire and the released gas may constitute a further source of hazard for the fire-fighter.

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## TensorGrip X41 Canister Spray Adhesive

	<p>► Cylinders without pressure-relief valves have no provision for controlled release and are therefore more likely to explode if exposed to fire.</p> <p>-----</p> <p>FIRE FIGHTING REQUIREMENTS:</p> <p>-----</p> <p>The need for proximity, entry and flash-over protection and special protective clothing should be determined for each incident, by a competent fire-fighting safety professional.</p>
Fire/Explosion Hazard	<p>► <b>HIGHLY FLAMMABLE:</b> will be easily ignited by heat, sparks or flames.</p> <p>► Will form explosive mixtures with air</p> <p>► Fire exposed containers may vent contents through pressure relief valves thereby increasing fire intensity and/ or vapour concentration.</p> <p>► Vapours may travel to source of ignition and flash back.</p> <p>► Containers may explode when heated - Ruptured cylinders may rocket</p> <p>► Fire may produce irritating, poisonous or corrosive gases.</p> <p>► Runoff may create fire or explosion hazard.</p> <p>► May decompose explosively when heated or involved in fire.</p> <p>► High concentration of gas may cause asphyxiation without warning.</p> <p>► Contact with gas may cause burns, severe injury and/ or frostbite.</p> <p>Combustion products include:</p> <p>carbon monoxide (CO)</p> <p>carbon dioxide (CO<sub>2</sub>)</p> <p>other pyrolysis products typical of burning organic material.</p> <p><b>Contains low boiling substance:</b> Closed containers may rupture due to pressure buildup under fire conditions.</p> <p>► Vented gas is more dense than air and may collect in pits, basements.</p>
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## 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	<p>► Avoid breathing vapour and any contact with liquid or gas. Protective equipment including respirator should be used.</p> <p>► <b>DO NOT enter confined spaces where gas may have accumulated.</b></p> <p>► Shut off all sources of possible ignition and increase ventilation.</p> <p>► Clear area of personnel.</p> <p>► Stop leak only if safe to do so.</p> <p>► Remove leaking cylinders to safe place. release pressure under safe controlled conditions by opening valve.</p> <p>► Orientate cylinder so that the leak is gas, not liquid, to minimise rate of leakage</p> <p>► Keep area clear of personnel until gas has dispersed.</p>
Major Spills	<p>► Clear area of all unprotected personnel and move upwind.</p> <p>► Alert Emergency Authority and advise them of the location and nature of hazard.</p> <p>► May be violently or explosively reactive.</p> <p>► Wear full body clothing with breathing apparatus.</p> <p>► Prevent by any means available, spillage from entering drains and water-courses.</p> <p>► Consider evacuation.</p> <p>► Shut off all possible sources of ignition and increase ventilation.</p> <p>► No smoking or naked lights within area.</p> <p>► Use extreme caution to prevent violent reaction.</p> <p>► Stop leak only if safe to do so.</p> <p>► Water spray or fog may be used to disperse vapour.</p> <p>► DO NOT enter confined space where gas may have collected.</p> <p>► Keep area clear until gas has dispersed.</p> <p>► Remove leaking cylinders to a safe place.</p> <p>► Fit vent pipes. Release pressure under safe, controlled conditions</p> <p>► Burn issuing gas at vent pipes.</p> <p>► DO NOT exert excessive pressure on valve; DO NOT attempt to operate damaged valve.</p>

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

## SECTION 7 Handling and storage

## Precautions for safe handling

Safe handling	<p>The conductivity of this material may make it a static accumulator., A liquid is typically considered nonconductive if its conductivity is below 100 pS/m and is considered semi-conductive if its conductivity is below 10 000 pS/m., Whether a liquid is nonconductive or semi-conductive, the precautions are the same., A number of factors, for example liquid temperature, presence of contaminants, and anti-static additives can greatly influence the conductivity of a liquid.</p> <p>► Containers, even those that have been emptied, may contain explosive vapours.</p> <p>► Do NOT cut, drill, grind, weld or perform similar operations on or near containers.</p> <p>· Electrostatic discharge may be generated during pumping - this may result in fire.</p> <p>· Ensure electrical continuity by bonding and grounding (earthing) all equipment.</p> <p>· Restrict line velocity during pumping in order to avoid generation of electrostatic discharge (&lt;=1 m/sec until fill pipe submerged to twice its diameter, then &lt;= 7 m/sec).</p> <p>· Avoid splash filling.</p> <p>· Do NOT use compressed air for filling discharging or handling operations.</p> <p>· Wait 2 minutes after tank filling (for tanks such as those on</p> <p>· road tanker vehicles) before opening hatches or manholes.</p> <p>· Wait 30 minutes after tank filling ( for large storage tanks)</p> <p>· before opening hatches or manholes. Even with proper</p> <p>· grounding and bonding, this material can still accumulate an</p> <p>· electrostatic charge. If sufficient charge is allowed to</p> <p>· accumulate, electrostatic discharge and ignition of flammable</p> <p>· air-vapour mixtures can occur. Be aware of handling</p> <p>· operations that may give rise to additional hazards that result</p>
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## TensorGrip X41 Canister Spray Adhesive

	<ul style="list-style-type: none"> <li>from the accumulation of static charges. These include but are</li> <li>not limited to pumping (especially turbulent flow), mixing,</li> <li>filtering, splash filling, cleaning and filling of tanks and</li> <li>containers, sampling, switch loading, gauging, vacuum truck</li> <li>operations, and mechanical movements. These activities may</li> <li>lead to static discharge e.g. spark formation. Restrict line</li> <li>velocity during pumping in order to avoid generation of</li> <li>electrostatic discharge (= 1 m/s until fill pipe submerged to</li> <li>twice its diameter, then = 7 m/s). Avoid splash filling.</li> <li>Do NOT use compressed air for filling, discharging, or handling operations</li> <li>Consider use in closed pressurised systems, fitted with temperature, pressure and safety relief valves which are vented for safe dispersal.</li> </ul> <p>Use only properly specified equipment which is suitable for this product, its supply pressure and temperature</p> <ul style="list-style-type: none"> <li>The tubing network design connecting gas cylinders to the delivery system should include appropriate pressure indicators and vacuum or suction lines.</li> <li>Fully-welded types of pressure gauges, where the bourdon tube sensing element is welded to the gauge body, are recommended.</li> <li>Before connecting gas cylinders, ensure manifold is mechanically secure and does not contain another gas. Before disconnecting gas cylinder, isolate supply line segment proximal to cylinder, remove trapped gas in supply line with aid of vacuum pump</li> <li>When connecting or replacing cylinders take care to avoid airborne particulates violently ejected when system pressurises.</li> <li>Consider the use of doubly-contained piping; diaphragm or bellows sealed, soft seat valves; backflow prevention devices; flash arrestors; and flow monitoring or limiting devices. Gas cabinets, with appropriate exhaust treatment, are recommended, as is automatic monitoring of the secondary enclosures and work areas for release.</li> <li>Use a pressure reducing regulator when connecting cylinder to lower pressure (&lt;100 psig) piping or systems</li> <li>Use a check valve or trap in the discharge line to prevent hazardous back-flow into the cylinder</li> <li>Check regularly for spills or leaks. Keep valves tightly closed but do not apply extra leverage to hand wheels or cylinder keys.</li> <li>Open valve slowly. If valve is resistant to opening then contact your supervisor</li> <li>Valve protection caps must remain in place must remain in place unless container is secured with valve outlet piped to use point.</li> <li>Never insert a pointed object (e.g. hooks) into cylinder cap openings as a means to open cap or move cylinder. Such action can inadvertently turn the valve and gas a gas leak. Use an adjustable strap instead of wrench to free an over-tight or rusted cap.</li> <li>A bubble of gas may buildup behind the outlet dust cap during transportation, after prolonged storage, due to defective cylinder valve or if a dust cap is inserted without adequate evacuation of gas from the line. When loosening dust cap, preferably stand cylinder in a suitable enclosure and take cap off slowly. Never face the dust cap directly when removing it; point cap away from any personnel or any object that may pose a hazard. under negative pressure (relative to atmospheric gas)</li> <li>Suck back of water into the container must be prevented. Do not allow backfeed into the container.</li> <li>Do NOT drag, slide or roll cylinders - use a suitable hand truck for cylinder movement</li> <li>Test for leakage with brush and detergent - <b>NEVER use a naked flame.</b></li> <li><b>Do NOT heat cylinder by any means to increase the discharge rate of product from cylinder.</b></li> <li>Leaking gland nuts may be tightened if necessary.</li> <li>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.</li> <li>Obtain a work permit before attempting any repairs.</li> <li><b>DO NOT attempt repair work on lines, vessels under pressure.</b></li> <li>Atmospheres must be tested and O.K. before work resumes after leakage.             <ul style="list-style-type: none"> <li>Avoid generation of static electricity. Earth all lines and equipment.</li> <li><b>DO NOT transfer gas from one cylinder to another.</b></li> </ul> </li> </ul>
Other information	<ul style="list-style-type: none"> <li>Cylinders should be stored in a purpose-built compound with good ventilation, preferably in the open.</li> <li>Such compounds should be sited and built in accordance with statutory requirements.</li> <li>The storage compound should be kept clear and access restricted to authorised personnel only.</li> <li>Cylinders stored in the open should be protected against rust and extremes of weather.</li> <li>Cylinders in storage should be properly secured to prevent toppling or rolling.</li> <li>Cylinder valves should be closed when not in use.</li> <li>Where cylinders are fitted with valve protection this should be in place and properly secured.</li> <li>Gas cylinders should be segregated according to the requirements of the Dangerous Goods Act(s).</li> <li>Cylinders containing flammable gases should be stored away from other combustible materials. Alternatively a fire-resistant partition may be used.</li> <li>Check storage areas for flammable or hazardous concentrations of gases prior to entry.</li> <li>Preferably store full and empty cylinders separately.</li> <li>Full cylinders should be arranged so that the oldest stock is used first.</li> <li>Cylinders in storage should be checked periodically for general condition and leakage.</li> <li>Protect cylinders against physical damage. Move and store cylinders correctly as instructed for their manual handling.</li> </ul> <p><b>NOTE:</b> A 'G' size cylinder is usually too heavy for an inexperienced operator to raise or lower.</p>

## Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> <li>Cylinder:</li> <li>Ensure the use of equipment rated for cylinder pressure.</li> <li>Ensure the use of compatible materials of construction.</li> <li>Valve protection cap to be in place until cylinder is secured, connected.</li> <li>Cylinder must be properly secured either in use or in storage.</li> <li>Cylinder valve must be closed when not in use or when empty.</li> <li>Segregate full from empty cylinders.</li> </ul> <p><b>WARNING:</b> Suckback into cylinder may result in rupture. Use back-flow preventive device in piping.</p>
Storage incompatibility	<p>Dimethyl ether:</p> <ul style="list-style-type: none"> <li>is a peroxidisable gas</li> <li>may be heat and shock sensitive</li> <li>is able to form unstable peroxides on prolonged exposure to air</li> <li>reacts violently with oxidisers, aluminium hydride, lithium aluminium hydride</li> <li>is incompatible with strong acids, metal salts</li> </ul> <p>Low molecular weight alkanes:</p> <ul style="list-style-type: none"> <li>May react violently with strong oxidisers, chlorine, chlorine dioxide, dioxygenyl tetrafluoroborate.</li> <li>May react with oxidising materials, nickel carbonyl in the presence of oxygen, heat.</li> <li>Are incompatible with nitronium tetrafluoroborate(1-), halogens and interhalogens</li> <li>may generate electrostatic charges, due to low conductivity, on flow or agitation.</li> <li>Avoid flame and ignition sources</li> </ul> <p>Redox reactions of alkanes, in particular with oxygen and the halogens, are possible as the carbon atoms are in a strongly reduced condition. Reaction with oxygen (if present in sufficient quantity to satisfy the reaction stoichiometry) leads to combustion without any smoke, producing carbon dioxide and water. Free radical halogenation reactions occur with halogens, leading to the production of haloalkanes. In addition, alkanes have been shown to interact with, and bind to, certain transition metal complexes. Interaction between chlorine and ethane over activated carbon at 350 deg C has caused explosions, but added carbon dioxide reduces the risk. The violent interaction of liquid chlorine injected into ethane at 80 deg C/10 bar becomes very violent if ethylene is also present. A mixture prepared at -196 deg C with either methane or ethane exploded when the temp was raised to -78 deg C. Addition of nickel carbonyl to an n-butane-oxygen mixture causes an explosion at 20-40 deg C. Alkanes will react with steam in the presence of a nickel catalyst to give hydrogen.</p>

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## TensorGrip X41 Canister Spray Adhesive

- ▶ Esters react with acids to liberate heat along with alcohols and acids.
- ▶ Strong oxidising acids may cause a vigorous reaction with esters that is sufficiently exothermic to ignite the reaction products.
- ▶ Heat is also generated by the interaction of esters with caustic solutions.
- ▶ Flammable hydrogen is generated by mixing esters with alkali metals and hydrides.
- ▶ Esters may be incompatible with aliphatic amines and nitrates.

**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	acetone	Acetone	500 ppm / 1185 mg/m3	2375 mg/m3 / 1000 ppm	Not Available	Not Available
Australia Exposure Standards	methyl acetate	Methyl acetate	200 ppm / 606 mg/m3	757 mg/m3 / 250 ppm	Not Available	Not Available
Australia Exposure Standards	dimethyl ether	Dimethyl ether	400 ppm / 760 mg/m3	950 mg/m3 / 500 ppm	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
acetone	2,500 ppm	Not Available
methyl acetate	Not Available	Not Available
naphtha petroleum, light, hydrotreated	Not Available	Not Available
styrene/ butadiene rubber	Not Available	Not Available
dimethyl ether	Not Available	Not Available

**MATERIAL DATA**

for methyl acetate:

Odour Threshold Value: 182 ppm (detection), 297 ppm (recognition)

Methyl acetate is metabolised to methanol in manner proportional to the exposure level and the TLV-TWA is analogous to that proposed for methanol. The TLV-TWA is thought to be protective against narcosis, eye and skin irritation and pulmonary irritation.

Odour Safety Factor(OSF)

OSF=43 (METHYL ACETATE)

for dimethyl ether:

The no-effect-level for dimethyl ether is somewhere between 2000 ppm (rabbits) and 50,000 ppm (humans) with possible cardiac sensitisation occurring around 200,000 ppm (dogs). The AIHA has adopted a safety factor of 100 in respect to the 50,000 ppm level in its recommendation for a workplace environmental exposure level (WEEL) which is thought to protect against both narcotic and sensitising effects. This level is consistent with the TLV-TWA of 400 ppm for diethyl ether and should be easily achievable using current technologies. The use of the traditionally allowable excursion of 1.25 to the level of 6.25 ppm is felt to be more than adequate as an upper safe limit of exposure.

Human data:

50,000 ppm (12 mins): Feelings of mild intoxication.

75,000 ppm (12 mins): As above plus slight lack of attenuation.

82,000 ppm (12 mins): Some incoordination, slight blurring of vision

(30 mins): As above plus analgesia of the face and rushing of blood to the face.

100,000 ppm (10-20 mins): Narcotic symptoms; (64 mins): Sickness (assumed to be nausea)

144,000 ppm (36 mins): Unconsciousness

Odour Threshold Value: 3.6 ppm (detection), 699 ppm (recognition)

Saturation vapour concentration: 237000 ppm @ 20 °C

NOTE: Detector tubes measuring in excess of 40 ppm, are available.

Exposure at or below the recommended TLV-TWA is thought to protect the worker against mild irritation associated with brief exposures and the bioaccumulation, chronic irritation of the respiratory tract and headaches associated with long-term acetone exposures. The NIOSH REL-TWA is substantially lower and has taken into account slight irritation experienced by volunteer subjects at 300 ppm. Mild irritation to acclimatised workers begins at about 750 ppm - unacclimatised subjects will experience irritation at about 350-500 ppm but acclimatisation can occur rapidly. Disagreement between the peak bodies is based largely on the view by ACGIH that widespread use of acetone, without evidence of significant adverse health effects at higher concentrations, allows acceptance of a higher limit.

Half-life of acetone in blood is 3 hours which means that no adjustment for shift-length has to be made with reference to the standard 8 hour/day, 40 hours per week because body clearance occurs within any shift with low potential for accumulation.

A STEL has been established to prevent excursions of acetone vapours that could cause depression of the central nervous system.

Odour Safety Factor(OSF)

OSF=38 (ACETONE)

for heptane (all isomers)

The TLV-TWA is protective against narcotic and irritant effects which are greater than those of pentane or n-hexane but less than those of octane. The TLV-TWA applies to all isomers.

Inhalation by humans of 1000 ppm for 6 minutes produced slight dizziness. Higher concentrations for shorter periods produce marked vertigo, incoordination and hilarity. Signs of central nervous system depression occur in the absence of mucous membrane irritation. Brief exposures to high levels (5000 ppm for 4 minutes) produce nausea, loss of appetite and a 'gasoline-like' taste in the mouth that persists for many hours after exposure ceases


May act as a simple asphyxiants; these are gases which, when present in high concentrations, reduce the oxygen content in air below that required to support breathing, consciousness and life; loss of consciousness, with death by suffocation may rapidly occur in an oxygen deficient atmosphere.

**CARE:** Most simple asphyxiants are odourless or possess low odour and there is no warning on entry into an oxygen deficient atmosphere. If there is any doubt, oxygen content can be checked simply and quickly. It may not be appropriate to only recommend an exposure standard for simple asphyxiants rather it is essential that sufficient oxygen be maintained. Air normally has 21 percent oxygen by volume, with 18 percent regarded as minimum under normal atmospheric pressure to maintain consciousness / life. At pressures significantly higher or lower than normal atmospheric pressure, expert guidance should be sought.

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## TensorGrip X41 Canister Spray Adhesive

## Exposure controls

<p><b>Appropriate engineering controls</b></p>	<p><b>CARE:</b> Use of a quantity of this material in confined space or poorly ventilated area, where rapid build up of concentrated atmosphere may occur, could require increased ventilation and/or protective gear</p> <p>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:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <ul style="list-style-type: none"> <li>Areas where cylinders are stored require good ventilation and, if enclosed need discrete/ controlled exhaust ventilation.</li> <li>Vented gas is flammable, and may spread from its origin. Vent path must not contain ignition sources, pilot lights, naked flames.</li> <li>Secondary containment and exhaust gas treatment may be required by certain jurisdictions.</li> <li>Local exhaust ventilation (explosion proof) is usually required in workplaces.</li> <li>Consideration should be given to the use of doubly-contained piping; diaphragm or bellows-sealed, soft-seat valves; backflow prevention devices; flash arrestors and flow- monitoring or limiting devices.</li> <li>Automated controls should ensure that workplace atmospheres do not exceed 25% of the lower explosive limit (LEL) (if available).</li> <li>Monitor the work area and secondary containments for release of gas.</li> <li>Automated alerting systems with automatic shutdown of gas-flow may be appropriate and may in fact be mandatory in certain jurisdictions.</li> <li>Respiratory protection in the form of air-supplied or self-contained breathing equipment must be worn if the oxygen concentration in the workplace air is less than 19%.</li> <li>Cartridge respirators <b>DO NOT</b> give protection and may result in rapid suffocation.</li> </ul> <p>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.</p> <table border="1"> <tr> <td>Type of Contaminant:</td><td>Air Speed:</td></tr> <tr> <td>gas discharge (active generation into zone of rapid air motion)</td><td>1-2.5 m/s (200-500 f/min.)</td></tr> </table> <p>Within each range the appropriate value depends on:</p> <table border="1"> <tr> <td>Lower end of the range</td><td>Upper end of the range</td></tr> <tr> <td>1: Room air currents minimal or favourable to capture</td><td>1: Disturbing room air currents</td></tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td><td>2: Contaminants of high toxicity</td></tr> <tr> <td>3: Intermittent, low production.</td><td>3: High production, heavy use</td></tr> <tr> <td>4: Large hood or large air mass in motion</td><td>4: Small hood-local control only</td></tr> </table> <p>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.</p> <ul style="list-style-type: none"> <li>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.</li> <li>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.</li> <li>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..</li> </ul> <p>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)</p>	Type of Contaminant:	Air Speed:	gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	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
Type of Contaminant:	Air Speed:														
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4: Large hood or large air mass in motion	4: Small hood-local control only														
<p><b>Individual protection measures, such as personal protective equipment</b></p>															
<p><b>Eye and face protection</b></p>	<ul style="list-style-type: none"> <li>Chemical goggles.</li> <li>Full face shield may be required for supplementary but never for primary protection of eyes.</li> <li>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]</li> </ul>														
<p><b>Skin protection</b></p>	<p>See Hand protection below</p>														
<p><b>Hands/feet protection</b></p>	<p>For esters:</p> <ul style="list-style-type: none"> <li>Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials.</li> <li>Neoprene rubber gloves</li> <li>When handling sealed and suitably insulated cylinders wear cloth or leather gloves.</li> <li>Insulated gloves:</li> <li>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.</li> </ul>														
<p><b>Body protection</b></p>	<p>See Other protection below</p>														
<p><b>Other protection</b></p>	<ul style="list-style-type: none"> <li>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.</li> <li>Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost.</li> </ul> <p>BREThERICK: Handbook of Reactive Chemical Hazards.</p> <ul style="list-style-type: none"> <li>Protective overalls, closely fitted at neck and wrist.</li> <li>Eye-wash unit.</li> </ul> <p><b>IN CONFINED SPACES:</b></p> <ul style="list-style-type: none"> <li>Non-sparking protective boots</li> </ul>														

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TensorGrip X41 Canister Spray Adhesive

- ▶ 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:  
TensorGrip X41 Canister Spray Adhesive

Material	CPI
BUTYL	A
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
TEFLON	C
VITON/NEOPRENE	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 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	AX-AUS / Class 1 P2	-	AX-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	AX-2 P2	AX-PAPR-2 P2
up to 50 x ES	-	AX-3 P2	-
50+ 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**

\*\* - Continuous-flow or positive pressure demand.  
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 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)	0.709
Odour	Not Available	Partition coefficient n-octanol / water	Not Available

## TensorGrip X41 Canister Spray Adhesive

<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	350
<b>pH (as supplied)</b>	Not Available	<b>Decomposition temperature (°C)</b>	Not Applicable
<b>Melting point / freezing point (°C)</b>	-141.5	<b>Viscosity (cSt)</b>	95
<b>Initial boiling point and boiling range (°C)</b>	-24.8	<b>Molecular weight (g/mol)</b>	Not Available
<b>Flash point (°C)</b>	-41.1	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	HIGHLY FLAMMABLE.	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	18.2	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	3.4	<b>Volatile Component (%vol)</b>	Not Available
<b>Vapour pressure (kPa)</b>	63	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Immiscible	<b>pH as a solution (1%)</b>	Not Available
<b>Vapour density (Air = 1)</b>	1.6	<b>VOC g/L</b>	Not Available
<b>Heat of Combustion (kJ/g)</b>	Not Available	<b>Ignition Distance (cm)</b>	Not Available
<b>Flame Height (cm)</b>	Not Available	<b>Flame Duration (s)</b>	Not Available
<b>Enclosed Space Ignition Time Equivalent (s/m3)</b>	Not Available	<b>Enclosed Space Ignition Deflagration Density (g/m3)</b>	Not Available

## SECTION 10 Stability and reactivity

<b>Reactivity</b>	See section 7
<b>Chemical stability</b>	<ul style="list-style-type: none"> <li>▶ Unstable in the presence of incompatible materials.</li> <li>▶ Product is considered stable.</li> <li>▶ Hazardous polymerisation will not occur.</li> </ul>
<b>Possibility of hazardous reactions</b>	See section 7
<b>Conditions to avoid</b>	See section 7
<b>Incompatible materials</b>	See section 7
<b>Hazardous decomposition products</b>	See section 5

## SECTION 11 Toxicological information

## Information on toxicological effects

<b>a) Acute Toxicity</b>	Based on available data, the classification criteria are not met.
<b>b) Skin Irritation/Corrosion</b>	There is sufficient evidence to classify this material as skin corrosive or irritating.
<b>c) Serious Eye Damage/Irritation</b>	There is sufficient evidence to classify this material as eye damaging or irritating
<b>d) Respiratory or Skin sensitisation</b>	Based on available data, the classification criteria are not met.
<b>e) Mutagenicity</b>	Based on available data, the classification criteria are not met.
<b>f) Carcinogenicity</b>	Based on available data, the classification criteria are not met.
<b>g) Reproductivity</b>	Based on available data, the classification criteria are not met.
<b>h) STOT - Single Exposure</b>	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
<b>i) STOT - Repeated Exposure</b>	Based on available data, the classification criteria are not met.
<b>j) Aspiration Hazard</b>	Based on available data, the classification criteria are not met.

<b>Inhaled</b>	<p>The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as the molecular weights and boiling points increase. Central nervous system depression, headache, drowsiness, dizziness, coma and neurobehavioral changes may also be symptomatic of overexposure. Respiratory tract involvement may produce mucous membrane irritation, dyspnea, and tachypnea, pharyngitis, bronchitis, pneumonitis and, in massive exposures, pulmonary oedema (which may be delayed). Gastrointestinal effects include nausea, vomiting, diarrhoea and abdominal cramps. Liver and kidney damage may result from massive exposures.</p> <p>Inhalation hazard is increased at higher temperatures.</p> <p>High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms</p>
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## TensorGrip X41 Canister Spray Adhesive

	<p>such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitizers and may cause ventricular fibrillations.</p> <p>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.</p> <p>Ethers produce narcosis following inhalation.</p> <p>Inhalation of lower alkyl ethers may result in central nervous system depression or stimulation, intoxication, headache, dizziness, weakness, blurred vision, seizures and possible coma. Cardiovascular involvement may produce hypotension, bradycardia and cardiovascular collapse, whilst respiratory symptoms might include irritation of nose and throat, cough, laryngeal spasm, pharyngitis, irregular respiration, depression, pulmonary oedema and respiratory arrest. Nausea, vomiting and salivation might also indicate overexposure.</p> <p>Convulsions, respiratory distress or paralysis, asphyxia, pneumonitis, and unconsciousness are all serious manifestations of poisoning. Fatalities have been reported. Kidney and liver damage with interstitial cystitis may result from massive exposures.</p> <p>Some aliphatic hydrocarbons produce axonal neuropathies. Isoparaffinic hydrocarbons produce injury to the kidneys of male rats. When albino rats were exposed to isoparaffins at 21.4 mg/l for 4 hours, all animals experienced weakness, tremors, salivation, mild to moderate convulsions, chromodacryorrhoea and ataxia within the first 24 hours. Symptoms disappeared after 24 hours.</p> <p>Several studies have evaluated sensory irritation in laboratory animals or odor or sensory response in humans. When evaluated by a standard procedure to assess upper airway irritation, isoparaffins did not produce sensory irritation in mice exposed to up to 400 ppm isoparaffin in air. Human volunteers were exposed for six hours to 100 ppm isoparaffin. The subjects were given a self-administered questionnaire to evaluate symptoms, which included dryness of the mucous membranes, loss of appetite, nausea, vomiting, diarrhea, fatigue, headache, dizziness, feeling of inebriation, visual disturbances, tremor, muscular weakness, impairment of coordination or paresthesia. No symptoms associated with solvent exposure were observed. With a human expert panel, odour from liquid imaging copier emissions became weakly discernible at approximately 50 ppm.</p> <p>Numerous long-term exposures have been conducted in animals with only one major finding observed. Renal tubular damage has been found in kidneys of male rats upon repeated exposures to isoparaffins. It does not occur in mice or in female rats. This male rat nephropathy has been observed with a number of hydrocarbons, including wholly vaporized unleaded gasoline. The phenomenon has been attributed to reversible binding of hydrocarbon to alpha2-globulin. Since humans do not synthesize alpha2-globulin or a similar protein, the finding is not considered to be of biological significance to man. No clinically significant renal abnormalities have been found in refinery workers exposed to hydrocarbons.</p> <p>When evaluated for developmental toxicity in rats, isoparaffins were neither embryotoxic nor teratogenic. Isoparaffins were consistently negative on standard bacterial genotoxicity assays. They were also non-genotoxic in <i>in vivo</i> mammalian testing for somatic or germ cell mutations (mouse micronucleus test and rat dominant lethal assay, respectively).</p> <p>Mullin et al: Jnl Applied Toxicology 10, pp 136-142, 2006</p> <p>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</p> <p>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.</p> <p>Symptoms of asphyxia (suffocation) may include headache, dizziness, shortness of breath, muscular weakness, drowsiness and ringing in the ears. If the asphyxia is allowed to progress, there may be nausea and vomiting, further physical weakness and unconsciousness and, finally, convulsions, coma and death. Significant concentrations of the non-toxic gas reduce the oxygen level in the air. As the amount of oxygen is reduced from 21 to 14 volume %, the pulse rate accelerates and the rate and volume of breathing increase. The ability to maintain attention and think clearly is diminished and muscular coordination is somewhat disturbed. As oxygen decreases from 14-10% judgement becomes faulty; severe injuries may cause no pain. Muscular exertion leads to rapid fatigue. Further reduction to 6% may produce nausea and vomiting and the ability to move may be lost. Permanent brain damage may result even after resuscitation at exposures to this lower oxygen level. Below 6% breathing is in gasps and convulsions may occur. Inhalation of a mixture containing no oxygen may result in unconsciousness from the first breath and death will follow in a few minutes.</p> <p>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.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p>
Ingestion	<p>Ingestion of alkyl ethers may produce symptoms similar to those produced following inhalation.</p> <p>Not normally a hazard due to physical form of product.</p> <p>Considered an unlikely route of entry in commercial/industrial environments</p> <p>Many aliphatic hydrocarbons create a burning sensation because they are irritating to the GI mucosa. Vomiting has been reported in up to one third of all hydrocarbon exposures. While most aliphatic hydrocarbons have little GI absorption, aspiration frequently occurs, either initially or in a semi-delayed fashion as the patient coughs or vomits, thereby resulting in pulmonary effects. Once aspirated, the hydrocarbons can create a severe pneumonitis.</p> <p>Rats given isoparaffinic hydrocarbons - isoalkanes- (after 18-24 hours fasting) showed lethargy and/or general weakness, ataxia and diarrhoea. Symptoms disappeared within 24-28 hours.</p> <p>Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage. Accidental ingestion of the material may be damaging to the health of the individual.</p>
Skin Contact	<p>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.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Dermally, isoparaffins have produced slight to moderate irritation in animals and humans under occluded patch conditions where evaporation cannot freely occur. However, they are not irritating in non-occluded tests, which are a more realistic simulation of human exposure. They have not been found to be sensitizers in guinea pig or human patch testing. However, occasional rare idiosyncratic sensitisation reactions in humans have been reported.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>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.</p> <p>Alkyl ethers may defat and dehydrate the skin producing dermatoses. Absorption may produce headache, dizziness, and central nervous system depression.</p> <p>Vapourising liquid causes rapid cooling and contact may cause cold burns, frostbite, even through normal gloves. Frozen skin tissues are painless and appear waxy and yellow. Signs and symptoms of frost-bite may include 'pins and needles', paleness followed by numbness, a hardening and stiffening of the skin, a progression of colour changes in the affected area, (first white, then mottled and blue and eventually black; on recovery, red, hot, painful and blistered).</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p>
Eye	<p>This material causes serious eye irritation.</p>

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## TensorGrip X41 Canister Spray Adhesive

	<p>Instillation of isoparaffins into rabbit eyes produces only slight irritation.</p> <p>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..</p> <p>Eye contact with alkyl ethers (vapours or liquid) may produce irritation, redness and lachrymation.</p> <p>Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.</p>																				
Chronic	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.</p> <p>Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory 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. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.</p> <p>Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms.</p> <p>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</p> <p>Animal studies:</p> <p>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.</p> <p>Principal route of occupational exposure to the gas is by inhalation.</p> <p>Chronic effects of exposure to methyl acetate may be similar to those from methanol exposure because methyl acetate can be hydrolysed to yield methanol and acetic acid. Optic nerve damage is the predominant hazard.</p> <p>Chronic exposure to alkyl ethers may result in loss of appetite, excessive thirst, fatigue, and weight loss</p> <p>Chronic solvent inhalation exposures may result in nervous system impairment and liver and blood changes. [PATTYS]</p> <p>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p>																				
TensorGrip X41 Canister Spray Adhesive	<table> <tr> <th>TOXICITY</th><th>IRRITATION</th></tr> <tr> <td>Not Available</td><td>Not Available</td></tr> </table>	TOXICITY	IRRITATION	Not Available	Not Available																
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naphtha petroleum, light, hydrotreated	<table> <tr> <th>TOXICITY</th><th>IRRITATION</th></tr> <tr> <td>dermal (rat) LD50: 3.35 mg/kg<sup>[2]</sup></td><td>Eye: no adverse effect observed (not irritating)<sup>[1]</sup></td></tr> <tr> <td>Inhalation (Rat) LC50: 0.26 mg/L4h<sup>[2]</sup></td><td>Skin: adverse effect observed (irritating)<sup>[1]</sup></td></tr> <tr> <td>Oral (Rat) LD50: 16.75 mg/kg<sup>[2]</sup></td><td></td></tr> </table>	TOXICITY	IRRITATION	dermal (rat) LD50: 3.35 mg/kg <sup>[2]</sup>	Eye: no adverse effect observed (not irritating) <sup>[1]</sup>	Inhalation (Rat) LC50: 0.26 mg/L4h <sup>[2]</sup>	Skin: adverse effect observed (irritating) <sup>[1]</sup>	Oral (Rat) LD50: 16.75 mg/kg <sup>[2]</sup>													
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TensorGrip X41 Canister Spray Adhesive

styrene/ butadiene rubber	TOXICITY		IRRITATION	
	Dermal (rabbit) LD50: >20000 mg/kg <sup>[2]</sup>		Eye (Rodent - rabbit): 500mg/24H - Mild	
	Oral (Rat) LD50: 71000 mg/kg <sup>[2]</sup>		Skin (Rodent - rabbit): 500mg - Mild	
dimethyl ether	TOXICITY		IRRITATION	
	Inhalation (Rat) LC50: >20000 ppm4h <sup>[1]</sup>		Skin: no adverse effect observed (not irritating) <sup>[1]</sup>	
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			

TensorGrip X41 Canister Spray Adhesive	<p>Generally,linear and branched-chain alkyl esters are hydrolysed to their component alcohols and carboxylic acids in the intestinal tract, blood and most tissues throughout the body. Following hydrolysis the component alcohols and carboxylic acids are metabolized</p> <p>Oral acute toxicity studies have been reported for 51 of the 67 esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids. The very low oral acute toxicity of this group of esters is demonstrated by oral LD50 values greater than 1850 mg/kg bw</p> <p>Genotoxicity studies have been performed in vitro using the following esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids: methyl acetate, butyl acetate, butyl stearate and the structurally related isoamyl formate and demonstrates that these substances are not genotoxic.</p> <p>The JEFCA Committee concluded that the substances in this group would not present safety concerns at the current levels of intake the esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids are generally used as flavouring substances up to average maximum levels of 200 mg/kg. Higher levels of use (up to 3000 mg/kg) are permitted in food categories such as chewing gum and hard candy. In Europe the upper use levels for these flavouring substances are generally 1 to 30 mg/kg foods and in special food categories like candy and alcoholic beverages up to 300 mg/kg foods</p> <p><b>International Program on Chemical Safety: the Joint FAO/WHO Expert Committee on Food Additives (JECFA)</b></p> <p><b>Esters of Aliphatic acyclic primary alcohols with aliphatic linear saturated carboxylic acids.; 1998</b></p>
ACETONE	<p>For acetone:</p> <p>The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m3 and in rats at 26,100 mg/m3. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m3 for both rats and mice.</p> <p>Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m3, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals.</p> <p>The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m3 have been reported.</p> <p>Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m3 were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m3 or greater.</p>
METHYL ACETATE	<p>for methyl acetate</p> <p><b>Acute toxicity:</b></p> <p>Methyl acetate is a water soluble substance with high volatility. The substance has narcotic properties if inhaled at concentrations of 34 mg/l (mice) and 56 mg/l (cats) with a short duration of the narcotic action after cessation of exposure.</p> <p>Methyl acetate is absorbed via the lungs in animals and humans, absorption via the oral route is demonstrated. After absorption the substance undergoes hydrolysis to methanol and acetic acid.</p> <p>From the available <i>in vitro</i> data it may be anticipated that the half-life of methyl acetate in blood ranges between 2 and 4 hours. Immediately after stopping a 6-hour inhalation exposure to rats (2,000 ppm (6,040 mg/m3)) blood concentrations below the limit of quantification (less than 4.6 mg/l) were determined indicating rapid hydrolysis and high clearance of the substance. It appears from these data that the systemic availability of methyl acetate is low.</p> <p>The main metabolite is methanol which itself is metabolised to formic acid. Formate is introduced into C1-metabolism after activation by reacting with tetrahydrofolate. Humans as well as monkeys are more sensitive to methanol poisoning compared with rats because of a lower tetrahydrofolate content in liver. Therefore interspecies differences in the metabolism were considered mainly of concern at dose levels leading to acute toxicity. Thus rat is a useful model to indicate subacute/subchronic toxic effects below sublethal dosages.</p> <p>Assessment of the available animal toxicology data indicates that methyl acetate is of low acute toxicity (rats LD50 oral: 6,482 mg/kg bw, dermal: &gt;2,000 mg/kg bw, LC50 inhalative &gt;49 mg/l/4h). After oral application and after inhalation of substance vapours, animals showed narcotic symptoms, spasms, dyspnea and vomiting; inhalation of vapours in addition caused irritation of eyes and upper respiratory tract.</p> <p>The narcotic concentration for mice starts at 34 mg/l and for cats with 56 mg/l inhaled.</p> <p>In humans, accidental inhalation of vapours of methyl acetate caused severe headache and considerable somnolence.</p> <p>Methyl acetate has proven to cause only weak skin irritation in humans and in rabbits (no oedema, erythema with maximum grade 1 reversible within 48 hours). Eye irritation however, was strong but reversible within 7 days in a Draize eye test with rabbits. Exposure to methyl acetate vapours causes irritation to eyes and respiratory tract of humans.</p> <p>Taking into account the long experience with human exposure to the substance, methyl acetate is not supposed to exhibit skin sensitising properties although no relevant human or animal data are available.</p> <p><b>Sensitisation:</b></p> <p>Relevant human data are not available. In a maximisation test with 25 volunteers no sensitisation was observed after exposure to 10% methyl acetate in petrolatum (Kligman, 1976). Taking into account the long experience with human exposure to the substance, and the absence of any reports on contact allergy in exposed persons , methyl acetate is not expected to exhibit skin sensitising properties, especially since the substance is hydrolysed in contact with water by non-specific tissue esterases to methanol and acetic acid. For these substances a skin sensitisation potential is either absent (methanol, or restricted to a few cases (acetic acid).</p> <p><b>Repeat dose toxicity:</b></p> <p>Overall, reliable experimental animal data on the local and systemic effects after repeated administration of methyl acetate are restricted to the inhalation exposure. After nose-only inhalation during a 28-day treatment period, methyl acetate induced degeneration/necrosis of the rat olfactory mucosa at a concentration of 2,000 ppm on 6 hours/day, 5 days/week (6,040 mg/m3). There was some concern on minimal effects of systemic toxicity at this concentration diureses, minimal liver cell dysfunction, adrenal weight increase, and reduced serum cholesterol concentrations).</p> <p>There are no adequate data from human experience on repeated or prolonged exposure.</p> <p>Based on general experience that acute and long-time or repeated exposure to methyl acetate defats skin and cause dryness and cracking of the skin.</p> <p><b>No-observed-adverse-effect-level (NOAEL)</b></p> <p><i>Inhalation route</i></p>



## TensorGrip X41 Canister Spray Adhesive

The NOAEC for local effects on the respiratory tract derived from an accurate 28-day inhalation study in rats was 350 ppm (1,057 mg/m<sup>3</sup>). The NOAEC for systemic effects also derived from a 28-day inhalation study was 350 ppm (1,057 mg/m<sup>3</sup>).

**Mutagenicity**

Methyl acetate is negative in a bacterial mutation test and a rat bone marrow micronucleus test. Furthermore, the hydrolysis products methanol and acetic acid do not reveal evidence for a mutagenic potential. There is no concern with respect to mutagenicity. Methyl acetate should not be classified as a mutagen.

**Reproductive toxicity:**

There are no data on reproductive toxicity of methyl acetate. However, due to the rapid hydrolysis of this compound it is justified to base hazard assessment with respect to reproduction on the toxicological properties of the immediate metabolites. Concerning the metabolites of methyl acetate, acetic acid appears to be of less significance, since there are no indications of a foetotoxic or teratogenic potential, whereas for methanol some embryo-/foetotoxic and teratogenic effects were demonstrated in rodents, however at relatively high concentrations, respectively maternal toxic concentrations only. A NOEC/fertility for methanol of 1,000 ppm (1,300 mg methanol/m<sup>3</sup>) was derived from a 2-generation inhalation study in rats. With the assumption that methyl acetate is immediately degraded to methanol at a molar ratio of 1, this value can be converted to NOAEC/fertility of about 3,000 mg methyl acetate/m<sup>3</sup>. A NOAEC/developmental toxicity for methanol of 1,000 ppm (1,300 mg methanol/m<sup>3</sup>) was derived from two studies in mice and rats from intermittent as well as from continuous inhalatory exposure, which can be converted to a NOAEC/developmental toxicity of about 3,000 mg methyl acetate/m<sup>3</sup>. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

**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/m<sup>3</sup>) 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 LBPNS 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/m<sup>3</sup>, 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/m<sup>3</sup>. 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/m<sup>3</sup> 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/m<sup>3</sup>, increased kidney weight was observed in male and female rats (increased kidney weight was also observed in males only at 870 mg/m<sup>3</sup>). Furthermore, decreased body weight in male and female mice was also observed at 6170 mg/m<sup>3</sup>. 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 LBPNS 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 LBPNS 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 effects of human exposure to LBPNS 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/m<sup>3</sup> 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 LBPNS group. Both the European Commission and the International Agency for Research on Cancer (IARC) have classified LBPNS 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 LBPNS, 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 LBPNS substances evaluated. Most of these studies were carried out by inhalation exposure in rodents. NOAEC



## TensorGrip X41 Canister Spray Adhesive

values for reproductive toxicity following inhalation exposure ranged from 1701 mg/m<sup>3</sup> (CAS RN 8052-41-3) to 27 687 mg/m<sup>3</sup> (CAS RN 64741-63-5) for the LBPNS group evaluated, and from 7690 mg/m<sup>3</sup> to 27 059 mg/m<sup>3</sup> 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/m<sup>3</sup>, 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/m<sup>3</sup> delivered pups with higher birth weights. Cognitive and memory impairments were also observed in the offspring. Low Boiling Point Naphthas [Site-Restricted]

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.

**TensorGrip X41 Canister Spray Adhesive & STYRENE/BUTADIENE RUBBER**

Occupational exposures in the rubber-manufacturing industry are carcinogenic to humans (Group 1). IARC Working Groups. There is sufficient evidence in humans for the carcinogenicity of occupational exposures in the rubber-manufacturing industry. Occupational exposures in the rubber-manufacturing industry cause leukaemia, lymphoma, and cancers of the urinary bladder, lung, and stomach. Also, a positive association has been observed between occupational exposures in the rubber-manufacturing industry and cancers of the prostate, oesophagus, and larynx. IARC Working Group.

The multiple genetic and cytogenetic effects observed among workers employed in the rubber-manufacturing industry provide strong evidence to support genotoxicity as one mechanism for the observed increase in cancer risks. However, due to the complexity and changing nature of the exposure mixture and the potential interactions between exposures in the rubber-manufacturing industry, other mechanisms are also likely to play a role. While it is clear that exposure to some agents in the rubber-manufacturing industry has been reduced over time, the results of recent cytogenetic studies continue to raise concerns about cancer risks.

The rubber-manufacturing industry has used and still uses a wide variety of substances that belong to many different chemical categories, e.g. carbon black, aromatic amines, PAH, N-nitrosamines, mineral oils, other volatile organic compounds from curing fumes, trace amounts

## TensorGrip X41 Canister Spray Adhesive

	of monomers from synthetic rubber like 1,3-butadiene, acetonitrile, styrene, vinyl chloride, ethylene oxide, etc.. For this reason, it has been difficult to relate the observed cancer hazards in the rubber-manufacturing industry to exposure to specific chemicals.			
<b>TensorGrip X41 Canister Spray Adhesive &amp; NAPHTHA PETROLEUM, LIGHT, HYDROTREATED</b>	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 than 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.			
<b>ACETONE &amp; METHYL ACETATE &amp; STYRENE/ BUTADIENE RUBBER</b>	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.			
<b>Acute Toxicity</b>	✗	<b>Carcinogenicity</b>	✗	
<b>Skin Irritation/Corrosion</b>	✓	<b>Reproductivity</b>	✗	
<b>Serious Eye Damage/Irritation</b>	✓	<b>STOT - Single Exposure</b>	✓	
<b>Respiratory or Skin sensitisation</b>	✗	<b>STOT - Repeated Exposure</b>	✗	
<b>Mutagenicity</b>	✗	<b>Aspiration Hazard</b>	✗	

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

## SECTION 12 Ecological information

## Toxicity

TensorGrip X41 Canister Spray Adhesive	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
acetone	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	6098.4mg/L	5
	EC50	72h	Algae or other aquatic plants	5600-10000mg/L	4
	LC50	96h	Fish	3744.6-5000.7mg/L	4
	EC50	96h	Algae or other aquatic plants	9.873-27.684mg/l	4
	NOEC(ECx)	12h	Fish	0.001mg/L	4
methyl acetate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	1026.7mg/l	1
	EC50	72h	Algae or other aquatic plants	>120mg/l	1
	NOEC(ECx)	72h	Algae or other aquatic plants	>=120mg/l	1
	LC50	96h	Fish	250mg/l	1
naphtha petroleum, light, hydrotreated	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	0.64mg/l	2
	LC50	96h	Fish	0.11mg/l	2
	EC50	96h	Algae or other aquatic plants	64mg/l	2
	NOEC(ECx)	504h	Crustacea	0.17mg/l	2
styrene/ butadiene rubber	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
dimethyl ether	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>4400mg/L	2
	EC50	96h	Algae or other aquatic plants	154.917mg/l	2
	NOEC(ECx)	48h	Crustacea	>4000mg/l	1
	LC50	96h	Fish	1783.04mg/l	2
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				

When released in the environment, alkanes don't undergo rapid biodegradation, because they have no functional groups (like hydroxyl or carbonyl) that are needed by most organisms in order to metabolize the compound.

However, some bacteria can metabolise some alkanes (especially those linear and short), by oxidizing the terminal carbon atom. The product is an alcohol, that could be next oxidised to an aldehyde, and finally to a carboxylic acid. The resulting fatty acid could be metabolised through the fatty acid degradation pathway.

Continued...

## TensorGrip X41 Canister Spray Adhesive

For petroleum distillates:

Environmental fate:

When petroleum substances are released into the environment, four major fate processes will take place: dissolution in water, volatilization, biodegradation and adsorption.

These processes will cause changes in the composition of these UVCB substances. In the case of spills on land or water surfaces, photodegradation-another fate process-can also be significant.

As noted previously, the solubility and vapour pressure of components within a mixture will differ from those of the component alone. These interactions are complex for complex UVCBs such as petroleum hydrocarbons.

Each of the fate processes affects hydrocarbon families differently. Aromatics tend to be more water-soluble than aliphatics of the same carbon number, whereas aliphatics tend to be more volatile. Thus, when a petroleum mixture is released into the environment, the principal water contaminants are likely to be aromatics, whereas aliphatics will be the principal air contaminants. The trend in volatility by component class is as follows: alkenes = alkanes > aromatics = cycloalkanes.

The most soluble and volatile components have the lowest molecular weight; thus there is a general shift to higher molecular weight components in residual materials.

Biodegradation:

Biodegradation is almost always operative when petroleum mixtures are released into the environment. It has been widely demonstrated that nearly all soils and sediments have populations of bacteria and other organisms capable of degrading petroleum hydrocarbons. Degradation occurs both in the presence and absence of oxygen. Two key factors that determine degradation rates are oxygen supply and molecular structure. In general, degradation is more rapid under aerobic conditions. Decreasing trends in degradation rates according to structure are as follows:

(1) n-alkanes, especially in the C10–C25 range, which are degraded readily;

(2) isoalkanes;

(3) alkenes;

(4) benzene, toluene, ethylbenzene, xylenes (BTEX) (when present in concentrations that are not toxic to microorganisms);

(5) monoaromatics;

(6) polynuclear (polycyclic) aromatic hydrocarbons (PAHs); and

(7) higher molecular weight cycloalkanes (which may degrade very slowly).

Three weathering processes-dissolution in water, volatilization and biodegradation-typically result in the depletion of the more readily soluble, volatile and degradable compounds and the accumulation of those most resistant to these processes in residues.

When large quantities of a hydrocarbon mixture enter the soil compartment, soil organic matter and other sorption sites in soil are fully saturated and the hydrocarbons will begin to form a separate phase (a non-aqueous phase liquid, or NAPL) in the soil. At concentrations below the retention capacity for the hydrocarbon in the soil, the NAPL will be immobile; this is referred to as residual NAPL. Above the retention capacity, the NAPL becomes mobile and will move within the soil.

Bioaccumulation:

Bioaccumulation potential was characterized based on empirical and/or modelled data for a suite of petroleum hydrocarbons expected to occur in petroleum substances.

Bioaccumulation factors (BAFs) are the preferred metric for assessing the bioaccumulation potential of substances, as the bioconcentration factor (BCF) may not adequately account for the bioaccumulation potential of substances via the diet, which predominates for substances with log Kow > ~4.5.

In addition to fish BCF and BAF data, bioaccumulation data for aquatic invertebrate species were also considered. Biota-sediment/soil accumulation factors (BSAFs), trophic magnification factors and biomagnification factors were also considered in characterizing bioaccumulation potential.

Overall, there is consistent empirical and predicted evidence to suggest that the following components have the potential for high bioaccumulation, with BAF/BCF values greater than 5000: C13–C15 isoalkanes, C12 alkenes, C12–C15 one-ring cycloalkanes, C12 and C15 two-ring cycloalkanes, C14 polycycloalkanes, C15 one-ring aromatics, C15 and C20 cycloalkane monoaromatics, C12–C13 diaromatics, C20 cycloalkane diaromatics, and C14 and C20 three-ring PAHs.

These components are associated with a slow rate of metabolism and are highly lipophilic. Exposures from water and diet, when combined, suggest that the rate of uptake would exceed that of the total elimination rate. Most of these components are not expected to biomagnify in aquatic or terrestrial foodwebs, largely because a combination of metabolism, low dietary assimilation efficiency and growth dilution allows the elimination rate to exceed the uptake rate from the diet; however, one study suggests that some alkyl-PAHs may biomagnify. While only BSAFs were found for some PAHs, it is possible that BSAFs will be > 1 for invertebrates, given that they do not have the same metabolic competency as fish.

In general, fish can efficiently metabolize aromatic compounds. There is some evidence that alkylation increases bioaccumulation of naphthalene but it is not known if this can be generalized to larger PAHs or if any potential increase in bioaccumulation due to alkylation will be sufficient to exceed a BAF/BCF of 5000.

Some lower trophic level organisms (i.e., invertebrates) appear to lack the capacity to efficiently metabolize aromatic compounds, resulting in high bioaccumulation potential for some aromatic compounds as compared to fish.

This is the case for the C14 three-ring PAH, which was bioconcentrated to a high level (BCF > 5000) by invertebrates but not by fish. There is potential for such bioaccumulative components to reach toxic levels in organisms if exposure is continuous and of sufficient magnitude, though this is unlikely in the water column following a spill scenario due to relatively rapid dispersal.

Bioaccumulation of aromatic compounds might be lower in natural environments than what is observed in the laboratory. PAHs may sorb to organic material suspended in the water column (dissolved humic material), which decreases their overall bioavailability primarily due to an increase in size. This has been observed with fish.

Ecotoxicity:

Diesel fuel studies in salt water are available. The values varied greatly for aquatic species such as rainbow trout and *Daphnia magna*, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. *Daphnia magna* had a 24-hour LC50 of 1.8 mg/L. The values varied greatly for aquatic species such as rainbow trout and *Daphnia magna*, demonstrating the inherent variability of diesel fuel compositions and its effects on toxicity. Most experimental acute toxicity values are above 1 mg/L. The lowest 48-hour LC50 for salmonids was 2.4 mg/L. *Daphnia magna* had a 24-hour LC50 of 1.8 mg/L.

The tropical mysid *Metamysidopsis insularis* was shown to be very sensitive to diesel fuel, with a 96-hour LC50 value of 0.22 mg/L; this species has been shown to be as sensitive as temperate mysids to toxicants. However, this study used nominal concentrations, and therefore was not considered acceptable. In another study involving diesel fuel, the effect on brown or common shrimp (*Crangon crangon*) a 96-hour LC50 of 22 mg/L was determined. A "gas oil" was also tested and a 96-hour LC50 of 12 mg/L was determined.

The steady state cell density of marine phytoplankton decreased with increasing concentrations of diesel fuel, with different sensitivities between species. The diatom *Phaeodactylum tricornutum* showed a 20% decrease in cell density in 24 hours following a 3 mg/L exposure with a 24-hour no-observed effect concentration (NOEC) of 2.5 mg/L. The microalga *Isochrysis galbana* was more tolerant to diesel fuel, with a 24-hour lowest-observed-effect concentration (LOEC) of 26 mg/L (14% decrease in cell density), and a NOEC of 25 mg/L.

Finally, the green algae *Chlorella salina* was relatively insensitive to diesel fuel contamination, with a 24-hour LOEC of 170 mg/L (27% decrease in cell density), and a NOEC of 160 mg/L. All populations of phytoplankton returned to a steady state within 5 days of exposure.

In sandy soils, earthworm (*Eisenia fetida*) mortality only occurred at diesel fuel concentrations greater than 10 000 mg/kg, which was also the concentration at which sub-lethal weight loss was recorded.

Nephrotoxic effects of diesel fuel have been documented in several animal and human studies. Some species of birds (mallard ducks in particular) are generally resistant to the toxic effects of petrochemical ingestion, and large amounts of petrochemicals are needed in order to cause direct mortality.

Most ethers are very resistant to hydrolysis, and the rate of cleavage of the carbon-oxygen bond by abiotic processes is expected to be insignificant.

Direct photolysis will not be an important removal process since aliphatic ethers do not absorb light at wavelengths >290 nm.

For n-heptane:

log Kow : 4.66

Koc : 2400-8100

Half-life (hr) air : 52.8

Half-life (hr) H<sub>2</sub>O surface water : 2.9-312

Henry's atm m<sup>3</sup>/mol: 2.06

BOD 5 if unstated: 1.92

COD : 0.06

BCF : 340-2000

log BCF : 2.53-3.31

**Environmental fate:**

Photolysis or hydrolysis of n-heptane are not expected to be important environmental fate processes. Biodegradation of n-heptane may occur in soil and water, however volatilisation and adsorption are expected to be more important fate processes. A high Koc (2400-8200) indicates n-heptane will be slightly mobile to immobile in soil. In aquatic systems n-heptane may partition from the water column to organic matter in sediments and suspended solids. The bioconcentration of n-heptane may be important in aquatic environments. The Henry's Law constant suggests rapid volatilisation from environmental waters and surface soils. The volatilisation half-lives from a model river and a model pond (the latter considers the effect of adsorption) have been estimated to be 2.9 hr and 13 days, respectively.

n-Heptane is expected to exist entirely in the vapour phase in ambient air. Reactions with photochemically produced hydroxyl radicals in the atmosphere have been shown to be important (estimated half-life of 2.4 days calculated from its rate constant of 7.15x10<sup>-12</sup> cm<sup>3</sup>/molecule-sec at 25 deg C). Data also suggests that night-time reactions with nitrate radicals may contribute to the atmospheric transformation of n-heptane, especially in urban environments. n-Heptane does not contain chromophores that absorb at wavelengths >290 nm and therefore is not expected to be susceptible to direct photolysis by sunlight.

Continued...

An estimated BCF of 2,000 using log Kow suggests the potential for bioconcentration in aquatic organisms is very high. Based on 100% degradation after 4 days in water inoculated with gasoline contaminated soil and 100% degradation after 25 days in water inoculated with activated sewage sludge, biodegradation is expected to be an important fate process for n-heptane in water.

**Ecotoxicity:**  
Fish LC50 (48 h): goldfish (*Carrasius auratus*) 4 mg/l; golden orfe (*Idus melanotus*) 2940 mg/l; western mosquitofish (*Gambusia affinis*) 4924 mg/l  
Daphnia LC50 (24 h): >10 mg/l  
Daphnia EC50 (96 h): 82 mg/l (immobilisation)  
Opposum shrimp (*Mysidopsis bahia*) LC50 (96 h): 0.1 mg/l  
Snail EC50 (96 h): 472 mg/l

For Ketones: Ketones, unless they are alpha, beta--unsaturated ketones, can be considered as narcosis or baseline toxicity compounds.  
Aquatic Fate: Hydrolysis of ketones in water is thermodynamically favourable only for low molecular weight ketones. Reactions with water are reversible with no permanent change in the structure of the ketone substrate. Ketones are stable to water under ambient environmental conditions. When pH levels are greater than 10, condensation reactions can occur which produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavourable. Based on its reactions in air, it seems likely that ketones undergo photolysis in water.

Terrestrial Fate: It is probable that ketones will be biodegraded by micro-organisms in soil and water.  
Ecotoxicity: Ketones are unlikely to bioconcentrate or biomagnify.

for acetone:  
log Kow: -0.24  
Half-life (hr) air: 312-1896  
Half-life (hr) H2O surface water: 20  
Henry's atm m3 /mol: 3.67E-05  
BOD 5: 0.31-1.76,46-55%  
COD: 1.12-2.07  
ThOD: 2.2  
BCF: 0.69

**Environmental fate:**  
Acetone preferentially locates in the air compartment when released to the environment. A substantial amount of acetone can also be found in water, which is consistent with the high water to air partition coefficient and its small, but detectable, presence in rain water, sea water, and lake water samples. Very little acetone is expected to reside in soil, biota, or suspended solids. This is entirely consistent with the physical and chemical properties of acetone and with measurements showing a low propensity for soil absorption and a high preference for moving through the soil and into the ground water  
In air, acetone is lost by photolysis and reaction with photochemically produced hydroxyl radicals; the estimated half-life of these combined processes is about 22 days. The relatively long half-life allows acetone to be transported long distances from its emission source.  
Acetone is highly soluble and slightly persistent in water, with a half-life of about 20 hours; it is minimally toxic to aquatic life.  
Acetone released to soil volatilises although some may leach into the ground where it rapidly biodegrades.  
Acetone does not concentrate in the food chain.  
Acetone meets the OECD definition of readily biodegradable which requires that the biological oxygen demand (BOD) is at least 70% of the theoretical oxygen demand (THOD) within the 28-day test period  
Drinking Water Standard: none available.  
Soil Guidelines: none available.  
Air Quality Standards: none available.

**Ecotoxicity:**  
Testing shows that acetone exhibits a low order of toxicity  
Fish LC50: brook trout 6070 mg/l; fathead minnow 15000 mg/l  
Bird LC0 (5 day): Japanese quail, ring-neck pheasant 40,000 mg/l  
Daphnia magna LC50 (48 h): 15800 mg/l; NOEC 8500 mg/l  
Aquatic invertebrate 2100 - 16700 mg/l  
Aquatic plant NOEC: 5400-7500 mg/l  
Daphnia magna chronic NOEC 1660 mg/l  
Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (*Tribolium confusum*) and the flour moth (*Ephestia kuehniella*) were exposed to an airborne acetone concentration of 61.5 mg/m3. The LT50 values for the eggs were 30-50% lower than for the adult. The direct application of acetone liquid to the body of the insects or surface of the eggs did not, however, cause any mortality.  
The ability of acetone to inhibit cell multiplication has been examined in a wide variety of microorganisms. The results have generally indicated mild to minimal toxicity with NOECs greater than 1700 mg/L for exposures lasting from 6 hr to 4 days. Longer exposure periods of 7 to 8 days with bacteria produced mixed results; but overall the data indicate a low degree of toxicity for acetone. The only exception to these findings were the results obtained with the flagellated protozoa (*Entosiphon sulcatum*) which yielded a 3-day NOEC of 28 mg/L.

**DO NOT** discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)
methyl acetate	LOW	LOW
dimethyl ether	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
acetone	LOW (BCF = 0.69)
methyl acetate	LOW (LogKOW = 0.18)
dimethyl ether	LOW (LogKOW = 0.1)

Mobility in soil

Ingredient	Mobility
acetone	HIGH (Log KOC = 1.981)
methyl acetate	MEDIUM (Log KOC = 3.324)
dimethyl ether	HIGH (Log KOC = 1.292)

SECTION 13 Disposal considerations

Waste treatment methods



Product / Packaging disposal	<ul style="list-style-type: none"><li>DO NOT allow wash water from cleaning or process equipment to enter drains.</li><li>It may be necessary to collect all wash water for treatment before disposal.</li><li>In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li><li>Where in doubt contact the responsible authority.</li></ul>
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TensorGrip X41 Canister Spray Adhesive

- ▶ 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	
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. (contains dimethyl ether)	
14.3. Transport hazard class(es)	Class	2.1
	Subsidiary Hazard	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Environmentally hazardous	
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. * (contains dimethyl ether)	
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	Environmentally hazardous	
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. (contains dimethyl ether)	
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	Marine Pollutant	
14.6. Special precautions for user	EMS Number	F-D , S-U
	Special provisions	274 362
	Limited Quantities	0

14.7. Maritime transport in bulk according to IMO instruments  
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
acetone	Not Available
methyl acetate	Not Available
naphtha petroleum, light, hydrotreated	Not Available
styrene/ butadiene rubber	Not Available
dimethyl ether	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
acetone	Not Available
methyl acetate	Not Available
naphtha petroleum, light, hydrotreated	Not Available
styrene/ butadiene rubber	Not Available
dimethyl ether	Not Available

SECTION 15 Regulatory information

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

acetone 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)

methyl acetate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals  
Australian Inventory of Industrial Chemicals (AIIC)

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

styrene/ butadiene rubber is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)  
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic  
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

dimethyl ether 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)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (acetone; methyl acetate; naphtha petroleum, light, hydrotreated; styrene/ butadiene rubber; dimethyl ether)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (styrene/ butadiene rubber)
Japan - ENCS	No (naphtha petroleum, light, hydrotreated)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
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	03/07/2025
Initial Date	27/04/2022

SDS Version Summary

Version	Date of Update	Sections Updated
4.5	03/07/2025	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (inhaled), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), Physical and chemical properties - Appearance, Toxicological information - Chronic Health, Hazards identification - Classification, Disposal considerations - Disposal, Exposure controls / personal protection - Engineering Control, Exposure controls / personal protection - Exposure Standard, Firefighting measures - Fire Fighter (extinguishing media), Firefighting measures - Fire Fighter (fire/explosion hazard), Firefighting measures - Fire Fighter (fire fighting), First Aid measures - First Aid (eye), First Aid measures - First Aid (inhaled), First Aid measures - First Aid (skin), First Aid measures - First Aid (swallowed), Handling and storage - Handling Procedure, Composition / information on ingredients - Ingredients, Exposure controls / personal protection - Personal Protection (other), Exposure controls / personal protection - Personal Protection (Respirator), Exposure controls / personal protection - Personal Protection (eye), Exposure controls / personal protection - Personal Protection (hands/feet), Accidental release measures - Spills (major), Accidental release measures - Spills (minor), Handling and storage - Storage (storage incompatibility), Handling and storage - Storage (storage requirement), Handling and storage - Storage (suitable container), Transport information - Transport

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
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code
- 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

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